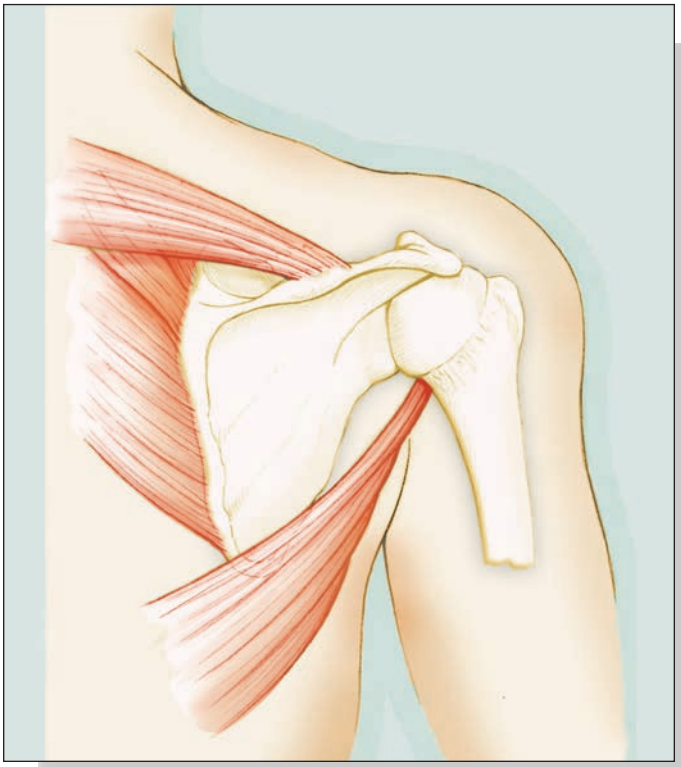
Disorders of the Shoulder
Diagnosis & Management

SECOND EDITION

VOLUME 1

Disorders of the Shoulder
Diagnosis & Management



SECOND EDITION

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To my son, Matthew, and my father, Frank, for their never-ending support. To my colleague and best friend, Jerry Williams.

—Joseph P. Iannotti

To all of my teachers, especially Charles A. Rockwood, Jr., M.D., for their wisdom and patience. To my friend and trusted colleague, Joseph Iannotti M.D., Ph.D., without whom my professional journey would be incomplete. To my family, especially my wife, Robin, and my children, Mark and Alexis, for the love and support we share with each other.

—Gerald R. Williams, Jr.

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Preface

Like the first edition of *Disorders of the Shoulder: Diagnosis and Management*, the second edition is intended to be a valuable reference text for any student of shoulder disorders—including shoulder, hand, and sports medicine subspecialists, orthopedic generalists with a subspecialty interest in shoulder disorders, orthopedic residents and fellows, and rehabilitation professionals. The contributing authors are experts in shoulder surgery from around the world, many of whom have trained with us over the last 15 years. In addition, we have authored or coauthored many of the chapters in the second edition and have contributed many figures to others, with the goal of producing a comprehensive text with a cohesive philosophy for diagnosis and treatment. The multidisciplinary aspect of shoulder disorders remains a common theme, with extensive coverage of supportive fields, including anesthesia, pain management, and rehabilitation.

Some of the best attributes of the first edition have been combined with new ideas to enhance the second edition. Treatment algorithms remain an important defining element, as we believe a logical, protocol-driven approach to common shoulder problems is valuable. Separate chapters on complications of surgical management of common shoulder disorders have been maintained. Basic science principles important to the pathogenesis and treatment of shoulder disorders continue to be emphasized, with a strong effort to correlate the basic science information with clinical practice. Treatment recommendations are grounded in peer-reviewed evidence and clinical experience, with concise, clinically relevant bibliographies.

The depth of certain chapters has been expanded to correspond to recent changes in clinical practice. Discussion of minimally invasive and arthroscopic surgery for a wide spectrum of rotator cuff pathology and instability problems has been added. Material on complex and revision surgery for joint replacement, rotator cuff repair, muscle transfer, and complex instability surgery has been updated. The chapters on scapular disorders and brachial plexus injuries contain new information on pathogenesis and treatment. Principles of tissue engineering that may be relevant in the future management of rotator cuff disorders are outlined in the chapter on anatomy, pathogenesis, and biomechanics of rotator cuff disease. Finally, reverse shoulder arthroplasty for arthritis and cuff deficiency and bone-sparing humeral resurfacing arthroplasty are new topics in the second edition.

One of the challenges of any comprehensive medical text is to stay current with the evolution of clinical practice. The field of shoulder surgery is evolving more rapidly than most orthopedic subspecialties. This is both a curse and a blessing. It is impossible to match the unrelenting pace of discovery in the diagnosis and management of shoulder disorders. However, it is a privilege to try—a privilege made possible by the interest of you, the readers. We hope the second edition meets your expectations and piques your interest enough to justify the creation of a third edition.

Rotator Cuff Disease and Tendon Ruptures

I

1 Anatomy, Biomechanics, and Pathophysiology of Rotator Cuff Disease

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ROTATOR CUFF AND RELATED ANATOMY

Normal Anatomy of the Rotator Cuff

Four muscle–tendon units make up the rotator cuff: the supraspinatus, infraspinatus, subscapularis, and teres minor. The shoulder comprises a complex of four articulations including the glenohumeral joint, scapulothoracic joint, sternoclavicular joint, and acromioclavicular joint that, under normal conditions, move in synchrony, affording smooth, unhindered motion of the arm. An intricate relation exists between the osseous elements and the surrounding muscles and ligaments. The tissues of the shoulder can be grouped into four separate and contiguous layers that alternate between muscular and fibrous elements.[56] Layer 1, the most superficial level, includes the deltoid and pectoralis major muscles. Layer 2 is a continuous fascial layer that extends circumferentially from anterior to posterior and incorporates the clavipectoral and posterior scapular fascia. Layer 3, again a muscular layer, includes the rotator cuff, and layer 4, the deepest level, comprises the fibrous capsular elements. This classification can contribute to a better understanding of the shoulder's complex anatomy and safeguard against deviation from internervous planes during surgical procedures (Fig. 1-1).

The four muscles that compose the rotator cuff take their origin from the body of the scapula and envelope the humeral head as they insert along the tuberosities of the proximal humerus.[66] The musculotendinous cuff is firmly adherent to the underlying glenohumeral capsule and provides circumferential reinforcement except at the rotator interval and axillary recess. The *rotator interval* is a triangular area that is made up of fibrous elements and bordered by the upper margin of the subscapularis tendon and anterior aspect of the supraspinatus.[73] Within the interval lie the coracohumeral ligament, the biceps tendon, and the superior

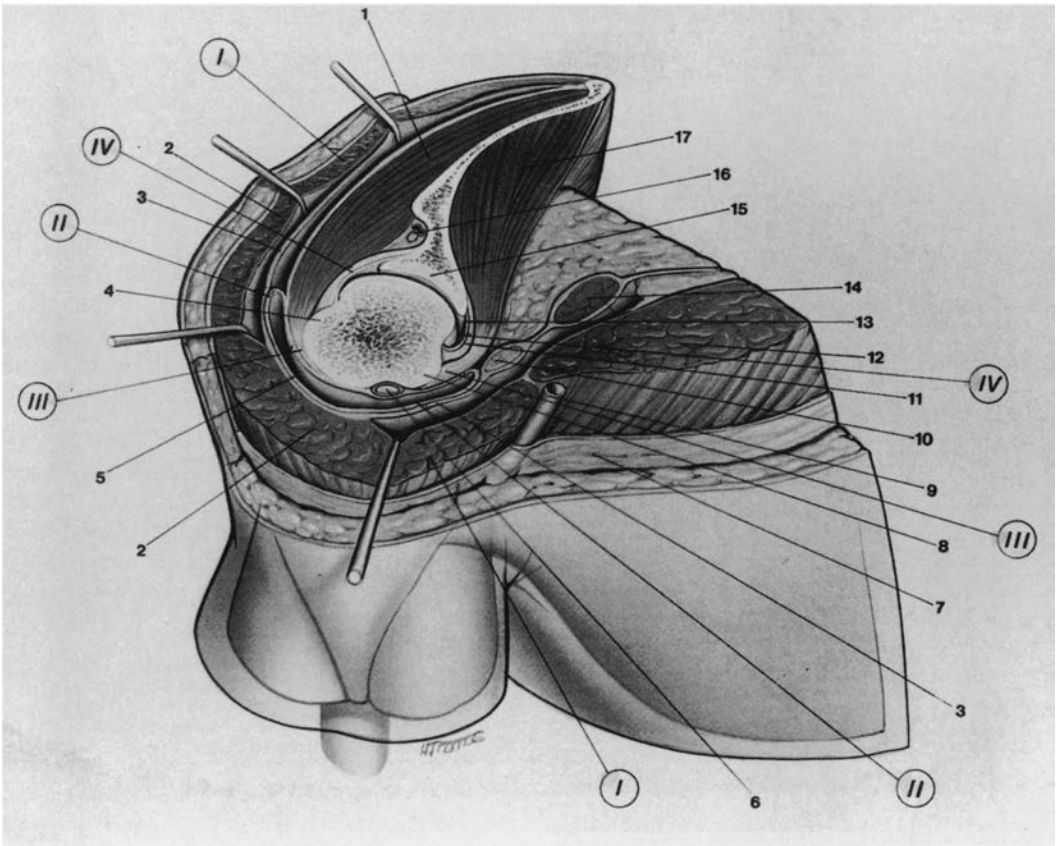

Figure 1-1 Supporting layers of the glenohumeral joint: **Layer I:** Deltoid (2) and pectoralis major muscles (12); **Layer II:** Clavipectoral fascia (3), conjoined tendon (10), coracoacromial ligament, posterior scapular fascia (3), and superficial bursal tissue (5); **Layer III:** Deep layer of subdeltoid bursa, rotator cuff (1,17); **Layer IV:** Glenohumeral joint capsule (11), synovium (13), coracohumeral ligament. (From Cooper DE, O'Brien SJ, Warren RF. Supporting layers of the glenohumeral joint. An antomic study. *Clin Orthop* 1993;289:144–155, with permission.)

glenohumeral ligament. The coracohumeral ligament is seen superficially, whereas the superior glenohumeral ligament reflects around the biceps tendon and serves as an internal pulley at the floor of this space.[127] The coracohumeral and superior glenohumeral ligaments take origin from the lateral base of the coracoid and superior labrum, respectively[99,173] (Fig. 1-2). In one report, through gross anatomic study, variability was observed at the insertion of the coracohumeral ligament. Seventy-four percent of specimens demonstrated a predominant insertion into the rotator interval; in the remainder, the principal attachment was to the supraspinatus tendon.[173] Multiple functions have been attributed to the coracohumeral ligament, and most notably, they include limitation of external rotation in the adducted arm and restraint against inferior translation.[18,107,109,186–188] The intraarticular boundaries of this space can be easily visualized arthroscopically and are marked by the glenoid rim, the upper subscapularis tendon, and the intraarticular portion of the biceps tendon (Fig. 1-3). The *axillary recess,* which also lacks muscular or tendinous coverage by the rotator cuff, demonstrates capsular redundancy at the inferior aspect of the joint. This tissue laxity affords normal arm abduction, whereby patulousness or contracture can mediate joint instability or restriction of motion.

The four rotator cuff muscles are often considered as distinct separate musculotendinous units that directly overlie the joint capsule and insert onto the proximal humerus. Clark and Harryman[48] performed gross anatomic and histologic studies in cadaveric specimens to better define the relation between the rotator cuff tendons and their underlying capsular elements. The fibers of the rotator cuff tendons interdigitate and fuse, forming a common insertion on the tuberosities of the humerus.[158] Fibers from both the subscapularis and infraspinatus interdigitate with respective fibers of the supraspinatus. Microscopically, the rotator cuff complex is stratified into five distinct layers that also receive reinforcement from the coracohumeral ligament and is contiguous with the glenohumeral capsule (Fig. 1-4).

The Subscapularis Muscle

Anteriorly, the *subscapularis* muscle takes its origin along the costal surface of the scapula and inserts onto the lesser

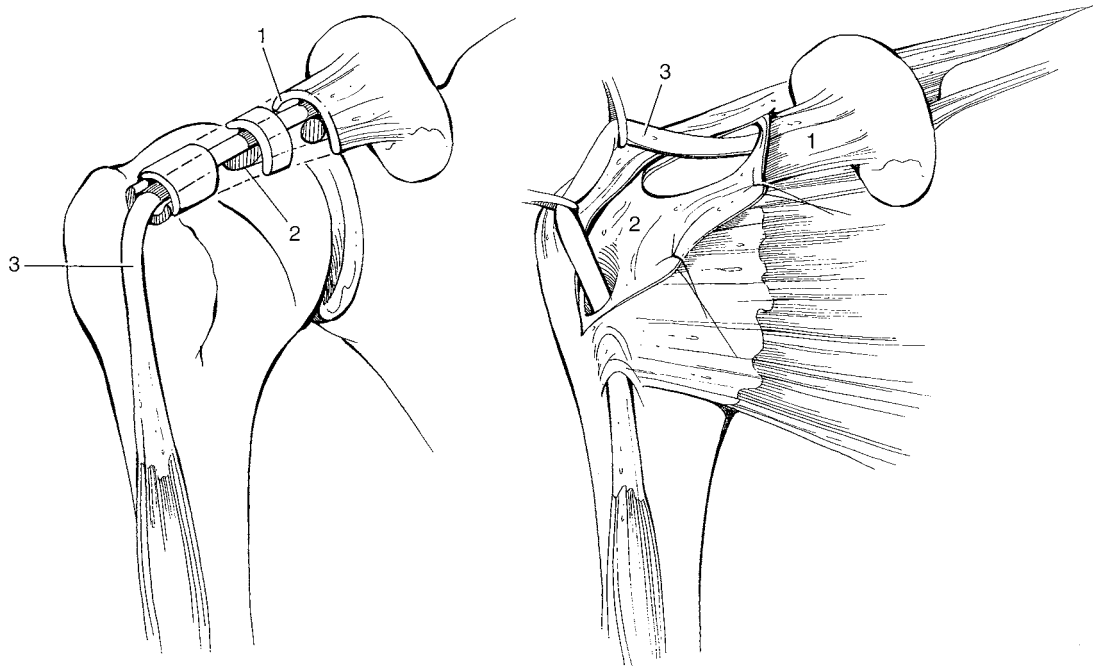

Figure 1-2 Anatomy of the rotator interval. Coracohumeral ligament (1); superior glenohumoral ligament (2); biceps tendon (3). (From Walch G, Laurent NJ, Levigne C, Renaud E. Tears of the supraspinatus tendon associated with "hidden" lesions of the rotator interval. *J Shoulder Elbow Surg* 1994;3:353–360, with permission.)

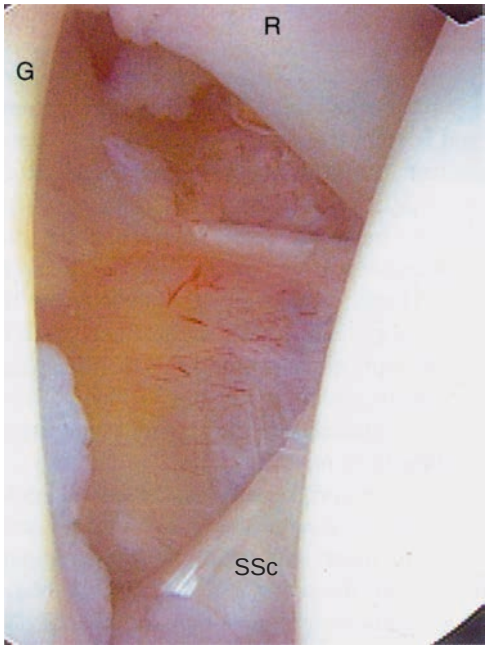

Figure 1-3 Region of rotator interval as viewed arthroscopically. Safe portal of entry lies between the biceps tendon (*R*), upper border of subscapularis tendon (*SSc*), and glenoid rim (*G*).

tuberosity of the humerus[26,159] (Fig. 1-5). It is the largest and most powerful of the rotator cuff muscles with its origin occuping greater than 90% of the anterior scapular surface. As the multipennate muscle funnels laterally toward its insertion, it has a dual innervation from both the upper and lower subscapular nerves that arise from the posterior cord of the brachial plexus. The upper subscapular nerve innervates a greater portion of the muscle. Although commonly considered to be an internal rotator of the shoulder, its contribution to arm abduction and humeral head depression has also been emphasized.[116,184,185,219] The subscapularis bursa lies between the subscapularis tendon and neck of the scapula. It is found just inferior to the coracoid process and protects the tendon as it courses along the scapular neck and coracoid.[159] The bursa communicates with the glenohumeral joint capsule and can harbor intraarticular loose bodies (Fig. 1-6). Zlatkin and colleagues[267] described three areas of capsular insertion along the anterior glenoid neck. A type I capsule is inserted adjacent to the anterior glenoid labrum, whereas types II and III are inserted progressively more medial on the scapular neck. This categorization likely reflects the variation in morphology and size of the subscapular bursa, which can be readily identified on coronal magnetic resonance images (MRIs). While a predisposition to anterior glenohumeral instability in patients with a medial capsular insertion has been suggested, no controlled studies elucidating this issue have yet been conducted.

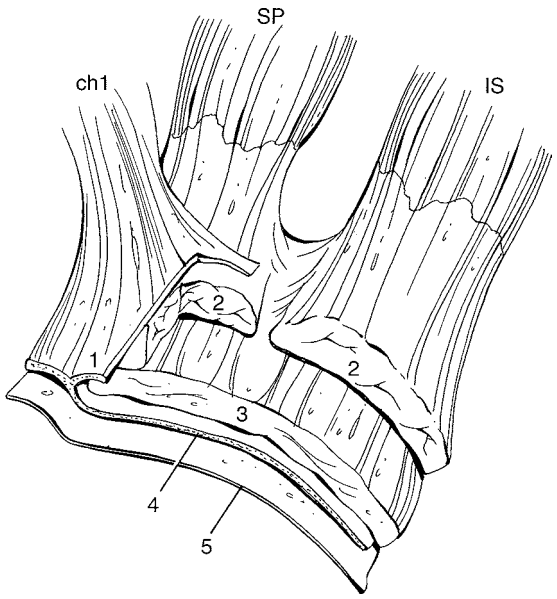

Figure 1-4 Schematic diagram of the supraspinatus and infraspinatus tendinous insertions. Five discrete layers are identified. The orientation of the fascicles in the numbered layers is indicated by the lines on their upper surfaces. *Layer 1* is composed of superficial fibers that overlie the cuff tendons and extend from the coracoid process to the greater tuberosity. These fibers form an extension of the coracohumeral ligament (*chl*). *Layers 2* and *3* contain the fibers of the supraspinatus (*SP*) and the infraspinatus (*IS*) tendons. The fibers in layer 2 are oriented parallel to the axes of the supraspinatus and infraspinatus tendons. The fibers of layer 3 are smaller and are obliquely oriented with respect to the fibers of layer 2. Within layer 3, the fibers of the supraspinatus tendon fan out and intermingle with the fibers of the adjacent infraspinatus and subscapularis tendons. This intermingling between the infraspinatus and supraspinatus creates the variation in alignment of the fibers observed in layer 3. In *layer 4*, the fibers make up the deep extension of the coracohumeral ligament. These fibers branch off the main body of the ligament at the anterior border of supraspinatus tendon and then course between the tendon and capsule as far as the junction between the infraspinatus and supraspinatus. *Layer 5* is the true joint capsule of the shoulder, which forms a continuous fibrous cylinder extending from the glenoid labrum to the neck of the humerus. The synovial lining of the capsule is in direct contact with the articular surface of the humeral head. The orientation of the fibers within the capsule is quite variable and not identified in this diagram. (From Clark JM, Harryman DT. Tendons, ligaments, and capsule of the rotator cuff. *J Bone Joint Surg Am* 1992;74:713–725, with permission.)

The Infraspinatus and Teres Minor Muscles

The *infraspinatus* and *teres minor* muscles make up the posterior portion of the rotator cuff. The infraspinatus is triangular and is often inseparable from the teres minor. They take origin from the infraspinatus fossa and dorsolateral border of the scapula, respectively, and insert onto the greater tuberosity of the humerus.[159] The suprascapular nerve innervates the infraspinatus, and the axillary nerve supplies the teres minor. These muscles externally rotate the humerus and stabilize the glenohumeral joint in concert with the subscapularis and supraspinatus muscles. The infraspinatus has a pennate muscle architecture with a central raphe that should not be confused with the intermuscular interval between it and the teres minor.

The Supraspinatus Muscle

The *supraspinatus* originates from the suprascapular fossa and inserts along the greater tuberosity of the humerus.[249] It receives its innervation from the suprascapular nerve, which arises from the upper trunk of the brachial plexus. This muscle contributes to glenohumeral compression during active shoulder motion and assists the deltoid in effecting humeral abduction.[52,53,114] The supraspinatus is situated between the humeral articular surface and the acromial arch, where it is protected by a synovial cavity on either side. The subacromial and subdeltoid bursae are found superficial to the tendon and separate it from the deltoid muscle. The bursa varies in size and extends laterally from the subacromial space to the proximal humeral metaphysis (Fig. 1-7).

The *suprascapular nerve* arises from the upper trunk of the brachial plexus and courses through the suprascapular notch just medial to the base of the coracoid process. It supplies the supraspinatus muscle before passing through the spinoglenoid notch, where it finally provides neural innervation to the infraspinatus (Fig. 1-8). Warner and associates[255] have highlighted the path and variational anatomy of the suprascapular nerve as it enters the posterior aspect of the shoulder. Eighty-four percent of 31 specimens revealed one or two branches of the nerve to the supraspinatus muscle. In 84% of specimens, the first branch originated either under the transverse scapular ligament or 1 mm distal to it. In 3%, the first motor branch originated proximal to the ligament and passed superficial to it. The infraspinatus muscle revealed three to four branches in approximately one-half of the specimens. Bigliani and coworkers[21] measured the distance of the nerve from fixed scapular landmarks and observed that it lay an average of 1.8 cm (range 1.4 to 2.5 cm) from the midposterior glenoid rim to the base of the scapular spine. The distance of the nerve from the supraglenoid tubercle to the base of the scapular spine measured an average of 2.5 cm (range 1.9 to 3.2 cm). These observations emphasize the caution required for surgical management of shoulder disorders such as mobilization of a torn and retracted rotator cuff, arthroscopic portal placement, transglenoid drilling, and neurolysis of an entrapped suprascapular nerve.

The Deltoid Muscle

Superficial to the subacromial and subdeltoid bursae lies the *deltoid* muscle. It is composed of three heads (anterior, middle, and posterior) that vary in structure and function. The muscle has an extensive origin, arising from the distal

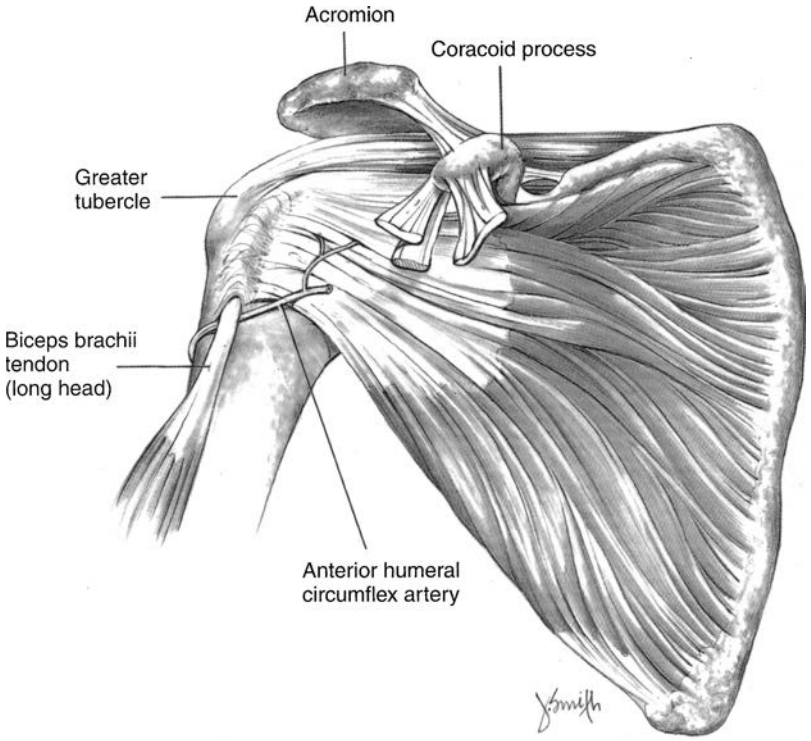

Figure 1-5 Anatomic course of the subscapularis muscle.

one-third of the clavicle, the acromion, and the lateral one-third of the scapular spine. It converges distally to insert on the deltoid tuberosity of the middiaphysis of the humerus (Fig. 1-9). Its broad origin, which is derived from the mobile scapula and clavicle, affords the deltoid a mechanical advantage by allowing the muscle to maintain its resting length at various arm positions. Furthermore, the bipennate structure of the large middle head contributes to abduction strength through contraction of its fibers at an angle to the line of pull, which also serves to maintain muscle fiber resting length and improve efficiency. In contrast, muscles with a parallel fiber arrangement, such as the anterior and posterior deltoid, by virtue of their structural configuration, result in considerably decreased strength during contraction.[60]

Differences in activity of the three portions of the deltoid relative to arm position have been observed through electromyographic analysis. The anterior and middle heads remain active at all angles of abduction and in multiple

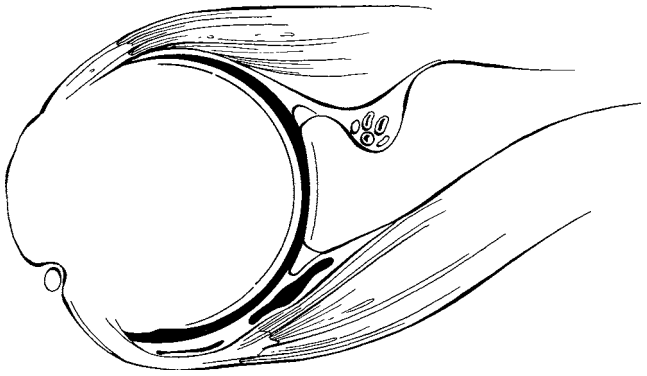

Figure 1-6 The subscapularis bursa. Note its subcoracoid location between the glenoid neck and subscapularis muscle belly.

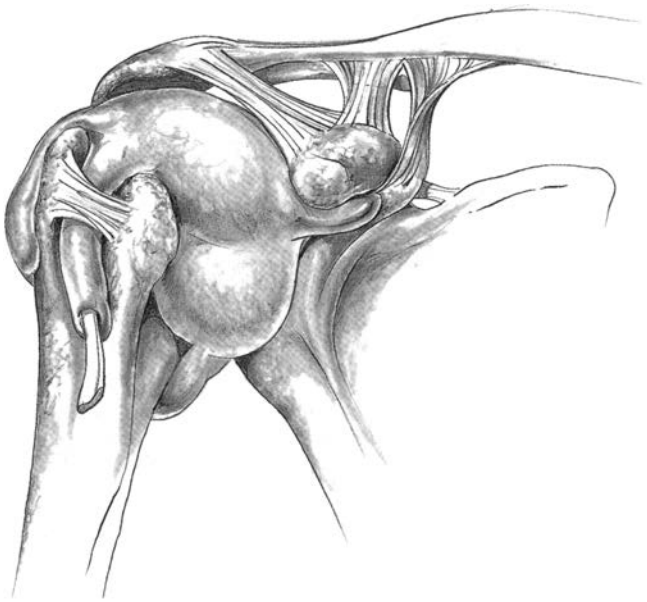

Figure 1-7 The subacromial and subdeltoid bursae.

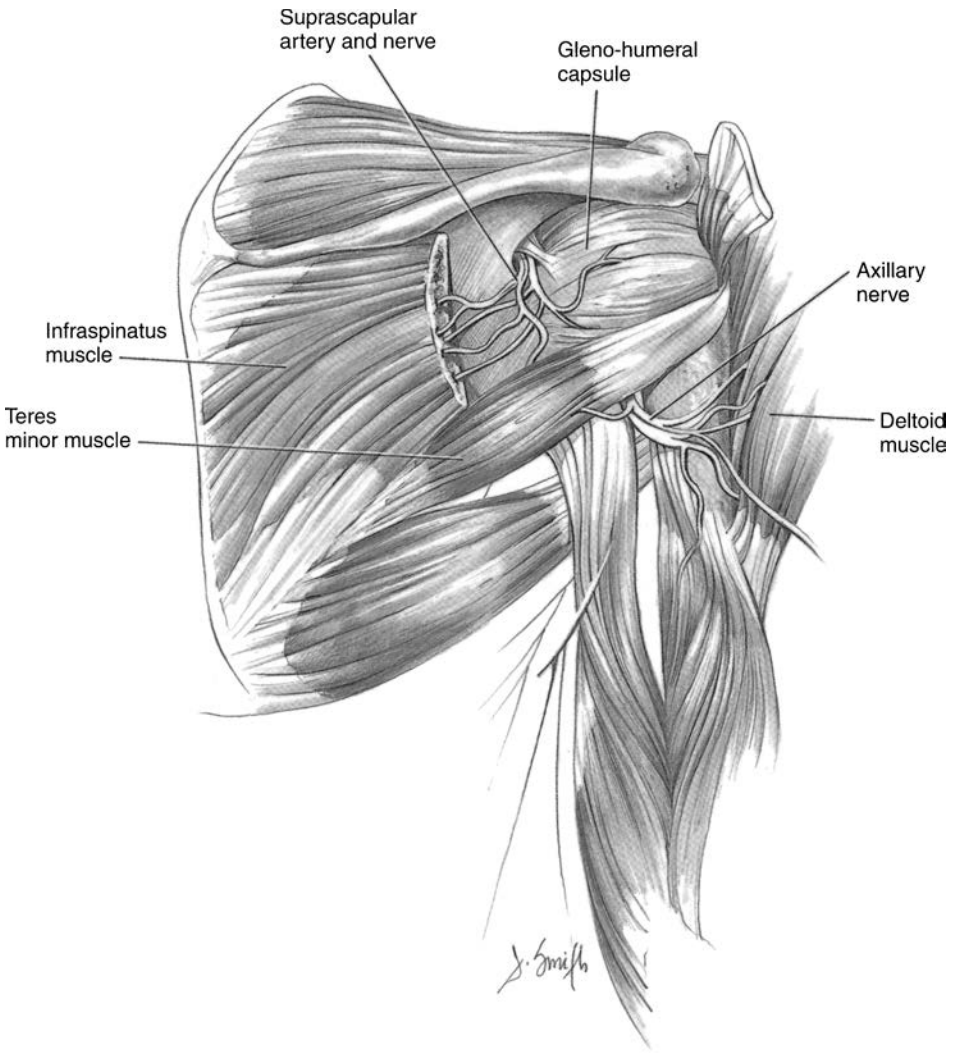

Figure 1-8 Anatomic course of the suprascapular nerve.

planes (coronal, scapular, and parasagittal), whereas the posterior deltoid, also an important shoulder extensor, contributes to elevation when the arm is above 110 degrees.[221] Moreover, when the arm is in abduction, the posterior deltoid functions as a secondary external rotator, for which its clinical importance is increased in patients with massive rotator cuff tears extending into the infraspinatus and teres minor tendons.

Neural innervation is afforded by the *axillary nerve,* which takes a circuitous path before entering the deltoid muscle. It arises from the posterior cord of the brachial plexus and courses across the inferolateral border of the subscapularis. It passes inferior to the glenohumeral axillary recess and exits the quadrangular space, along with the posterior humeral circumflex artery, where it divides into two trunks. The posterior trunk splits and innervates the teres minor and posterior deltoid before terminating as the superior lateral cutaneous nerve. The anterior trunk winds around the humerus and innervates the remaining deltoid muscle. It becomes subfascial and intramuscular at a point between the anterior and middle heads[35] (Fig. 1-10). Burkhead and colleagues[35] observed that the axillary nerve can occupy a position as close as 3.1 cm from the lateral tip of the acromion and that in 20% of specimens it was less than the generally accepted 5-cm distance.[1]

Vascular Supply to the Rotator Cuff

Multiple vessels contribute to the vascularity of the rotator cuff. The anterior and posterior humeral circumflex arteries both supply the superior, as well as the anterior and posterior portions of the cuff, respectively. The suprascapular artery also supplies the superior cuff, and in the majority of persons the acromial branch of the thoracoacromial artery will nourish the supraspinatus. Additional contributions may include branches of the subscapular and the suprahumeral branches of the axillary artery. Osseous vessels emanating from the tuberosities of the proximal humerus have also been included in the vascular makeup of the rotator cuff[5,43,83,145,161,208] (Fig. 1-11).

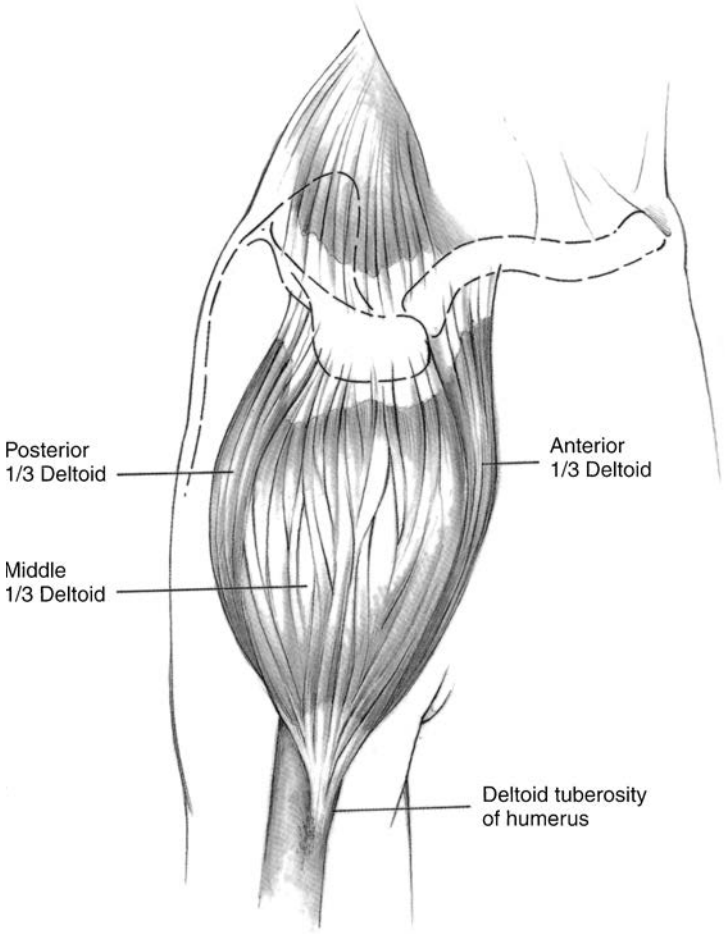

Figure 1-9 The deltoid muscle.

ROTATOR CUFF FUNCTION

The complex interaction of the rotator cuff and surrounding muscles is largely responsible for the shoulder's considerable range of motion and the preservation of glenohumeral joint stability. Although debate continues over a few functions of the rotator cuff, the preponderance of data support its role as a dynamic stabilizer, providing humeral depression, humeral rotation, abduction, and joint compression. Its role in "dynamization" or tensioning of the glenohumeral ligaments in the midranges of motion remains unclear and will require validation with further study. Furthermore, the rotator cuff has an integral part in maintaining force couples in multiple planes, whereby its absence could potentially result in abnormal kinematics, an unstable fulcrum, and abnormal humeral head excursion.

The rotator cuff comprises a group of muscles that are considerably smaller in size and cross-sectional area when compared with the more superficial structures, such as the deltoid, pectoralis major, latissimus dorsi, and trapezius. In addition, because they lie deep in the shoulder and in close proximity to the center of rotation of the glenohumeral joint, these muscles are collectively unable to generate the same degree of torque as the larger and more superficial structures. In part, the relatively shorter lever arm, or distance of the muscle from the center of rotation, accounts for observable differences in generated force. Consequently, given its anatomic architecture, maintenance of a stable glenohumeral fulcrum during active arm motion is one function that is both important and well suited to the rotator cuff (Fig. 1-12).

A normal-functioning rotator cuff achieves dynamic stability through multiple mechanisms. It acts through direct joint compression as well as through asymmetric contraction and "steering" of the humeral head into the glenoid during active motion.[19,94,142,180,192,212,261,263] Compression is achieved through the perpendicular vector of pull by the humeral head into the glenoid that serves to minimize tendencies toward joint subluxation.[142,180] Multiple forces pass across the shoulder during active motion to achieve a desired arm position. Force couples across the shoulder remain integral to maintaining normal function, especially when placed in a perspective of the minimally constrained

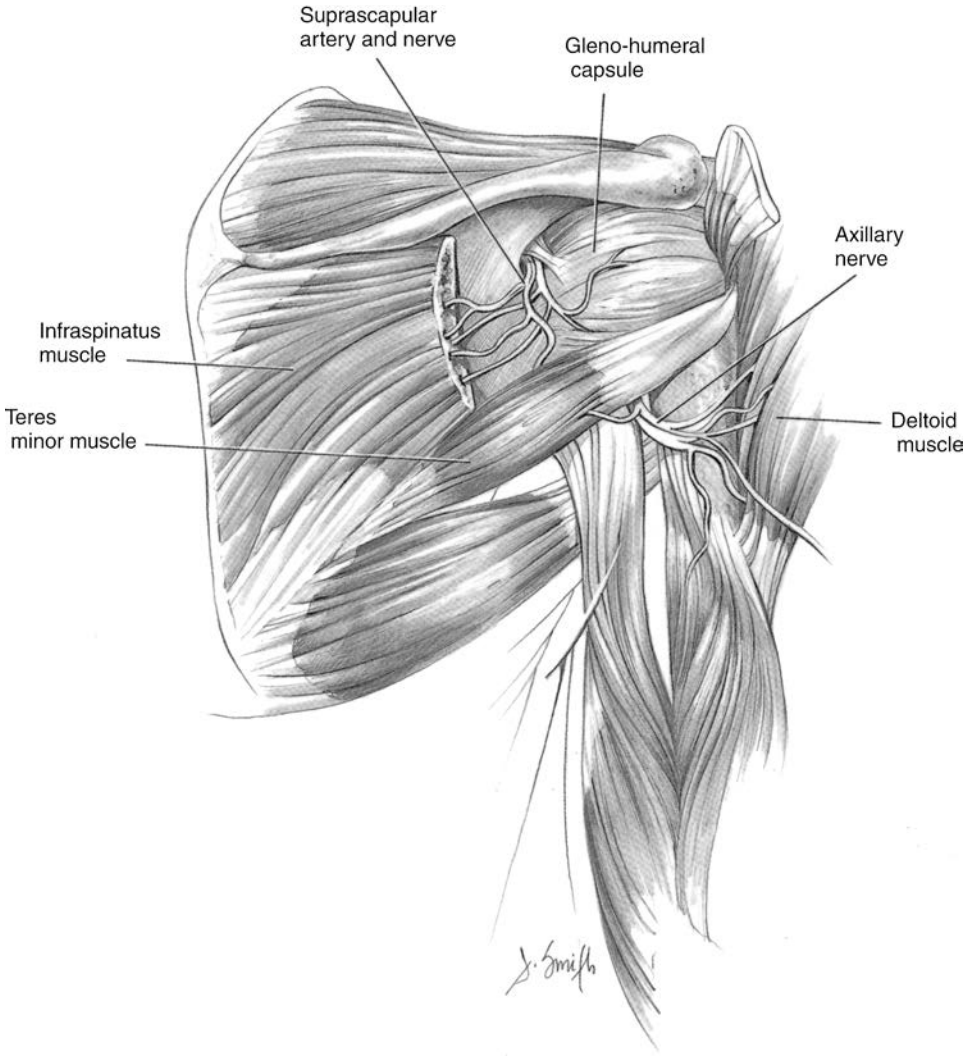

Figure 1-10 Anatomic course of the axillary nerve.

design of the glenohumeral joint. Force couples in various planes exist that can be defined as the action of two opposing muscle groups required to achieve a given movement. Inman and associates[116] initially described force couples involving the glenohumeral joint. They noted that the deltoid muscle acts to pull the humeral head in a cephalad direction, while the subscapularis, infraspinatus, and teres minor act as a functional unit counteracting the deltoid and effecting depression of the humeral head.

Other biomechanical studies have highlighted the role of the infraspinatus and subscapularis in maintenance of normal glenohumeral kinematics.[32,218] In one cadaveric investigation, the isolated absence of an applied supraspinatus force appeared to have no appreciable difference on humeral head migration when measured radiographically in the anteroposterior plane. Absence of force generated by the infraspinatus, teres minor, and subscapularis, on the other hand, resulted in an increase in superior humeral translation because the deltoid was unopposed.[218] Burkhart[32] expanded on the importance of force couples in preserving normal kinematics in patients with rotator cuff tears. He noted that balanced forces in both the coronal and transverse planes afforded normal glenohumeral motion patterns as long as anterior and posterior portions of the cuff were preserved beyond a critical threshold (Fig. 1-13). The location, as opposed to size of the tendon tear, was suggested to be a more significant determinant in resultant glenohumeral kinematics.

The rotator cuff contributes strength to the arm. Howell and coworkers[114] noted that, after selective blockade of the suprascapular and axillary nerves in normal volunteers, the supraspinatus and deltoid muscles contribute equally to measured torque in abduction. These findings are comparable with other reports on selective blocking of the axillary nerve in which it was noted that approximately 60% of the strength in abduction was attributable to the deltoid muscle.[52,53] Compromise of rotator cuff or deltoid function in such individuals could be expected to result in a

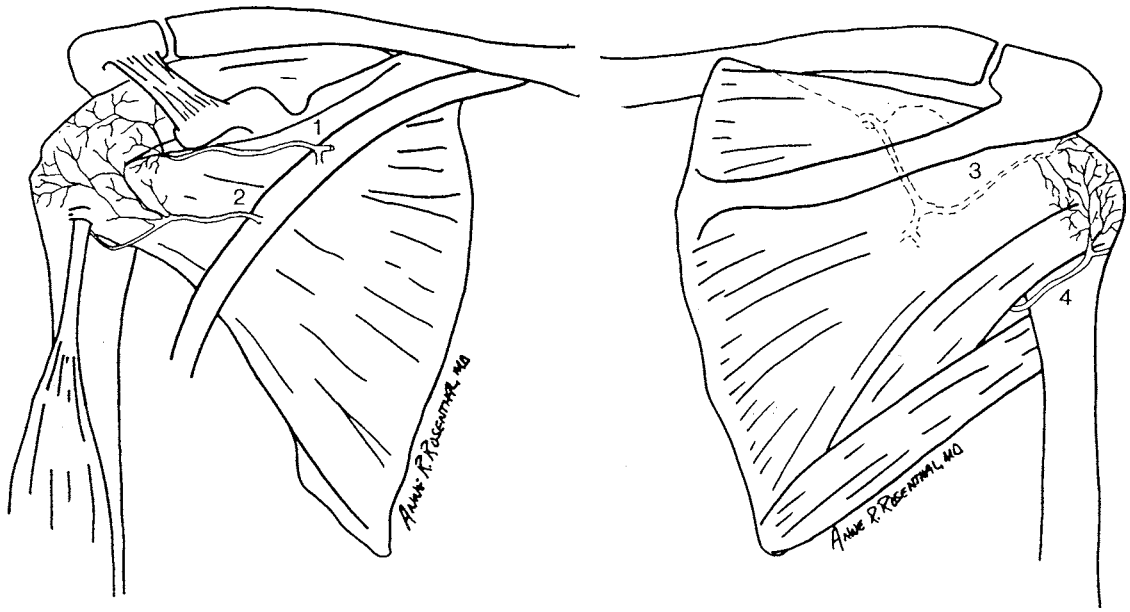

Figure 1-11 Vascular supply to the rotator cuff. 1 = suprahumeral branch; 2 = anterior humeral circumflex artery; 3 = suprascapular artery; 4 = posterior humeral circumflex artery. (From Choi HR, Kondo S, Hirose K, Ishiguro N, Hasegawa Y, Iwata H. Expression and enzymatic activity of MMP-2 during healing process of the acute supraspinatus tendon tear in rabbits. *J Orthop Res* 2002; 20(5):927–933, with permission.)

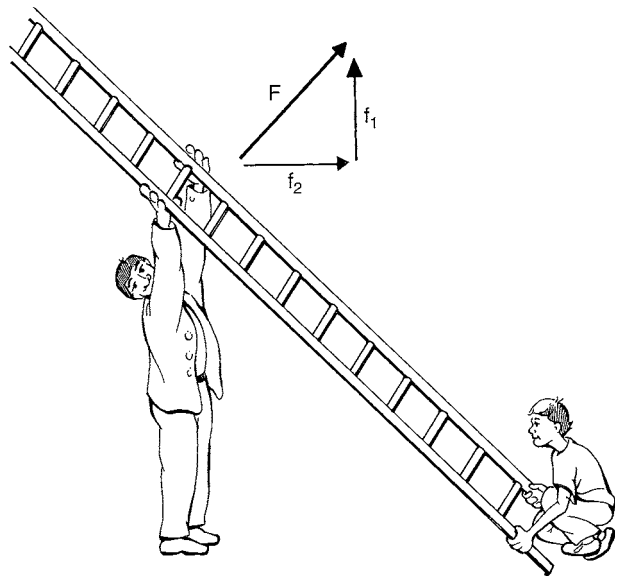

Figure 1-12 The shoulder muscles can be grouped as either primary movers or primary stabilizers. This situation is somewhat analagous to that of a large man and a small boy teaming up to lift a ladder. Typically, the stronger one will lift or move the ladder, while the weaker one will hold it from sliding or lifting off the ground (stabilizer). There is a point at which the force generated by the stronger one can overpower the resistance of the weaker one and stability is lost. (From O'Driscoll SW. Atraumatic instability: pathology and pathogenesis. In: Matsen FA, Fu FH, Hawkins RJ, eds. *The shoulder: a balance of mobility and stability.* Rosemont, IL: American Academy of Orthopaedic Surgeons, 1993: 305–316, with permission.)

progressive loss of muscle force with arm elevation and in early fatigability. The infraspinatus has also been implicated as a contributor to arm elevation. Otis and associates[185] reported decreases in abduction and external rotation torque of up to 45% and 75%, respectively, after selective paralysis of the infraspinatus muscle. These findings contrast with one electromyographic (EMG) study that demonstrated silent electrical activity in the infraspinatus when it was elevated to 120 degrees.[198]

In an effort to clarify the collective role of the infraspinatus, teres minor, and subscapularis, Sharkey and coworkers,[219] in a biomechanical cadaveric investigation, evaluated the contribution of these muscles to force in abduction. Because the tendons of the subscapularis and infraspinatus insert both above and below the humeral center of rotation, it is conceivable that portions of these muscles may act as arm abductors as well as humeral head depressors. Their data suggested that the combined contractions of these muscles contribute to arm abduction and that the magnitude of their contribution was similar to that of the supraspinatus. Otis and colleagues[184] provided further indirect evidence of the functional relation of the rotator cuff and deltoid to humeral elevation. By calculation of changes in moment arms and measurement of muscular excursion in cadaveric specimens, they demonstrated that both the infraspinatus and subscapularis contribute to abduction. Changes in rotation further affected the capacity of either muscle to augment elevation in the scapular plane. Internal and

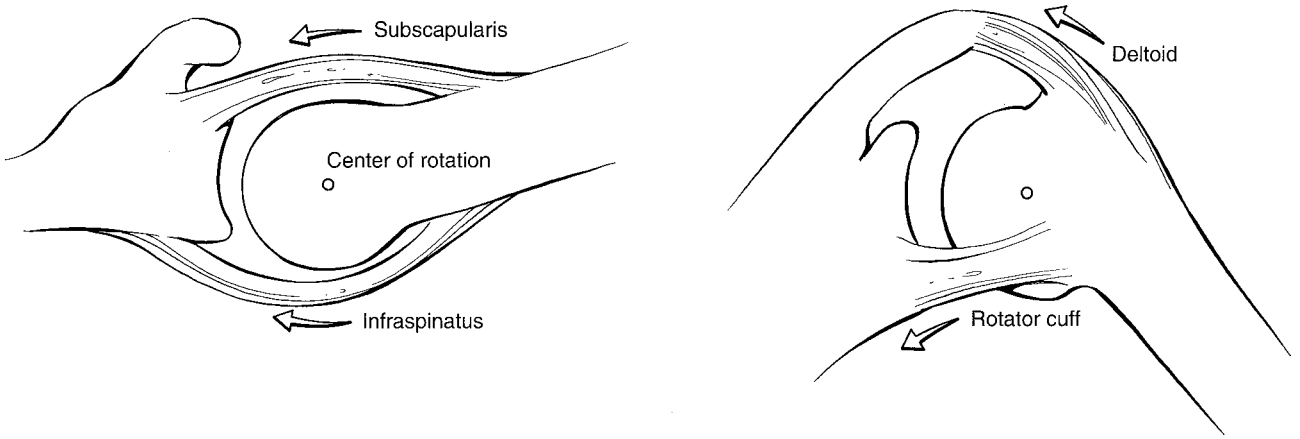

Figure 1-13 Force couples of the shoulder. **Left:** The transverse plane force couple balances the opposing forces of the anterior and posterior cuff. **Right:** The coronal plane force couple consists of the deltoid counterpoised against the inferior portion of the rotator cuff. (From Burkhart SS. Fluoroscopic comparison of kinematic patterns in massive rotator cuff tears. A suspension bridge model. *Clin Orthop* 1992;284:144–152, with permission.)

external rotation enhanced the ability of the upper portions of the infraspinatus and subscapularis, respectively, to abduct the arm. The data help to partly explain how a supraspinatus defect may not necessarily limit functional abduction of the arm.

ROTATOR CUFF DISEASE

Rotator cuff disease is characterized by specific signs, symptoms, and altered anatomy. It has many causes, which will be covered later in this chapter. Clinically patients can present with complaints of pain, weakness, or a combination of both. Radiographically, cystic changes may be seen along the superior aspect of the greater tuberosity with sclerosis or erosion on the undersurface of the acromion from pathologic contact. Cystic changes can also be seen (more easily with MRI) in the lesser tuberosity from coracoid impingement. MRI is useful in defining the extent of the rotator cuff disease and is very sensitive and specific.

Cuff tendonitis, tendinosis, calcific tendonitis, and partial- and full-thickness rotator cuff tears are common manifestations of the disease.

Rotator Cuff Tears

Tears of the rotator cuff typically involve the supraspinatus tendon and, to a variable degree, they will often include the posterior cuff. The subscapularis tendon, although less frequently involved, may be easily overlooked if not considered during diagnostic evaluation.

A universally accepted classification scheme for rotator cuff disease does not exist, making studies evaluating the results of surgical treatment difficult to compare. However, important parameters to consider when describing rotator cuff lesions include the duration, depth, and size of a tear, as well as the condition of the muscle and the tendon. Tears may be acute which typically present with the sudden onset of pain and dysfunction after a traumatic event. Chronic tears are present for longer than 3 months and may be associated with a variable degree of weakness and discomfort. Some patients may have a previously documented chronic rotator cuff tear and develop an acute extension of the lesion after a traumatic insult.

The depth of a tear will differentiate partial- from full-thickness lesions. Moreover, partial-thickness tears can be present on either the articular or bursal surface, or they may be intrasubstance. The thickness of the lesion may also vary until it extends through the entire tendon, at which point the subacromial space communicates with the glenohumeral joint. Gradation of partial-thickness lesions has been described; however, difficulty lies in the definition and accurate assessment of such lesions. For example, fraying of the tendon observed during an operation may be considered as a partial tear by some surgeons and not by others. Moreover, the incidence of such lesions in relation to symptoms and the results of treatment are not easily determined because of variability in imaging capabilities, interpretation, and lack of uniformity in classification.

Full-thickness tears may be described as small (less than 2 cm in diameter), medium (2 to 4 cm), large (4 to 5 cm), or massive (more than 5 cm). Additionally, a torn rotator cuff may be retracted, deficient, attenuated, or friable at the time of surgical assessment. The muscle can be best assessed with the aid of quality MRI scans on which cross-sectional area, degree of fatty infiltration, and alterations in overall muscle signal intensity can be determined. MRI investigation looked into the correlation of supraspinatus muscle atrophy in association with rotator cuff tears and residual function using EMG analysis. As the supraspinatus

became more atrophic, the EMG function of the muscle decreased.[163] Although these observations suggest a decrease in function in conjunction with MRI-evident supraspinatus atrophy, the influence of these findings on outcome after surgical repair of the rotator cuff will require further study.

Tears involving the rotator interval, although less common, have also been described.[135,253] Surgical exploration of this interval in 116 patients with supposed isolated tears of the supraspinatus tendon revealed occult lesions of the coracohumeral ligament, superior glenohumeral ligament, and upper border of the subscapularis in 19 cases. Treatment consisted of a rotator cuff repair and reconstruction of the rotator interval complex.[253] Although these lesions can be difficult to diagnose, their association with pain or dysfunction and the optimal method for their treatment remain undetermined.

Glenohumeral abnormalities have also been associated with tears of the rotator cuff. A prospective series of 100 patients with full-thickness tears who underwent a diagnostic arthroscopy revealed glenohumeral abnormalities in 74% overall. Common observations included lesions of the anterior labrum in 62%, intraarticular biceps tendon tears in 16%, and articular cartilage abnormalities in 28%.[157] Clearly, such lesions are being detected with increased frequency with the more widespread application of arthroscopic techniques.

Tearing of the rotator cuff may also result from differences in the mechanical properties of the bursal and articular surfaces of the tendon. Nakajima and coworkers[164] performed histologic and biomechanical analyses of the rotator cuff tendon in 20 autopsy specimens. The bursal layer demonstrated distinct tendon bundles that were more resistant to applied tensile loads. In contrast, tendon fibers of the articular surface were thinner, variable in their architectural arrangement, and more susceptible to tearing under tension. Fukuda and colleagues[77] histologically studied intratendinous tears of the rotator cuff in surgical specimens and implicated shear between the bursal and joint layers in the pathogenesis of observed lesions. Although such investigations provide further insight into potential underlying causes of rotator cuff tears, it is unlikely that one mechanism can explain the different types of tendon abnormalities. Additional study will be required to help define the causes of the disparate lesions observed.

Etiology and Pathogenesis of Rotator Cuff Disease

Multiple etiologic factors have been associated with the development of rotator cuff disease (Table 1-1). Tendon degeneration, vascular factors, impingement, trauma, glenohumeral instability, scapulothoracic dysfunction, and congenital abnormalities all appear to contribute, in some combination, to the formation and progression of rotator cuff lesions. Recognition of such processes will lead to an increased ability to effectively diagnose and treat patients who present with symptoms related to the rotator cuff. Controversy continues to exist concerning the pathogenesis of rotator cuff disease. The heterogeneity of the disorder, as well as the notion that rotator cuff disease may not actually represent a continuum of the same process but rather is a compilation of independent disorders, may partly explain the differing viewpoints on its origin.

Age-Related Degeneration or Senescence

In early anatomic investigations, Codman and Akerson[50] had suggested that degenerative processes, in association with trauma, were responsible for the genesis of rotator cuff tears. He noted that tendinous defects within the supraspinatus were commonly found 1 cm medial to its insertion on the greater tuberosity. The frequency of his findings increased with age. Subsequently, other anatomic investigations provided further evidence of degenerative changes about the shoulder with advancing age.[49,50,57,61,62,95,100,129,183,258]

Senescent changes of the rotator cuff likely occur in a manner similar to that of other joints in the body.[46,61,156] Through anatomic studies, DePalma[61,62] detailed these changes in the glenohumeral joint and rotator cuff. Observations included tearing of the cuff tendon and synovium, which became more pronounced with each successive decade of life. Moreover, DePalma and others[61,62,100,129,168,198] noted that partial-thickness tears typically begin to occur between 40 and 60 years of age and are also more frequent in older persons. Brewer[27] studied the rotator cuff in autopsy specimens and observed age-related changes, including a loss of cellularity, disorganization, and fragmentation, that led to dissolution of the cuff in older subjects. It is difficult to determine whether degenerative processes are directly responsible for observed lesions or whether they predispose the rotator cuff to tearing through alternative mechanisms. Additionally, although cadaveric age-related rotator cuff findings suggest that partial-thickness lesions develop into full-thickness tears, sufficient data in support of this concept are lacking.

Vascular Factors

The relation between the microvascular blood supply of the rotator cuff and tendon degeneration remains a subject of debate. Conflicting reports describing the vascularity of the supraspinatus tendon exist; however, in many investigations, the methods employed were limited. Moreover, although vascular-mediated mechanisms have been suggested as an important factor in the genesis of rotator cuff disease, studies have been unable to sufficiently attribute hypovascularity as a direct cause for observed tears of the rotator cuff.

TABLE 1-1
FACTORS CONTRIBUTING TO PATHOGENESIS OF ROTATOR CUFF DISEASE

Traumatic factors
Rotator cuff
Acute high-velocity trauma (acute partial- or full-thickness tears)
Repetitive low-velocity microtrauma (overuse, athletic, or work-related syndromes)
Supraspinatus outlet
Acromioclavicular separation
Coracoid nonunion or malunion
Greater tuberosity malunion
Acromial malunion or nonunion
Degenerative factors
Proliferative and degenerative changes of the acromion, coracoacromial ligament, acromioclavicular joint, or greater tuberosity
Intrinsic degenerative changes of the rotator cuff
Dystrophic calcification
Developmental factors
Os acromiale
Coracoid malformation
Type II or type III acromial morphology
Low-lying acromioscapular angle
Capsuloligamentous factors
Instability
Traumatic, unidirectional
Atraumatic, multidirectional
Capsular contracture
Tight posterior capsule
Scapulothoracic neuromuscular dysfunction
Chronic cervical spondylosis
Serratus anterior palsy (long thoracic nerve injury)
Trapezius nerve palsy (spinal accessory nerve injury)
Scapulofascial muscular dystrophy
Scapulohumeral neuromuscular dysfunction
Entrapment syndromes
Axillary nerve
Suprascapular nerve
Inflammatory disease
Calcific tendonitis or bursitis
Rheumatoid arthritis
Crystal-induced arthropathy
Iatrogenic or acquired disorders
Hardware placement
Foreign materials
Inferior placement of the humeral prosthesis
Corticosteroid-induced tendonopathy

From Iannotti JP, ed. *Rotator cuff disorders: evaluation and treatment. American Academy of Orthopaedic Surgeons Monograph Series.* Park Ridge, IL: American Academy of Orthopaedic Surgeons, 1991:2, with permission.

Mayer[151] initially dismissed the view that tendons were avascular and showed that they receive a vascular supply from three sources: muscular, osseous, and direct tendinous sites. Codman[49,50] subsequently proposed that a critical portion existed within the distal rotator cuff tendon predisposing it to degeneration and calcification. He suggested a vascular or ischemic mechanism, which, in association with trauma, leads to tearing of the rotator cuff. McMaster[154] further supported this concept and showed that when normal musculotendinous specimens were placed under mechanical loads, the muscle became the initial point of failure. However, if the tendon was compromised secondary to an interrupted blood supply and repeated stress, it then became the initial site of failure while the integrity of the muscle was preserved.

Several investigators have observed a decrease in vascularity of the rotator cuff tendon. Notably, Lindblom[141] performed histologic analysis of 12 cadaveric shoulders and demonstrated a region of hypovascularity of the rotator cuff tendon near its insertion to the greater tuberosity. Rothman and Parke[208] also confirmed a region of relative hypovascularity within the distal rotator cuff tendon after arterial latex injection into 72 cadaveric shoulders. They noted consistent vascular patterns, which were independent of age, in their specimens; however, they felt that their methods were not sufficiently accurate to reflect age-related changes. Brooks and colleagues[28] performed postmortem quantitative histologic evaluations of the rotator cuff that showed a decrease in vessel number, size, and percentage of tendon occupied by vessels in both the distal supraspinatus and infraspinatus tendons. Because the hypovascular zone was not isolated to just the supraspinatus tendon, the authors concluded that other factors must be important in the pathogenesis of rotator cuff disease. This was further confirmed by the difference in observed frequency of tears of the supraspinatus and infraspinatus tendons.

In contrast, other reports failed to show similar decreases in vascularity of the rotator cuff tendon. Moseley and Goldie[161] performed microinjection studies on 72 cadaver shoulders and demonstrated a "watershed" area or zone of anastomoses between osseous and tendinous vessels supplying the rotator cuff tendon. They termed this region the "critical zone," which was 1 cm proximal to the rotator cuff insertion and was previously noted by Codman[49,50] to be the frequent site of rotator cuff tendon failure. Their findings were also independent of age.

Rathbun and Macnab[202] used both microangiographic and histologic techniques to evaluate the tendon vascularity of the rotator cuff in cadavers. They showed adequate vascularity when injections were performed on the abducted arm, but in the adducted arm the vessels were "wrung out" and no longer perfused. They hypothesized that the critical zone was subject to a transient hypovascularity that was mediated by position of the arm.

More recently, a vascular study in 18 anatomic specimens revealed differential vascularity between the bursal and articular surfaces of the rotator cuff. The bursal surface was observed to be well vascularized, whereas the articular surface demonstrated a sparse arteriolar pattern.[145] The authors concluded that deficient tendon vascularity differentially predisposed the articular surface of the rotator cuff to degenerative changes and failure.

Intraoperative laser Doppler flowtometry has also been used to assess rotator cuff tendon vascularity in symptomatic patients. One investigation was aimed at clarifying the discrepancy between surgical findings of increased vascularity in patients with impingement syndrome and previous cadaveric reports demonstrating a hypovascular zone within the supraspinatus tendon. The patients with tendinitis and intact tendons always demonstrated increased vascularity in the area of greatest mechanical impingement, or the critical zone. Increased vascularity was also observed at the tendon margins of those patients with partial-thickness tears. Patients with complete tendon tears had variable degrees of vascularity at the tendon edges. The authors concluded that impingement generates a hypervascular response that results in the resorption of injured tendon fibers by neovascular tissue and mediates the progression of rotator cuff disease.[233] These findings also corroborate an earlier cadaveric investigation in which an increase in the number of blood vessels was observed in areas of degenerative tendon. These vessels invaded degenerative tendons through ingrowth of granulation tissue from both the bursal and synovial joint surfaces; this was never observed in normal tendons. Interestingly, in normal tendons, the number of arterioles was noted to decrease with age.[258]

Despite the findings of earlier microinjection studies, many of the conclusions are limited by the inherent shortcomings of the methods used. Lack of small-vessel perfusion in a cadaveric specimen may not satisfactorily reflect vascular characteristics in vivo. Moreover, microinjection studies that do not use histologic techniques may not afford adequate assessment of tissue vascularity, for capillary networks cannot be identified. In addition, postmortem investigations lack the clinical correlation necessary to attribute specific findings to clinical symptoms. With further study, use of in vivo methods, and employment of microscopic tissue evaluation, some of the limitations of earlier reports can be overcome and may permit a more detailed assessment of tendon vascularity in relation to clinical findings.

Impingement and the Coracoacromial Arch

Impingement

Neer[166,167] initially popularized the concept of impingement syndrome, noting that the rotator cuff was potentially

subject to repeated mechanical insult by the coracoacromial arch during elevation of the arm. His observations highlighted the anterior functional arc of shoulder motion, with resultant impingement of the rotator cuff by proliferative spurs and excrescences extending from the anterior third of the acromion and coracoacromial ligament. This was in contrast with impingement by the lateral acromion, as had been generally accepted.[10,223] He subsequently described three stages of the impingement syndrome that exist as a continuum, ultimately leading to tears of the rotator cuff. Stage I, characterized by subacromial edema and hemorrhage, was typical in symptomatic patients younger than 25 years of age. Stage II included fibrosis and tendinitis and was more common in persons 25 to 40 years old. With continued progression, stage III, or rotator cuff failure, would result and was characterized by partial or complete tendon tears typically in persons older than 40 years of age. He attributed 95% of all rotator cuff lesions to primary mechanical impingement.

Some biomechanical investigations have validated Neer's observations implicating the anterior acromion as a source of impingement.[36,69,75,148,165,166] Burns and Whipple[36] studied the anatomic sites of tendon compression against the coracoacromial arch. In the neutral arm position, the supraspinatus and intertubercular portion of the biceps tendon lies inferolateral to the coracoacromial ligament and anterior to the acromion. Arm elevation in the scapular plane resulted in contact of the supraspinatus and the anterior acromion and coracoacromial ligament. Biceps tendon impingement, on the other hand, occurred predominantly against the lateral free edge of the coracoacromial ligament. Flatow and colleagues,[75] using stereophotogrammetry to evaluate subacromial contact areas with arm elevation, noted a progressive decrease in the acromiohumeral interval with scapular plane abduction. The humerus and acromion were at their closest proximity between elevations of 60 and 120 degrees. Moreover, contact and proximity were observed to begin at the anterolateral aspect of the acromion at 0 degrees elevation and shift medially with progressive arm elevation. Only the anterior aspect of the acromion demonstrated the potential for subacromial contact. Additionally, acromiohumeral distances were decreased in shoulders with a hooked acromion morphology. In a subsequent investigation, Bigliani and colleagues[20] studied the effect of anterior acromioplasty on subacromial contact in seven cadaveric specimens. Their data suggested that flattening of the anterior third of the acromion was required to eliminate impingement. Although inherent limitations in a cadaveric model exist, they did note that flattening of just the anterior ridge of the acromion, rather than the anterior third, was insufficient to eliminate impingement in 50% of specimens.

Jobe and associates[124,125] popularized the concept of secondary mechanical impingement in throwing athletes. They noted that rotator cuff lesions are the end result of a continuum that progresses from instability, subluxation, impingement, and tension overload of the cuff, with resultant tearing. Repeated mechanical stresses cause failure of the glenohumeral static restraints and place increased demands on the dynamic stabilizers. The rotator cuff eventually fatigues, resulting in abnormal translation of the humeral head and secondary impingement. If left untreated, the impingement can progress and cause tearing of the cuff. The ability to distinguish between primary and secondary impingement, as described by Neer and Jobe, respectively, is paramount to effectively treating patients affected with these disorders. Although satisfactory results have been reported for subacromial decompression in patients with primary impingement, the same does not hold true for individuals with the secondary type.[3,70,81,93,102,108,196,216,229,235,239,250] Rather, treatment in this group requires attention to the underlying instability.

Coracoacromial Arch

The coracoacromial arch marks the superior boundary of the subacromial space. It comprises the coracoid process, the coracoacromial ligament, and the acromion (see Fig. 1-7). Mechanical forces about the coracoacromial arch, while not fully understood, have also been linked to the development of rotator cuff disease.[77,110,164,224]

The Acromion

The shape of the acromion exhibits variability among individuals. In an anatomic study of 140 cadaveric shoulders, Bigliani et al.[22] identified three predominant acromial forms when assessed in the sagittal plane (Fig. 1-14). A type I acromion had a flat undersurface and was present in 17% of cases. A type II acromion revealed a curved undersurface and was found in 43% of specimens. A hooked acromion, or type III, although present in 39% of cases, was found in 70% of shoulders with observed tears of the rotator cuff. A follow-up clinical study, using supraspinatus outlet radiographs to assess acromial morphology and arthrograms to determine rotator cuff integrity, affirmed the association between a "hooked" acromion and the presence of rotator cuff tears in patients presenting with various shoulder complaints.[160]

Despite the potential value of correlating specific acromial forms with lesions of the rotator cuff, other investigators have been unable to demonstrate comparable findings and have questioned the reliability of radiographic acromial morphology assessment in the sagittal plane.[13,16,72,96,120,268] Moreover, even though a flat acromion could be readily detected by most clinicians, confusion has arisen with regard to discernment of the curved (type II) and hooked (type III) patterns. Jacobson and colleagues[120] studied 126 supraspinatus outlet radiographs in an attempt to determine the reliability of detecting acromion

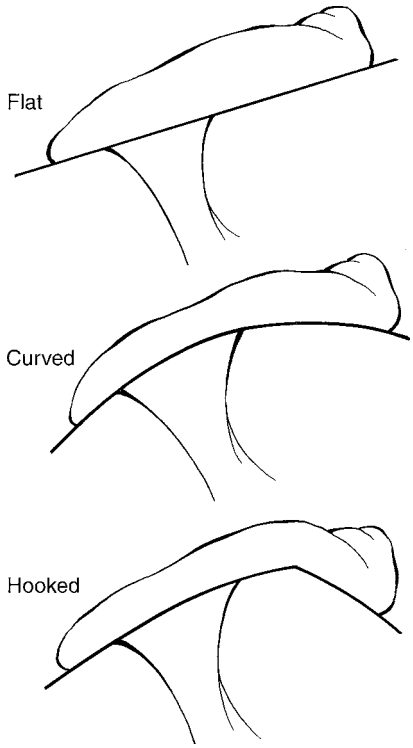

Figure 1-14 Acromion morphology: **type I:** flat; **type II:** curved; **type III:** hooked. (Adapted with permission from Bigliani LU, Morrison DS, April EW. The morphology of the acromion and rotator cuff impingement. *Orthop Trans* 1986;10:288.)

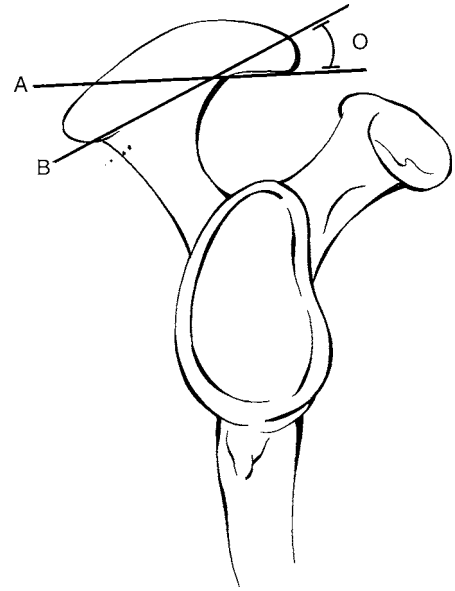

Figure 1-15 Depiction of acromial angle. (From Toivonen DA, Tuite MJ, Orwin JF. Acromial structure and tears of the rotator cuff. *J Shoulder Elbow Surg* 1995;4:376–383, with permission.)

morphology. Inter- and intraobserver reliability coefficients of 0.516 and 0.888 were demonstrated, respectively. The data suggest that observers were each consistent in using a given set of classification criteria, but that these criteria differed among examiners. Furthermore, they also reflect the qualitative component of acromion morphology assessment and the potential difficulties in comparison of published studies. Some of the observed differences may best be explained by the potential for acromion architecture to exist as a continuum, ranging from a flat to a hooked configuration, with varying degrees of curvature within the extremes.

In an effort to objectively quantitate and standardize the classification of acromion morphology, Toivonen and coworkers[243] devised the measurement of an "acromial angle." This angle was formed between two lines drawn along the undersurface of the anterior third and posterior two-thirds of the acromion (Fig. 1-15). The authors reported reproducible methods and demonstrated a significant association between increasing acromial angle and rotator cuff tears. The types I, II, and III acromion had acromial angles of 0 to 12, 13 to 27, and greater than 27 degrees, respectively. Moreover, their findings were consistent with those of Bigliani et al.[22] in that 89% of type III acromions were associated with tears of the rotator cuff.

Kitay and associates[132] called into question the use of supraspinatus outlet radiographs alone in evaluating acromial morphologic condition. Analysis of anteroposterior, axillary, 30-degree caudal tilt and supraspinatus outlet views in 23 surgically treated patients with impingement syndrome revealed the greatest interobserver reliability for the 30-degree caudal tilt view (0.84; Fig. 1-16). The caudal tilt x-ray film reflected the acromial spur in its combined anterior and inferior projections, whereas the outlet view provided information on the inferior projection of the spur. The combined use of these two radiographs was believed to be the best predictor of intraoperative acromial spur size.

The effect of age on acromial morphologic condition has not been sufficiently studied, leaving many questions unanswered about the potential for developmental alterations in acromial shape. Nicholson and coworkers[178] attempted to address this issue by quantifying osseous dimensions of the acromion and evaluating the relation between morphologic condition and age. Analysis of 420 scapulae in different age groups revealed no trends toward the alteration of acromial morphology. Although age did correlate with an increase in frequency of anterior acromial spur formation, it did not significantly change the dimensions of the acromion or alter morphology when assessed using supraspinatus outlet radiographs. The data suggest that the acromion's morphologic condition functions both independently and in association with age-related degenerative processes in the development of rotator cuff disease.

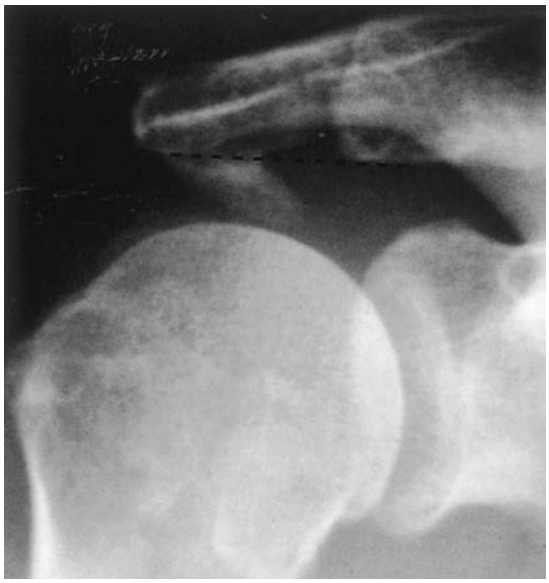

Figure 1-16 Thirty-degree caudal tilt radiograph depicting anterior–inferior projection of an acromial spur. The *solid line* designates the anterior cortical margin of the distal clavicle.

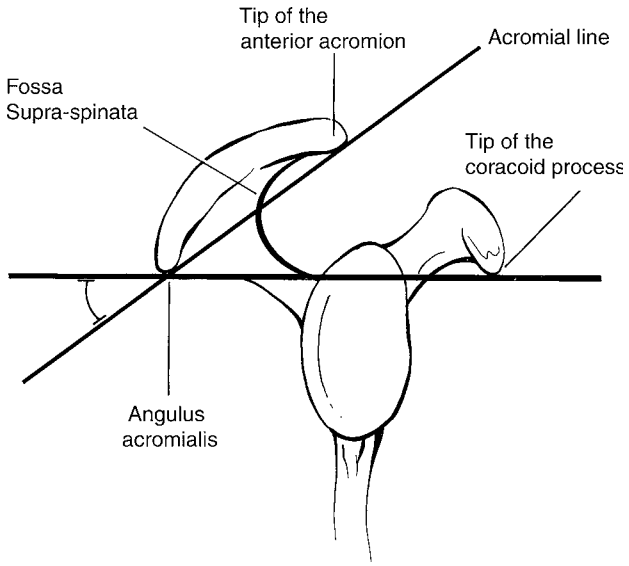

Figure 1-17 Acromial slope (tilt). This angle reflects the pitch of the acromion and is formed by a line across the posterior acromion and coracoid tip and along the undersurface of the acromion. (From Aoki M, Ishii S, Usui M. Clinical application for measuring the slope of the acromion. In: Post M, Morrey B, Hawkins R, eds. *Surgery of the shoulder.* St Louis: Mosby-Year Book, 1990: 200–203, with permission.)

Neer and Poppen[169,172] suggested that both the slope of the acromion and acromioclavicular joint spurs can compromise the integrity of the rotator cuff through impingement mechanisms. Aoki and coworkers[8,9] measured acromial slope and found a decreased angle in patients with impingement syndrome when compared with normal controls. Qualitatively, this parameter refers to the pitch of the acromion in the sagittal plane. A more horizontal acromion would have a lower pitch and a corresponding low angle (Fig. 1-17). This lower pitch can result in a reduced area of the supraspinatus outlet, thereby creating the potential for rotator cuff compromise.[268]

Zuckerman and colleagues[268] studied the spatial anatomy of the coracoacromial arch and supraspinatus outlet as they relate to full-thickness tears of the rotator cuff. In an anatomic investigation of 140 shoulders, they demonstrated a significant association between rotator cuff tears and measured parameters, including a reduced supraspinatus outlet area, lower acromial tilt, and larger anterior projection of the acromion. These findings support the contention that elements other than sagittal plane acromial morphology can be important factors leading to disorders of the rotator cuff. Their work was further supported by Edelson and Taitz,[67] who, in an anatomic study of 200 scapulas, observed that acromial slope, length, and height were most closely associated with osseous degenerative changes of the coracoacromial arch. Although such factors have also been implicated in the pathogenesis of rotator cuff disease, standard acromioplasty may not adequately address these elements and may explain failed surgical treatment in specific cases.

Attention to acromial morphology in other planes has also become an area of increasing interest. Banas and colleagues[13] described the "lateral acromion angle" after retrospectively reviewing 100 shoulder MRI scans in symptomatic patients. This angle is formed by a line along the undersurface of the acromion, as viewed in the coronal plane, and a second line joining the most superior and inferior margins of the glenoid (Fig. 1-18). As the lateral acromion angle decreased, a statistically significant increase in rotator cuff disease was observed. This parameter was felt to be an independent predictor of rotator cuff disease and further highlights the importance of acromial morphology assessment in multiple planes.

Ozaki and coworkers[189] looked at the relation between anatomic changes of the acromion undersurface and pathologic findings within the rotator cuff. Histologic and radiographic evaluation demonstrated an association between bursal-side partial rotator cuff tears and abnormalities of the acromion undersurface. Changes on the acromion correlated with the severity of the bursal tear, and the prevalence increased with advancing age. Interestingly, shoulders that demonstrated joint surface partial tears revealed an intact acromion undersurface. Their data further support the contention that rotator cuff tears represent a degenerative age-related process and that acromial abnormalities reflect secondary changes resulting from a bursal surface cuff tear. Whether precarious tendon vascularity or mechanical insult by the overlying coracoacromial arch also mediate this process remains undetermined;

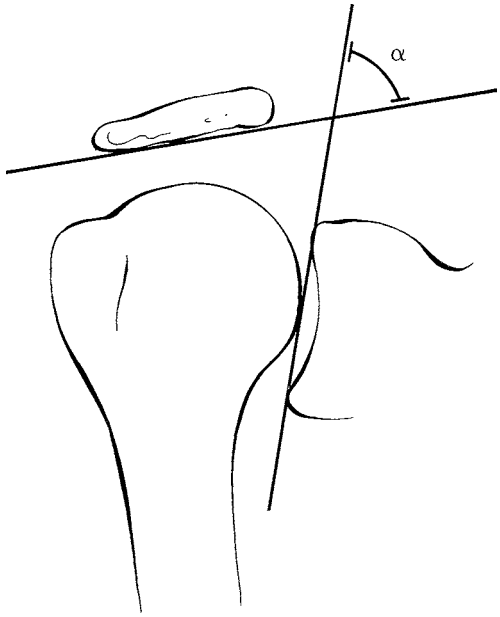

Figure 1-18 Lateral acromion angle. This angle is formed by the intersection of a line along the undersurface of the acromion in the coronal plane and a second line connecting the superior and inferior margins of the glenoid. (From Banas MP, Miller RJ, Totterman S. Relationship between the lateral acromion angle and rotator cuff disease. *J Shoulder Elbow Surg* 1995;4:454–461, with permission.)

however, it seems conceivable that both factors play a contributing role.

The Coracoacromial Ligament

The contribution of the coracoacromial ligament alone in the impingement syndrome has been investigated. It has been suggested that thickening of this ligament may predispose some patients to shoulder impingement. Uhtoff and coworkers[245] performed histologic analysis of the coracoacromial ligament in 17 patients with painful arc syndrome. Although they observed diffuse degenerative changes within the ligament, excessive proliferation of fibrous tissue could not be identified. Impingement in these patients was thought to occur as a result of expansion of the volume of the rotator cuff tendon or bursa, rather than the ligament itself. They highlighted the concept that any process that causes a decrease in the volume of the supraspinatus outlet can result in impingement syndrome. The coracoacromial ligament was believed to be one component of an unyielding tunnel that becomes too restrictive for its expanded contents.

While commonly described as having an inverted "Y" configuration, other morphologic types have been noted.[113,213] Holt and Allibone[113] performed an anatomic and histologic analysis of the coracoacromial ligament in 50 shoulders and noted variable forms among individuals. They observed three predominant variants and described them as "quadrangular, Y-shaped or broadbanded" (Fig. 1-19). The quadrangular and Y forms demonstrated a frequency of 48% and 42%, respectively. In the Y type, the two limbs take origin from the medial and lateral aspects of the coracoid. As they project superolaterally to insert onto the undersurface of the acromion, the bands fuse and form the anterior soft tissue boundary of the coracoacromial arch (Fig. 1-20).

Biomechanical and geometric testing of the coracoacromial ligament has demonstrated that the lateral band was both shorter in length and smaller in cross-sectional area in shoulders with rotator cuff tears. Although histologically there were no structural differences in the ligament between normally formed shoulders and those with rotator cuff tears, there was evidence of decreased mechanical properties in the latter. The reduction in mechanical integrity of the ligament was thought to reflect the multiple directional loads imposed on this structure in shoulders with rotator cuff tears.[224] One additional investigation employing scanning electron micrographs in eight cadaveric shoulders demonstrated that observed degenerative changes of the rotator cuff were characteristic of alterations resulting from frictional and rubbing mechanisms. Observations support the contention that degenerative changes already present in the cuff, irrespective of cause, can be aggravated by proposed frictional or abrading type forces.[110] Additional reports have suggested that increases in measured subacromial pressures in patients with impingement syndrome may mediate the development and progression of rotator cuff disorders.[121,222,262]

In contrast with its implicated role in the pathogenesis of impingement syndrome, the coracoacromial ligament has been shown to function as a dynamic brace that lends support to the acromion and coracoid during loads imposed by the surrounding musculature. Putz and colleagues[201] used strain gauges to measure distortion of the coracoid and acromion after resection of the coracoacromial ligament in eight cadavers. With applied loads, significantly more distortion of the acromion was observed. Soslowski and associates[228] have noted that the coracoacromial arch acts as a buffer against superior translations when the humeral head is not centered in the glenoid. This may occur during normal obligate translation of the joint with humeral rotation and also in patients with glenohumeral instability. Furthermore, its role as a secondary restraint to anterosuperior migration of the humeral head in patients with large rotator cuff deficiencies has also been emphasized.[11,74,97,228,257] Salter and coworkers[213] have suggested that the coracoacromial ligament provides mechanical support to the acromioclavicular joint. Gross and microscopic anatomic evaluation revealed that fibers at its insertion under the anterior acromion were contiguous with the inferior acromioclavicular joint capsule.

Although such studies have provided quantitative analysis of the coracoacromial arch in relation to shoulder function,

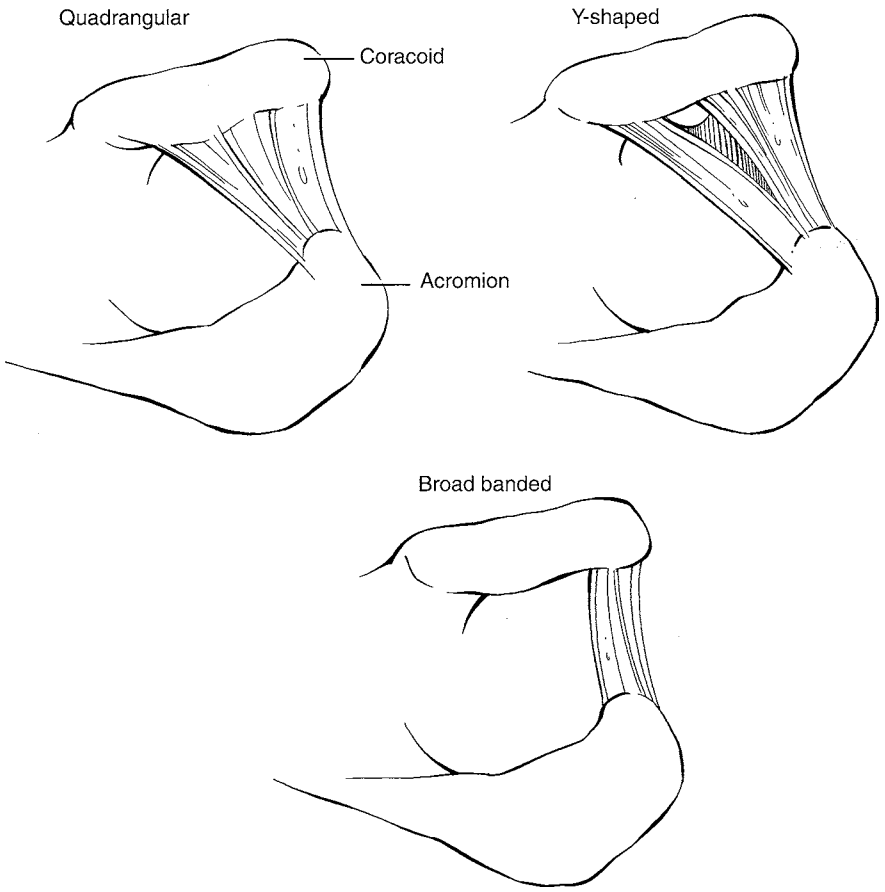

Figure 1-19 Anatomic variants of the coracoacromial ligament. (From Holt EM, Allibone RO. Anatomic variants of the coracoacromial ligament. *J Shoulder Elbow Surg* 1995;4: 370–375, with permission.)

additional investigations are needed to further clarify subacromial stresses and contact areas in multiple planes of motion and in different pathologic states. Increased understanding of these issues may enhance our ability to effectively evaluate and treat patients with rotator cuff disease.[128]

Others have noted that coracoacromial ligament division alone may be sufficient for adequate decompression of the subacromial space in selected patients.[126,256] One report highlighted sectioning of the ligament in a subgroup of persons with clinical findings of impingement

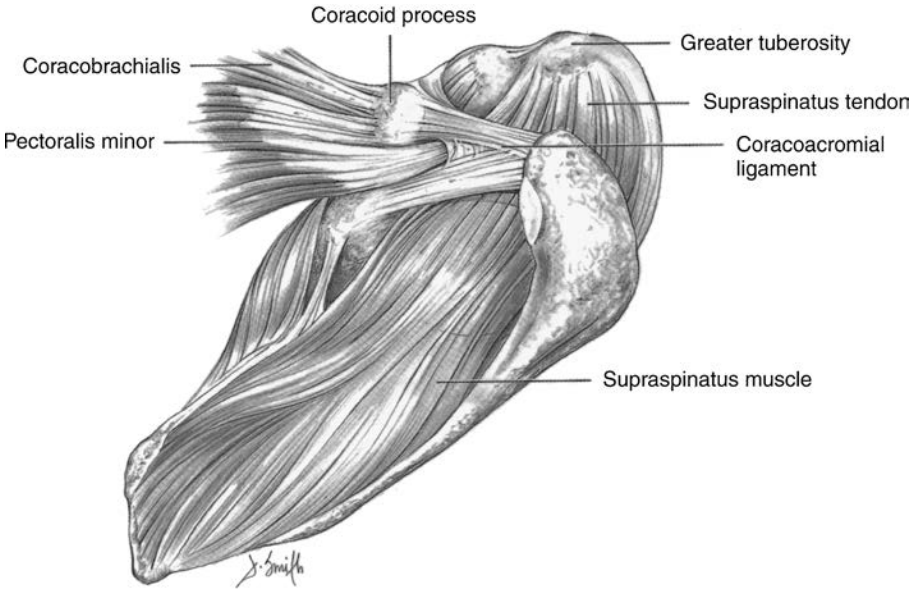

Figure 1-20 The coracoacromial arch. Note the Y configuration of the coracoacromial ligament.

syndrome despite an absence of degenerative spur formation and the presence of a flat-appearing acromion.[126] Although satisfactory results were reported for most patients, the role of coracoacromial ligament division alone in such patients has not been sufficiently studied. Furthermore, although this structure has been implicated in the pathogenesis of rotator cuff disease, recent emphasis has focused on its important functional role as a secondary passive restraint to anterosuperior migration of the humeral head in cuff-deficient patients.[74,97,257] Questions on the benefits of limited subacromial decompression remain unanswered and continue to be an area of active investigation.

Subacromial Spurs

The distinction between native acromion morphology and developmental subacromial osseous excrescences has not always been clear.[36,47,181,189] Although a strong association between degenerative subacromial hypertrophic spur formation and full-thickness tears of the rotator cuff exists, a causal relation remains difficult to prove.[22,50,78,137,156,160,166,168,181,183,189,206] Proponents of intrinsic pathogenic mechanisms support the contention that subacromial spurs represent secondary changes occurring as a result of existing tears of the rotator cuff.[189] Conversely, others have suggested that observed lesions of the rotator cuff occur because of mechanical insult by inferiorly projecting subacromial bony excrescences.[166,168] The formation of such spurs has been suggested to occur as a result of repeated tension exerted on the coracoacromial ligament.[106,181]

Despite controversy over the initial lesion, subacromial spurs appear to have a role in the development and progression of commonly observed rotator cuff tears. One histologic evaluation of bursal side rotator cuff tears in surgical specimens revealed variable-thickness tears of the supraspinatus corresponding with areas of impingement of the overlying acromion and coracoacromial ligaments. Also observed were avascular regions of the proximal edge of the torn tendon. The combination of findings led the authors to conclude that multiple causes, including both intrinsic and extrinsic causes, were responsible for the observed abnormalities.[78]

Coracoid and Acromioclavicular Osteophyte Impingement

Alternative sources of impingement have been implicated in the development of rotator cuff disease. Distally pointing acromioclavicular osteophytes, the coracoid process, and the posterosuperior aspect of the glenoid can contribute to shoulder pain and rotator cuff lesions in certain patients.[59,64,84,85,98,123,133,143,166,168,169,172,190,194,217,234,242,252,254] Petersson and Gentz[194] studied the relation between distally pointing acromioclavicular osteophytes and ruptures of the supraspinatus tendon. By using radiographic analysis in patients with arthrographically confirmed rotator cuff tears and anatomic dissections in cadaveric subjects, they were able to demonstrate a strong association between ruptures of the supraspinatus tendon and periarticular osteophytes. Although acromial excrescences were also observed, their frequency in subjects with rotator cuff ruptures was less than that of acromioclavicular bone spurs. Seeger and coworkers[217] reviewed MRI scans in 107 patients with painful shoulders. Bony and soft tissue abnormalities clinically described in impingement syndrome were evident in 53 persons. In these cases, the supraspinatus was noted to be compressed by either osseous spurs, hypertrophic capsular tissue of the acromioclavicular joint, or a low-lying acromion.

Whereas abnormalities of the acromioclavicular joint have been associated with rotator cuff disorders, acromioclavicular joint arthrosis has also been observed in 65% of asymptomatic persons with and without tears of the rotator cuff.[220] These findings suggest that acromioclavicular joint abnormalities on imaging studies alone may not be a reliable predictor of disease in the absence of correlative symptomatology. Moreover, treatment of such periarticular abnormalities based on imaging studies alone, such as excision of small inferior acromioclavicular osteophytes during subacromial decompression, may convert a painless condition to a symptomatic joint in certain patients.[134] However, patients with clinical evidence of the impingement syndrome and symptomatic arthritis of the acromioclavicular joint have been treated successfully with combined subacromial decompression and distal clavicle resection.[147]

Coracoid Impingement

Impingement of the rotator cuff between the humeral head and coracoid process can also occur with certain arm positions in some patients.[64,84,85,98,133,190] Gerber and coworkers[84] recognized the subcoracoid space (region between the tip of the coracoid process and humeral head or lesser tuberosity) as a source of shoulder pain and reported on idiopathic, iatrogenic, and traumatic causes of abnormalities affecting either the coracoid, glenoid, or humeral head. In all types, anterior shoulder pain was reproduced with either internal rotation of the arm in 90 degrees of abduction or adduction with the shoulder flexed to 90 degrees. Computed tomography scans of 47 shoulders in normal volunteers highlighted dimensional parameters of the subcoracoid space and suggested variational anatomic features that may predispose certain individuals to coracoid impingement. The subcoracoid space was confirmed as not being a free space, but rather a region just sufficient to accommodate gliding of the soft tissues between the coracoid process and humeral head. The distance between the coracoid and humerus decreased with the arm in forward

flexion and internal rotation, especially in those persons with a coracoid projecting far laterally and close to the scapular neck. Moreover, the soft tissues in the space became folded with the arm in this position. Alterations in the coracohumeral relation (e.g., osteotomy or fracture) as studied in cadaveric controls highlighted the potential for impingement with the arm in a flexed and internally rotated position.[85] Although this entity remains an uncommon form of impingement and often a difficult diagnosis, satisfactory results have been reported with surgical treatment in selected patients.[64,84]

Internal Glenoid Impingement

Arthroscopic evaluations of throwing athletes who have the painful arc syndrome have demonstrated impingement of the deep surface of the rotator cuff against the posterosuperior glenoid rim with the arm in 90 to 150 degrees of abduction and maximal external rotation.[59,122,123,143,155,242,252,254] It has been suggested that abduction and external rotation of the arm can entrap a portion of the supraspinatus tendon between the humeral head and glenoid in susceptible persons. Increased glenohumeral external rotation, decreased humeral retroversion, scapulothoracic dysfunction, and poor throwing technique all have been implicated in the development of this disorder. Associated findings include partial-thickness tearing of the undersurface of the infraspinatus and supraspinatus tendon, degenerative lesions of the posterosuperior glenoid labrum, and osteochondral impression fractures of the humeral head. Patients will typically complain of posterior shoulder pain that is elicited by overhead activity. Although these observations help emphasize alternative sources of shoulder impingement, further study is needed to better define this entity, its pathomechanics, and optimal treatment.

Acute Trauma and Rotator Cuff Tears

Most symptomatic rotator cuff tears are a result of an acute injury in the setting of some preexisting rototar cuff disease. Traumatic insults to the shoulder can result in tearing of the rotator cuff tendon. Neviaser and coworkers[177] reported on a series of 30 patients who had a concurrent rupture of the rotator cuff with an anterior dislocation of the glenohumeral joint. All patients were older than 40 years of age and were unable to sufficiently elevate the arm in the postinjury period. The supraspinatus was torn in most of the patients, with variable degrees of infraspinatus involvement. All patients with recurrent anterior instability had disruption of the subscapularis tendon. These individuals were satisfactorily treated with primary repair of the tendon without reconstruction of the capsulolabral complex. Similar findings were noted in other reports that also included disruption of the infraspinatus and teres minor tendons in a patient with recurrent posterior dislocations.[118,175,176,203]

Ruptures of the rotator cuff can occur in 14% to 63% of patients after an acute anterior or inferior dislocation.[112,149,195,203,241] The incidence increases in older persons and has been reported in 63% of patients older than 50 years of age.[203] Presumption that the rotator cuff tear is a result of the dislocation is based on negative patient accounts of shoulder pain or dysfunction before occurrence of the traumatic event.[175,176] However, it is conceivable that a tear may have been present before the injury and was extended or exacerbated after the dislocation. This may parallel patients who demonstrate acute extensions of a chronic, preexisting rotator cuff tear in the absence of instability. Although the chronicity of a potential preexisting rotator cuff lesion is sometimes difficult to determine, its influence on the prognosis and treatment should be considered. In such instances, surgical reconstruction of the rotator cuff may be more challenging than anticipated owing to the chronic component of the injury.

Fractures of the greater tuberosity, with or without a glenohumeral dislocation, can also result in tears of the rotator cuff. Neer[167] reported that a displaced greater tuberosity fracture results in an obligate longitudinal cuff tear at the region of the rotator interval. Posterior dislocations can result in a fracture of the lesser tuberosity, with disruption of the subscapularis. Interestingly, recurrent instability after fracture dislocations of the greater tuberosity is rare and is reported to range from 1% to 4%.[153,209,211]

Other forms of traumatic rotator cuff lesions include small partial-thickness tears of the supraspinatus or subscapularis in young, repetitive overhead athletes. Sports such as tennis, swimming, and baseball may predispose certain persons to rotator cuff tears through repeated mechanical stresses. One proposed mechanism suggests that fatigue of the scapular stabilizers results from repeated throwing, causing the humeral head and rotator cuff to abut against the acromion during arm elevation. The scapula is thought to "lag" behind the humerus, becoming unable to abduct sufficiently. Patients with neural impairments causing weakness of the trapezius or serratus anterior muscles can also develop secondary impingement through similar mechanisms. Loss of the suspensory mechanism of the scapula, such as in traumatic disruption of the acromioclavicular and coracoclavicular ligaments, may result in comparable rotator cuff abnormalities. In such cases, rotator cuff impingement occurs secondary to abnormal functional mechanics of the scapula.

Congenital Abnormalities: Os Acromiale

An association between an unfused acromial epiphysis, or "os acromiale," and tears of the rotator cuff has also been described.[162,170,179] The acromion has three centers of ossification that typically unite with the scapular spine by age 12

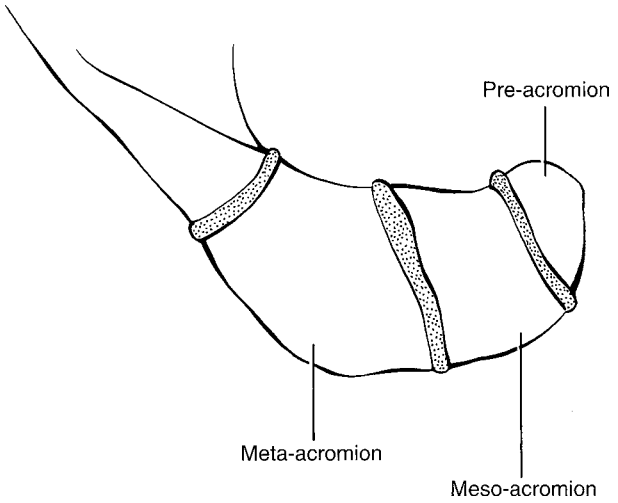

Figure 1-21 Three ossification centers of the acromion. (From Liberson F. Os acromiale: a contested anomaly. *J Bone Joint Surg* 1937;683–689, with permission.)

and with each other by ages 15 to 18[76,138,174, 232] (Fig. 1-21). These three centers are designated as the preacromion, mesoacromion, and metaacromion. The prevalence of os acromiale has been reported to range from 1% to 15%, with a 62% frequency of bilateral involvement.[68,99,138,178] Abnormal motion at the synostosis or synchondrosis is thought to decrease the capacity of the subacromial space and contribute to mechanical insult of the underlying rotator cuff.

An os acromiale can be identified on an axillary radiograph and should not be mistaken for a fracture of the acromion. Failure to recognize this entity can potentially compromise the results of treatment. Consensus on the optimal management is lacking and has ranged from conservative treatment to excision versus internal fixation and bone grafting.[33,115,136,162,179] Norris and associates[179] evaluated a group of 29 patients with os acromiale and suggested bone grafting in patients in whom the os was considered to be unstable. Standard acromioplasty was believed to be satisfactory in patients with a stable synostosis. Arthroscopic subacromial decompression alone has been reported to yield poor results in some patients, for symptoms were noted to recur within 1 year of surgery.[115] Whereas smaller fragments may be amenable to excision in symptomatic patients, no controlled studies exist comparing excision with internal fixation and bone grafting for meso os acromiale. Because the acromion serves as the origin for the deltoid, potential compromise of this important muscle should be considered during surgical management of affected patients.

The finding of an os acromiale may be incidental and not necessarily correlate with a patient's symptoms. Normal shoulder function has been noted in patients with an os acromiale.[33,138] Burkhart[33] reported on a high-performance tennis player who had competed without pain or dysfunction for 15 years before evaluation. Furthermore, clinical experience has demonstrated that many patients who present with shoulder pain and have an os acromiale had been asymptomatic for many years before clinical presentation. Often, a traumatic event has been presumed to precipitate symptoms surrounding the os acromiale. If the prevalence of this anomaly is estimated at 1% to 15% of the general population, then it seems likely that many individuals with an os acromiale never develop symptoms given the frequency of cases seen in a clinical shoulder practice.

NATURAL HISTORY

The rotator cuff is subject to substantial forces because it maintains the humeral head within the shallow glenoid. It is situated in a potentially tight subacromial space and undergoes senescent structural changes commonly observed in other joints of the body.[46,61,156] When the cuff fails, spontaneous healing of the torn tendon is not expected to occur, and multiple factors may be responsible. Its fibers are under tension and typically retract on tearing. In full-thickness lesions, only bursal tissue may bridge the area of tendon loss. Histologic evaluation of partial tears in surgical specimens has also demonstrated patterns of incomplete healing. Observations included neovascular tissue at the distal margin of the defect and relative avascularity of the proximal stump. Although the potential for a reparative process was felt to exist, there was no evidence of closure of the defect in any of the specimens. The findings were suggestive of a futile attempt at healing.[78] Other investigators have noted that resorption of tendon fibers by neovascular tissue can occur.[233,258] This may potentially weaken surrounding intact fibers placed under increased loads as a result of a tear. Because the torn cuff is bathed in synovial fluid, factors responsible for normal healing and formation of fibrin clots may be disrupted. Moreover, tearing may further impair the blood supply to a relatively dysvascular tendon.

The prevalence of rotator cuff tears in the general population can be extrapolated from both cadaveric and MRI studies. The frequency of complete and partial rotator cuff tears ranges from 5% to 39% and 13% to 3%, respectively.[50,79,100,129,130,168,189,194,220,258] Although anatomic studies have reported an increase in pathologic findings about the cuff with advancing age, they are limited by an inability to sufficiently correlate findings with symptomatology. Given the prevalence of rotator cuff tears in cadaveric studies alone, it remains unclear whether observed findings could be considered part of the normal aging process.

A prospective investigation was conducted to determine the prevalence of rotator cuff tears in an entirely asymptomatic population by using shoulder MRI scans in 96 normal volunteers. Overall, complete and partial-thickness

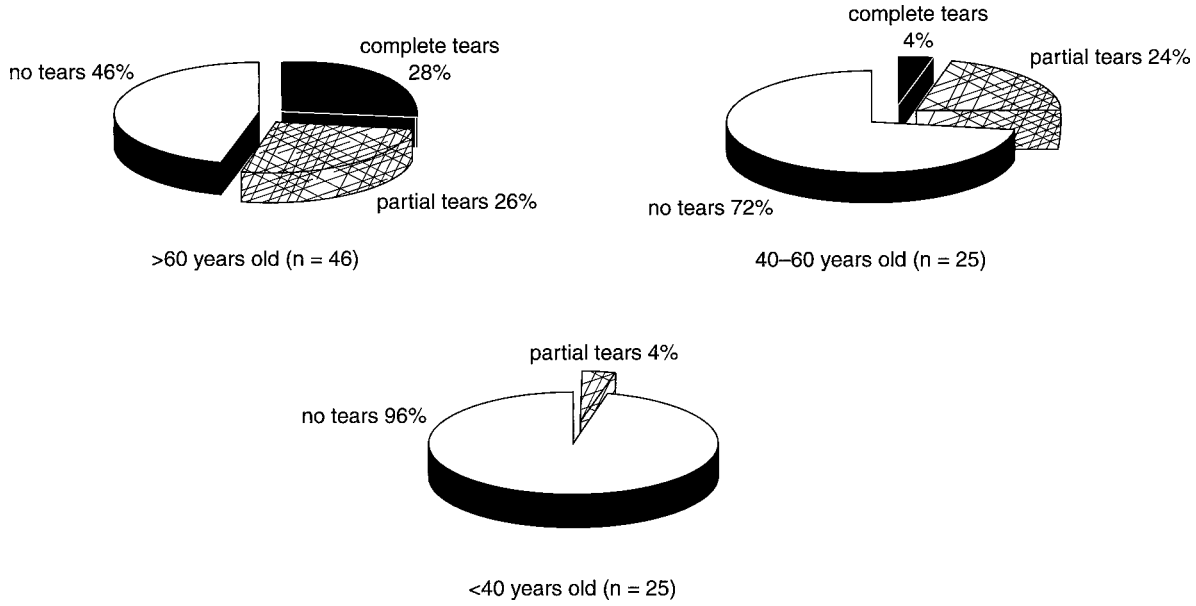

Figure 1-22 Prevalence of magnetic resonance imaging–evident rotator cuff tears in (*left*) asymptomatic volunteers older than 60 years of age, (*right*) those 40 to 60 years, and (*bottom*) those younger than 40 years. (Adapted with permission from Sher JS, Uribe JW, Posada A, Murphy BJ, Zlatkin MB. Abnormal findings on magnetic resonance images of asymptomatic shoulders. *J Bone Joint Surg Am* 1995;77:10–15.)

tears were found in 14% and 20% of individuals, respectively. In persons older than 60 years of age, the prevalence of complete and partial tears was 28% and 26%, respectively[220] (Fig. 1-22). The results provided in vivo evidence that asymptomatic individuals with rotator cuff tears can exhibit normal shoulder function. Moreover, they emphasized the dangers of basing operative decisions on MRI scans alone. The high prevalence of cuff tears in an asymptomatic population and their direct correlation with age support the contention that some rotator cuff tears occur as part of a normal age-related process.

The question of why some patients with rotator cuff tears develop symptoms and others do not remains an area of interest. Both prior reports and clinical observation affirm that many patients with cuff tears may not demonstrate significant pain or dysfunction.[25,29,152,210,214] Clearly, some individuals with symptomatic tears respond well to conservative treatment despite the persistence of a tendon defect. Yamaguchi et al. examined longitudinally the natural history of asymptomatic rotator cuff tears over a 5-year period to assess the risk factors for symptoms and tear progression. They found that 51% (23 shoulders) became symptomatic over a mean of 2.8 years and returned on their own. Of the remaining patients who returned for evaluation, only nine were asymptomatic. Of these nine, two asymptomatic tears had cuff tear progression.[265]

Other authors have documented good functional results, in the short term, in patients who had undergone débridement, rather than repair of a torn rotator cuff. Furthermore, satisfactory pain relief was predictably achieved despite lack of closure of the tendon tear.[30,136,182,205,207,269] As such lesions do not typically heal, it would seem conceivable that factors other than the tendon defect itself must contribute to the generation of symptoms. Fukuda and associates[77] suggested that subacromial bursal inflammation, as evidenced in surgical histologic specimens of partial-thickness rotator cuff tears, mediates symptoms in affected patients. They noted that the degree of inflammation likely correlates with the patient's level of symptoms. Others have suggested additional sources of shoulder pain that potentially include synovitis, intraarticular pathology, and mechanical factors.[34,69,166,168]

Alterations in normal glenohumeral kinematics have also been considered in the development of symptoms, but equally important, may be individual differences in scapular mechanics, compensatory action of surrounding muscles, and variable tolerances to pain. Multiple studies have shown abnormal superior migration of the humeral head during active arm elevation in shoulders with rotator cuff tears and the impingement syndrome.[32,63,144,197,218,264] In normal shoulders, on the other hand, the geometric center of the humeral head remains centered on the glenoid during active arm elevation.[4,197,218] Abnormal glenohumeral patterns of motion have thus been suggested to play a role in mediating pain. Burkhart[32] emphasized the concept of an anatomically deficient, but biomechanically intact, rotator cuff. Fluoroscopy was used to assess kinematics of the glenohumeral joint in patients with massive tears of the rotator cuff. Normal patterns were demonstrated, provided sufficient anterior and posterior cuff were present to preserve the normal

transverse plane force couple. Location rather than size of the tear was felt to have a more important role in preserving normal glenohumeral motion. Another biomechanical study evaluating glenohumeral motion in artificially created tendon defects highlighted the potential for normal kinematics, provided only a portion of the cuff was violated. Lesions of the supraspinatus did not alter normal motion patterns, whereas defects involving both the supra- and infraspinatus demonstrated an increase in humeral cephalad migration.[144]

An investigation attempted to address the relation between glenohumeral kinematics and symptoms in patients with rotator cuff tears. Computer-enhanced radiographic measurements were obtained during arm elevation in patients with known tears of the rotator cuff. An asymptomatic and a symptomatic population were studied, which revealed progressive superior humeral head translation in both groups when compared with normal controls.[264] These data demonstrate that loss of glenohumeral kinematics, as measured in the coronal plane, does not correlate with the presence of symptoms. More likely, symptoms are the result of multiple factors that may not necessarily be independent of one another.

The fate of the many types of cuff lesions cannot always be predicted. It is difficult to conclude that all rotator cuff lesions fall within a continuum progressing from tendinitis to full-thickness tears, because conclusive evidence supporting this concept is lacking. Rather, it seems more plausible that the types of pathology observed reflect the multifactorial cause and pathogenetic mechanisms so far identified. Moreover, we cannot definitively determine that partial tears heal, for evidence to the contrary exists.[78] The significance of bursal and joint surface partial lesions in relation to symptoms and their pathogenesis also remains uncertain.

The natural history of patients with symptomatic full-thickness rotator cuff tears is variable. Moreover, our ability to reliably predict a given patient's course is limited and may partly reflect insufficient knowledge about its pathogenesis. Clinical experience has demonstrated that patients with similar-appearing lesions may have differences in symptoms, function, and response to treatment. Cofield[51] noted that conservative management of patients with chronic painful rotator cuff tears will likely result in a successful outcome less than 50% of the time. Others have noted similar findings after nonoperative treatment and reported gradual deterioration of shoulder function with time in some patients.[117,214,259] Neer and coworkers[171] estimated that cuff tear arthropathy will develop in 4% of patients with complete rotator cuff tears. If the tear was not sufficiently large or became sealed off by bursal tissue, then development of cuff-related arthropathy was less likely. The surgical recommendations for patients with symptomatic complete tears are generally individualized and based on the duration of symptoms, severity of pain, degree of dysfunction, and functional goals. Although satisfactory results can usually be achieved with surgical treatment in many patients, isolating those individuals likely to achieve similar outcomes with nonsurgical management continues to be a challenge. The heterogeneity, lack of uniform classification, nonuniformity in treatment strategies, and existence of similar lesions in normal asymptomatic persons make it difficult to predict the likely outcome of these patients. The use of appropriate animal models and execution of large longitudinal follow-up studies can help further identify prognostic criteria for rotator cuff lesions.

Given the evidence to date, it seems likely that the rotator cuff has some degree of reserve that affords functional use of the arm in cases of limited tendon deficiencies. Moreover, location rather than size of a tear may be more important in the development of symptoms; however, this issue requires elucidation with further study. Factors such as synovitis, subacromial bursitis, and intra-articular abnormalities may contribute to pain and dysfunction, but further basic science and clinical research can help define and isolate specific causes of pain in affected patients. Enhancement of our ability to identify individuals with cuff lesions prone to progression and dysfunction will afford the development of optimal treatment approaches individualized to a given patient's clinical findings.

ANIMAL MODELS OF THE ROTATOR CUFF

Animal models have been developed to test hypotheses related to the pathogenesis of rotator cuff disease. Such models are necessary to test hypotheses in a repeatable and controllable manor. Criteria for selecting an animal model of rotator cuff disease include animal size, anatomic relationship to humans, intrasynovial versus extrasynovial tendon location, intraarticular versus extraarticular tendon location, ease of tendon manipulation, ease of measurement of tissue properties, and animal availability and affordability. Anatomic similarity to the human shoulder includes evaluation of the shoulder musculature and associated bony anatomy (acromion, coracoid, clavicle, humerus), particularly the relationship between the supraspinatus tendon and the acromion or other structures immediately superior to the tendon, as well as articulations (glenohumeral, subacromial, acromioclavicular) and planes of motion.[226]

In designing any animal model, the clinical question being investigated will dictate the appropriateness of the selected model.[42] Therefore, while one animal may be ideal to study one aspect of rotator cuff disease, another species may be more appropriate for another area of rotator cuff pathology. As a result, multiple animal models of

the rotator cuff have arisen to assess different areas of rotator cuff pathology, such as intrinsic tendon degeneration, tendon injuries or tears, and subsequent tendon healing or surgical repair. These include rat, rabbit, avian, canine, goat, and sheep models (Table 1-2).

Soslowsky et al.[225,226] developed a rat model for studying multiple aspects of rotator cuff disease. The initial study evaluated 33 animal species as potential models. Gross anatomic dissections, radiographic assessments, and activity monitoring demonstrated that the rat was the only animal of the 33 with anatomic and functional relationships comparable to the human shoulder. In particular, the rat was the only animal with an acromion immediately positioned over the supraspinatus tendon, as in humans. The tendon passes repetitively under the acromion when the rat is ambulating, which may parallel repetitive over-

TABLE 1-2
COMPARISON OF COMMON ANIMAL MODELS FOR ROTATOR CUFF DISEASE

Species	Positives	Negatives
Rat (supraspinatus–infraspinatus tendon)	1. Acromial arch complex immediately positioned over supraspinatus tendon 2. Supraspinatus tendon passes repetitively under acromion during ambulation—may parallel repetitive overhead activities in humans 3. Controllable, reproducible alterations in tendon with potentially similar histologic, biologic, and biomechanical changes to human rotator cuff disease 4. Infraspinatus tendon long: covered entirely by acromion making ideal for use as impingement model	1. Quadruped: weight-bearing upper extremity 2. Acromial arch complex differences: acromion, AC ligament, clavicle, and coracoid in rat vs. acromion, CA ligament, and coracoid in humans 3. Small size: problem for experimental manipulations, such as creating tears or surgical repair, and applicability of techniques to humans 4. Supraspinatus tendon short: acromion covers muscle rather than tendon
Rabbit (supraspinatus tendon)	1. Supraspinatus tendon–bone insertion at the greater tuberosity identical to tendon–bone interface in humans: four zones of transition (tendon proper, nonmineralized fibrocartilage, mineralized fibrocartilage, and bone) 2. Delayed and immediate tendon repair models	1. Quadruped: weight-bearing upper extremity 2. Shoulder anatomy not as similar to human as rat model 3. Small size: problem for experimental manipulations, such as creating tears or surgical repair, and applicability of techniques to humans
Sheep (infraspinatus tendon)	1. Tendon dimensions, properties similar to the human supraspinatus tendon 2. Larger size allows evaluation of clinically applicable surgical techniques of rotator cuff repair 3. Delayed and immediate tendon repair models	1. Quadruped: weight-bearing upper extremity 2. Tendon heals spontaneously after detached: need to actively prevent healing to study effect of a delayed repair 3. Complex limb immobilization, partial weight-bearing protocol postinjury and repair to allow surgical repair healing
Canine/beagle (infraspinatus tendon)	1. Larger size allows evaluation of clinically applicable surgical techniques of rotator cuff repair	1. Quadruped: weight-bearing upper extremity 2. Shoulder anatomy not as similar to human as rat model
Chicken (supracoracoid tendon)	1. Non–weight-bearing, hanging shoulder joint as in human 2. Anatomic similarity: bursa-like structure superior to tendon, joint capsule inferiorly	1. Lack of an acromion, other structural differences: prevent creation of insertional tendon injury, tendon–to–bone repair 2. Small size: problem for experimental manipulations, such as creating tears or surgical repair, and applicability of techniques to humans

AC, acromioclavicular; CA, coracoacromial.

head activities in humans. While certain species of nonhuman primates also satisfied the selection criteria, they were eliminated because of questions of practicality of use due to expense and management issues.

This rat model was initially used to examine both intrinsic (degeneration) and extrinsic (compression) mechanisms of rotator cuff disease.[40] An intratendinous injection of bacterial collagenase was used to simulate intrinsic tendon degeneration, while an Achilles tendon allograft placed immediately underneath the acromion was used to simulate an extrinsic mechanism of subacromial impingement by decreasing the subacromial space. Both alterations resulted in controllable and reproducible injuries in the supraspinatus tendon, with a tissue response bearing significant similarity to a state of tendinosis seen in adult human tendon.[226] This rat model has subsequently been used to study numerous other areas of rotator cuff pathology, including the role of repetitive overuse activity of the supraspinatus tendon in tendinosis through the use of a rat treadmill running protocol,[40,193] the healing response of rotator cuff tears by creation of a defect or complete detachment of the supraspinatus tendon,[17,41,90,91,237] and the healing response of rotator cuff tears after surgical repair by complete detachment and repair of the supraspinatus tendon.[236,238] In addition to separately studying the intrinsic, extrinsic, and overuse injury models as factors in the development of rotator cuff tendinosis, the effect of a combination of these mechanisms in the development of rotator cuff tendinosis has been investigated.[227] These numerous studies demonstrate that controllable and reproducible alterations in the supraspinatus tendon can be made in this model, with potentially similar histologic, biologic, and biomechanical changes to human rotator cuff disease.

Despite its many benefits, the rat model does show some limitations. Rats have been noted to have high healing potential. One potential explanation is based on the size of the tissue. Considering that a rat supraspinatus tendon is only about 4 to 6 mm in width, and taking into account that cell size in rats and human are the same, rats may possess similar healing potential if the same actual size tear is made in a physiologically similar human (relative age). The rat is a quadruped, using its arms for ambulation and, thus, weight bearing. Human shoulders do bear significant loads, but it is unclear how similar the human situation is to the rat model. Anatomically, the rat acromial arch complex differs slightly from humans, consisting of the acromion, acromioclavicular ligament, clavicle, and coracoid, compared to the acromion, coracoacromial ligament, and coracoid in humans. Finally, the small size of the animal limits its use in certain experimental manipulations. For example, evaluating surgical techniques of human rotator cuff repair, such as tendon grasping or tendon-to-bone fixation, are best studied in animals with tendon size and dimensions more similar to the human rotator cuff.

Variations in the rat rotator cuff model have been attempted by others. Intrinsic tendon degeneration has also been modeled through the use of an injection of carrageenan, a polysaccharide.[240] Schneeberger et al.[215] created a rat model of subacromial impingement using the infraspinatus tendon. The authors felt that the supraspinatus tendon was not ideal to study subacromial impingement because the tendon is short in rats and the medial side of the acromion covers the supraspinatus muscle rather than the tendon. In contrast, the infraspinatus tendon is long in the rat and is found in close contact with the undersurface of the acromion across its entire tendon width. To create subacromial impingement, one or two bony plates, approximately 2 $\times$ 2 mm in size, were harvested from the scapular spine and placed on the undersurface of the acromion. Fixation was achieved with one absorbable suture stitch placed through two 0.4-mm drill holes made in the bony plate(s) and the acromion. One or two plates could be fixed to the acromion to vary the degree of impingement. This impingement model reproducibly led to bursal-sided infraspinatus tears of variable thickness, with no evidence of articular-sided or intratendinous tears noted. However, the bony plates displaced from the subacromial space in 31% of the rats.[215]

Several authors have investigated rabbit models of rotator cuff disease. Bjorkenheim et al.[23,24] examined healing properties of a supraspinatus defect in a rabbit model, including the use of intraarticular pressure readings in the glenohumeral joint to follow healing response. A triangular-shaped defect in the supraspinatus tendon was made near its insertion to the greater tuberosity, and subsequently it was shown that tissue resistance to hydrodynamic pressure in the glenohumeral joint was a reliable method of assessing the strength of the healing defect.[24] Uhthoff et al. also examined healing of a supraspinatus defect in an acute repair rabbit model.[247,248] In one study, the tendon was surgically exposed and transected near its insertion at the greater tuberosity. A bony trough at the greater tuberosity was immediately burred to expose cancellous bone, and the tendon edge was repaired back to the trough. Two weeks after repair, the cellularity of the cancellous bone underlying the bony trough and the thickness of the subacromial bursa were significantly increased, while the cellularity of the supraspinatus tendon stump was significantly decreased.[247] Changes in vascularity paralleled these changes in cellularity. The findings suggest that the underlying bone and subacromial bursa contribute to the early phases of tendon healing following surgical repair, while the tendon stump does not.

This rabbit supraspinatus detachment model has also been used to investigate healing response in nonrepaired tendon, as well as tendon undergoing delayed

repair.[44,150,246] In the delayed repair model, following supraspinatus detachment, the proximal tendon stump was wrapped in polyvinylidine fluoride membrane to prevent spontaneous reattachment. After 6 or 12 weeks, the tendon was reattached to the greater tuberosity. The supraspinatus tendon was successfully repaired using this model, but repair and healing did not reverse muscle and fatty changes that had developed while the tendon was detached. Reattachment of the supraspinatus tendon at 6 weeks did not reverse muscle atrophy or fat accumulation in the ensuing 6 weeks, but it did prevent an increase in fat accumulation when compared with later reattachment at 12 weeks.[150,246]

The rabbit model of rotator cuff disease has many of the same advantages and disadvantages of the rat model. The rabbit shoulder anatomy is less similar to human anatomy than the rat,[226] but the rabbit has been chosen by many investigators because the supraspinatus tendon–bone insertion at the greater tuberosity is identical to the tendon–bone interface in humans with four zones of transition: tendon proper, nonmineralized fibrocartilage, mineralized fibrocartilage, and bone.[247] Like the rat, the rabbit has four-legged ambulation and, thus, bears weight on its upper extremities. Although larger than the rat, the rabbit is still small in size and may also be less applicable for use in certain investigations.

Larger-size animals have also been used as rotator cuff models, particularly to evaluate surgical repair of rotator cuff tears, and include the sheep,[55,86–88] the goat,[230] and the beagle or other canines.[6,7,131] All of these large animal models have focused on the infraspinatus tendon, with the sheep most extensively studied. Without a suitable animal model for in vivo testing of rotator cuff repair techniques, Gerber et al. attempted to develop such a model in sheep.[86–88] An initial cadaveric study demonstrated that the sheep infraspinatus tendon has similar size, shape, and mechanical properties to the human supraspinatus tendon and is almost indistinguishable on histologic examination, making it an ideal animal model for evaluating surgical techniques of rotator cuff repair applicable to humans. Multiple suturing methods for tendon grasping and tendon-to-bone repair were tested with this sheep cadaveric model, and the modified Mason-Allen stitch was found to have the highest tensile strength and cause the least tendon strangulation.[87]

An attempt was made to repeat this in vitro study in an in vivo sheep model while also testing bone augmentation techniques at the repair site. To best simulate properties of long-standing rotator cuff tears, development of a chronic injury model with delayed tendon repair was attempted.[88] Infraspinatus tendon transection was made at the greater tuberosity insertion, with delayed repair at 4 to 6 weeks postinjury to replicate properties of chronic tears, including tendon atrophy, retraction, shortening, and greater tuberosity osteoporosis. Osteoporosis was created by decortication of the greater tuberosity, leaving exposed cancellous bone at the insertion site. Problems were encountered in the pilot studies of this model, however, and it was abandoned for an immediate-repair model. The difficulties with the model included complex limb immobilization and weight-bearing protocols postinjury and postrepair. The tendon repairs could not withstand full weight bearing on the extremity, and it became necessary to cement a rubber ball to the hoof of the operated limb to protect weight bearing and protect the surgical repair. These weight-bearing issues again highlight the problem of developing an animal model of rotator cuff disease in a quadruped. Most significantly, however, after 4 to 6 weeks of infraspinatus detachment, the tendon–bone junction was covered in such extensive scar that normal tendon and scar tissue were indistinguishable. As a result, repair sutures were frequently placed in peritendinous scar tissue rather than tendon, producing a high failure rate of tendon repair (12 of 15 animals). Even after abandoning this chronic model for an immediate-repair protocol, a reasonable success rate of surgical repair (8 of 10 sheep) was achieved only after tendon was repaired under no tension and weight bearing was protected postoperatively for 5 weeks.[88]

With recognition of the aggressive spontaneous healing response in the infraspinatus tendon following detachment, the delayed repair sheep model was reattempted using techniques to actively prevent healing of the tendon edge until the time of delayed repair. Gerber et al.[86] modified the surgical protocol by releasing the tendon at the time of injury with a greater tuberosity osteotomy, leaving a 2×1.5 cm fragment of greater tuberosity attached to the tendon edge to allow for better localization and fixation of the tendon at the time of delayed repair. In addition, the end of the infraspinatus tendon was covered in a 5 cm–long silicone rubber tube to prevent spontaneous healing prior to delayed repair. These modifications were successful, and delayed repairs were performed between 40 and 42 weeks posttendon release to mimic repair of a chronic rotator cuff tear. Indeed, at the time of repair, the detached tendon was found to have many properties that mimic a chronically torn rotator cuff, including significant retraction, muscle atrophy, and fatty infiltration.[86]

Coleman et al.[55] also developed a chronic rotator cuff repair model with the sheep infraspinatus tendon and utilized another technique to actively prevent healing of the tendon edge until the time of delayed repair. The infraspinatus tendon was detached from its insertion at the greater tuberosity and then repaired at different time points: immediate repair to simulate an acute tear model; 6-week delayed repair to simulate a chronic, repairable tear model; and 18-week delayed repair to simulate a chronic, irreparable tear model. Active healing or scarring prior to

surgical repair was prevented in the delayed repair groups by wrapping the tendon end in a Gore-Tex dura substitute, shown to inhibit scar formation.[15] Protected weight bearing for 5 weeks postoperatively was also utilized in this model by affixing a rubber ball to the hoof of the surgically repaired extremity. This protocol resulted in successful surgical repair of tendon back to bone in both delayed repair groups, with the detached tendon edge easily identifiable at the time of repair. The 18-week irreparable tear group showed twice as much tendon retraction as the 6-week repairable tear group at the time of repair and required a polylactic acid patch to span the tendon–bone gap and allow tendon reattachment. In addition, the earlier repair of tendon (6 weeks vs. 18 weeks) was found to result in more rapid recovery of infraspinatus muscle function and tendon elasticity.[55]

Unlike the other quadruped animal models, Kobayashi et al.[133] attempted to develop a rotator cuff model in a species with a non–weight-bearing, hanging shoulder joint as in the human, and chose the avian shoulder. Despite the lack of an acromion, the avian supracoracoid tendon has considerable anatomic similarity to the human rotator cuff tendon, including a bursa-like structure superior to it and the joint capsule inferiorly. An acute injury and repair model was examined, with a long transverse full-thickness laceration, approximately 60% of tendon width, created in the chicken supracoracoid tendon. This injury was made proximal to the insertion site, unlike in other models due to anatomic differences, and then immediately repaired with a single simple stitch. Analysis with histology and in situ hybridization found that peritendon cells of the bursal side of the tendon played a significant role in the repair process, which then progressed to the articular side of the tendon. Several limitations in this model relate to the differences in avian and human shoulder anatomy. In addition to the lack of an acromion, other structural differences prevented evaluation of an insertional injury and tendon-to-bone repair process.

Canine models, such as the beagle, have also been used to study the role of synthetic materials, such as a polylactic acid patch, in irreparable rotator cuff tears. As with the sheep, the beagle rotator cuff model utilizes the infraspinatus tendon. A polytetrafluoroethylene (PTFE) graft was tested in an acute injury and repair model by first detaching the infraspinatus and then removing 1 cm of tendon at the insertion edge to create an irreparable defect.[131] The injuries were immediately repaired, with the PTFE felt graft sutured to the tendon edge and to a bone trough created in the greater tuberosity to span the irreparable defect and bring tendon back to bone. All reconstructed infraspinatus tendons healed. A poly-L-lactic acid (PLLA) felt graft was also tested with this same model. All but two of the tendons healed back to bone using this graft. PTFE and/or PLLA grafts may become clinically useful bioabsorbable materials for rotator cuff reconstruction.

A well-chosen animal model and well-designed experimental protocol can be powerful tools with which to test hypotheses related to pathogenesis or potential mechanisms of rotator cuff disease. Essential to this statement is appropriately matching the animal model to the condition being studied. In general, larger animals, such as sheep, are necessary to closely model and evaluate surgical techniques used clinically. Smaller animals are more practical in terms of expense, care, and handling, but their size can make tissue access and analysis more difficult at times. With research focusing more and more on tissue engineering techniques and gene therapy as novel approaches to the treatment of musculoskeletal diseases such as rotator cuff pathology, animal models will be essential for rigorously testing hypotheses related to such therapies.

ROTATOR CUFF TISSUE ENGINEERING

Basic Principles of Tissue Engineering Design

The goals of tissue engineering are to increase the healing response of the body to insults to regenerate tissues efficiently and to produce new tissues that are comparable to healthy tissue in strength and function. In the case of tendon, this goal translates to a restoration of load-bearing function with a decreased risk of recurrent failure. The rotator cuff is a musculotendonous complex that is likely to benefit from the development of tissue engineering models for tendon repair.[251]

To maximize success of a tissue engineering approach for the treatment of rotator cuff disorders, there are three key steps to accomplish. Step 1 is the identification of the targets and intended goals. To accomplish step 1, there needs to be an understanding of events involved with the rotator cuff disease process, with a focus on the identification of therapeutic targets. This includes defining the temporal, spatial, cellular, and molecular events involved with cuff degeneration of repair as well as the origin of the responding cells. Step 2 is defining biomechanical properties and testing protocols. This will allow for the ability to quantitatively evaluate the properties of the engineered tissue. This assessment demands established standards of successful biomechanical restoration.[101,103,104,119] Step 3 is choosing the engineering strategy best suited to achieve desired effect.[37,80] Engineered tissue can be created by altering the responding cells, augmenting the healing signals, blocking the inhibitor pathways, or creating de novo tissue in a bioreactor, while functional tissue engineering uses stem cells placed on a scaffold that can be manipulated with signaling molecules in the correct biomechanical environment to form a tendon. Choosing the strategy best suited for your intended goal is paramount for a successful program.[251]

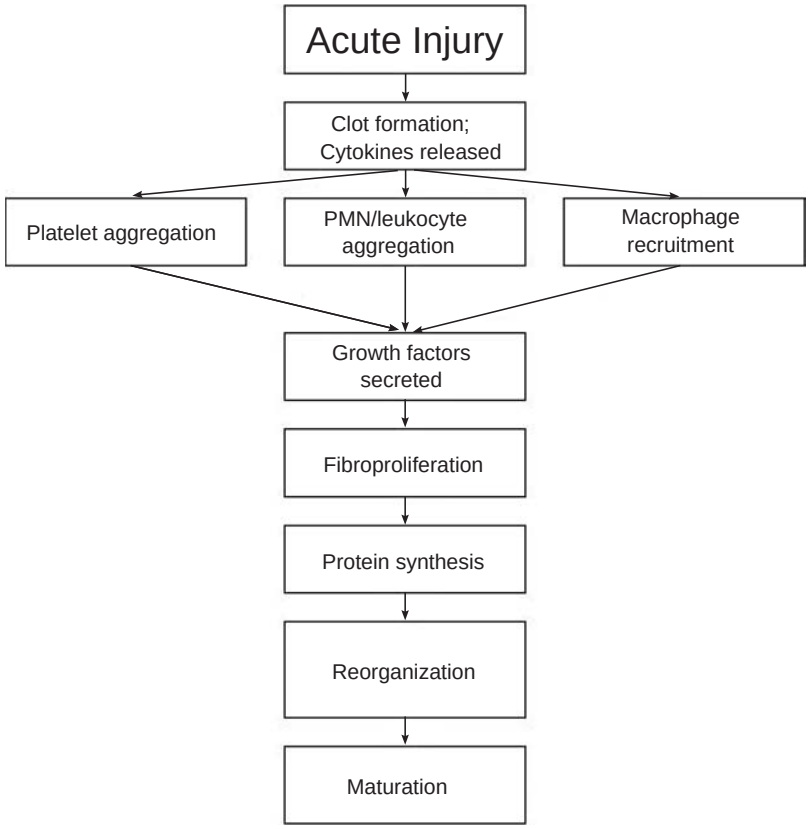

Figure 1-23 Sequence of biologic and mechanical events in the healing process in tendon following acute injury. PMN, polymorphonuclear. (From Van Kleunen JP, Soslowsky LJ, Glaser DL. Tissue engineering of rotator cuff tendons. In: Wnet G, Bowlin G, eds. Encyclopedia of Biomaterials and Biomedical Engineering, 2004:1622–1628, with permission.)

Step 1: Cellular and Molecular Events Involved in Cuff Degeneration and Repair

Pathologic responses to a loaded environment may be a result of lower metabolic activity in tendon associated with an inability to react to changes in load. An increase in the proportion of collagen III, increased fibril diameter, reduced collagen turnover, increased mature crosslinking, decreased proteoglycan and water content, and reduced cellularity have been found to occur with aging. These changes produce a less compliant tissue that is more susceptible to injury and inadequately prepared to heal efficiently following pathologic events. All of these changes can be addressed with tissue engineering.[14,105,204]

Injury may be precipitated by direct events or may be purely a result of chronic degeneration. Initially following insult, the tissue surrounding the tendonous injury undergoes a hemorrhagic response that triggers the inflammatory pathway. Secretion of growth factors, including platelet-derived growth factor (PDGF), transforming growth factor-α and -β (TGF-α, TGF-β), basic fibroblast growth factor (bFGF), and epidermal growth factor (EGF), accompanies this response to direct the attraction and proliferation of fibroblasts and to stimulate collagen and protein synthesis. Fibroblasts play the central role in healing and remodeling tendon at the site of injury. This process takes considerable time, and even at maturation, the biomechanical properties of healed tissue are inferior to those of uninjured tendon due to a complicated system including a proportional decrease in the amount of collagen I and an increase in the amount of collagen III (Fig. 1-23). Again, these cytokines can be targets for tissue engineering.[260]

Specific targeting of the cellular pathways involved in tendon healing has been impossible because the identity of the stem cell population that contributes cells to the healing tissue remains unknown. Both extrinsic and intrinsic sources of cells have been described and no certain answer has been elucidated.[82,199] Glaser et al. recently investigated the heritage of cells involved in the healing tendon. The objective of this study was to examine the specific ancestry of cells that participate in tendon healing including myoblasts, activated satellite cells, differentiated skeletal muscle cells, vascular and nonvascular smooth muscle cells, pericytes, endothelial cells of developing and mature blood vessels, and bone marrow–derived cells. This study suggested that smooth muscle cells from mature or immature vessels or from pericytes are a major source of responding cells in the fibroproliferative stage of tendon healing and that cells of endothelial origin respond to a tendon injury by participating in neovascularization, but do not contribute to fibroproliferation. Despite the

hypothesized role satellite cells play in healing, activated satellite cells do not have a major role in healing tendon injuries. Cells of bone marrow origin contributed to the inflammatory process. However, bone marrow–derived cells did not contribute to the fibroproliferative response or develop into tendon cells. This study helped to elucidate the origins of cells involved in the various steps of the healing process.[92,140]

Step 2: Defining the Standards of a Rotator Cuff Tendon

Before a tissue engineering model can be created, defining standards for the engineered tissue is an initial step and guides the remaining developmental process. The ability of an engineered tissue to withstand forces approximating those experienced during a healthy state helps to define this goal. Biomechanical properties for a given tissue must be understood and prioritized. Stress and strain parameters should be measured in both normal and repaired tissue. Biomechanical analyses have identified principle tensile units in each component of the rotator cuff that define the mechanical parameters that result in failure. The anterior third of the supraspinatus, midsuperior and inferior portions of the infraspinatus, and superior and midsuperior portions of the subscapularis tendons have been identified as these primary units (Table 1-3).[103,104,119]

Step 3: Common Tissue Engineering Strategies

To achieve these goals, attempts at tissue engineering in rotator cuff tendons have taken several directions. Several of these studies have demonstrated promise as potential means of augmenting the healing response in damaged tendon. Unfortunately, no studies have combined all of these techniques in one system. In this section, we will describe several models of tissue engineering that have demonstrated improvements in tendon healing. Rehabilitation is a mechanical therapy that aims to improve tendon healing through the application of regulated mechanical stresses on the healing tendon. Cell therapy focuses upon the implantation of stem cells, or other pluripotent cells, into a site of tendon healing. These cells may serve a role in regenerating tissue and producing growth factors important in the healing process. Small intestine submucosa is an acellular biologic scaffold that has come under recent examination as an organized matrix for the direction of connective tissue healing. Gene therapy is a broad field of study that is based on the incorporation of genetic material into host tissue to augment the healing process from a

TABLE 1-3
TENSILE PROPERTIES MEASURED IN HUMAN ROTATOR CUFF TENDONS

Tendon	Cross-Sectional Area (mm^2)	Ultimate Load (N)	Stiffness (N/m)	Ultimate Stress (MPa)	Elastic Modulus (MPa)
SS Anterior Third	25.5	411	N.R.	17	N.R.
SS Middle Third	24.7	153	N.R.	6	N.R.
SS Posterior Third	21.9	88	N.R.	4	N.R.
IS Superior Portion	29.0	463	134	16	120
IS Midsuperior Portion	26.3	677	171	27	156
IS Midinferior Portion	20.8	315	97	16	111
IS Inferior Portion	26.2	550	149	22	140
TM	49.0	67	23	1	14
Sub. Superior Portion	40.3	623 (0°) 478 (60°)	147 (0°) 209 (60°)	N.R.	N.R.
Sub. Midsuperior Portion	N.R.	706 (0°) 598 (60°)	175 (0°) 182 (60°)	N.R.	N.R.
Sub. Midinferior Portion	N.R.	454 (0°) 400 (60°)	128 (0°) 130 (60°)	N.R.	N.R.
Sub. Inferior Portion	27.3	75 (0°) 30 (60°)	27 (0°) 10 (60°)	N.R.	N.R.

Note: Values for the infraspinatus and teres minor tendons are combined measurements for both 0 degrees and 60 degrees of abduction. Values are listed for the subscapularis tendon in both 0 degrees and 60 degrees of abduction.
IS, infraspinatus; N.R., not recorded; SS, supraspinatus; Sub., subscapularis; TM, teres minor.
From Van Kleunen JP, Soslowsky LJ, Glaser DL. Tissue engineering of rotator cuff tendons. In: Wnet G, Bowlin G, eds. Encyclopedia of Biomaterials and Biomedical Engineering, 2004:1622–1628.

genetic biochemical level. The proposed mechanisms by which each of these models serves to augment tendon healing will be discussed, in addition to their appropriate roles in the repair of the rotator cuff.

Cell Therapy

Mesenchymal stem cells (MSCs) and other stem cells are pluripotent cells isolated from various sources that have not yet differentiated into a specific cell line. These cells have the potential to develop into a wide variety of tissues including bone, cartilage, tendon, fat, marrow stromal cells, and muscle. Studies examining the transplantation of allogeneic and xenogeneic stem cells into human tissue both in vitro and in vivo have shown that these cells maintain their pluripotency following transplantation and undergo site-specific differentiation.[139,200] In this manner stem cells transplanted into a region of damaged tendon may differentiate into endothelial cells, osteoblasts, and fibroblasts to act as a biologic patch and to further augment the healing process through the increased production of collagen and proteoglycan.

While the potential for improved healing is substantial in this model of tissue engineering, many challenges exist that must be met to develop a functional application of cell therapy to tendon repair. An adequate source of stem cells must exist for a substantial transplantation of stem cells to be a reality. Autogenous donation is ideal in terms of delivering an immunologically low-risk population of cells, but the collection process may incur other morbidities. Allogeneic and xenogeneic sources may be easier to develop but carry the risk of potential rejection or disease transmission.[38] Once a reliable population of stem cells is isolated, a delivery vehicle must be selected that will protect the cells but will also allow them to function normally following transplantation. Once both of these needs are met, a technique must be developed to combine the stem cells with the carrier in vitro and deliver them in vivo. This composite must be provided in such a way that makes its use feasible, safe, and efficacious.[38]

Early studies in animal models and in humans have demonstrated successful implementation, survival, and differentiation of MSCs in tendon, but more work is required to fully elucidate the recipient response to donor cells and to determine optimal conditions for successful implementation of the technique.[12,111,191,266] Standardization of a safe, effective delivery system will facilitate the potential use of cell therapy as an adjunct to tendon repair.

Gene Transfer

Gene transfer therapy is a model that seeks to directly induce greater healing ability in the cells already in existence around a site of injury. Several growth factors have been implicated in the healing response of tendon, including TGF-β, TGF-α, bFGF, PDGF, insulin-like growth factor (IGF)-I, and EGF.[2,45,65,105,231] The goals of gene transfer, with regard to tendon healing, are to up-regulate the synthesis of these growth factors and to suppress production of endogenous proteins that may inhibit efficient, organized remodeling of damaged tendon.

For transfer to occur to host cells, an appropriate vector must be developed that will successfully deliver genetic material to the desired target and allow implementation into the host's genome. Viruses, liposomes, and gene guns are all methods of delivery that have demonstrated potential use for this purpose.[37,58,244,260] Successful delivery and implementation of genetic material into the host's genome will lead to synthesis of the proteins encoded by the delivered gene or to down-regulation in the production of an unwanted protein from a separate path. The advantages of this model of tissue engineering are that it induces a significant, local production of a substance that augments healing and that it is not complicated by morbidities associated with insertion of donor tissue. The disadvantages are those most commonly associated with the vectors used for gene delivery. The production of certain viral vectors is a difficult process with a frequently low yield. Several viral strains carry a risk of mutagenesis that may interfere with the intended genetic result. Some strains of virus, adenovirus the most notable, are capable of stimulating an inflammatory response and secondarily immunogenic rejection in the host. Gene guns, which involve particle bombardment of genetic material into host tissue, require specialized equipment that significantly increases the costs of production and utilization. Liposomes and naked DNA, while less immunogenic than other vectors, are not as successful as other methods in transfecting host cells. Because of these limitations, vector development remains an extremely active component of gene transfer research.[71]

While the use of gene transfer is in its early stages, some studies have demonstrated successful incorporation of marker genes into tendon and ligament.[89,146,224] Promising studies have explored the transfer of antagonists to FAK, a protein kinase linked to adhesion formation, and of genes encoding PDGF.[71,260] Future studies will continue to examine the insertion of genes to augment the production of growth factors involved in the healing processes of tendon or of antagonists to inhibit the production of proteins found to interfere with organized remodeling.

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2 Diagnosis, Patient Selection, and Clinical Decision Making

Allen Deutsch *Matthew L. Ramsey* *Joseph P. Iannotti*

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INTRODUCTION

It is crucial to properly diagnose the extent of the rotator cuff pathology to direct clinical decision making. Historical factors, including the mechanism of injury and the presence or absence of a traumatic cause, can help determine the severity of the injury and whether the injury is acute or degenerative in nature. Patient age and activity level can guide the discussion of surgical goals and patient expectations. Diagnostic imaging aids in determining tear characteristics, such as tendon involvement, tear size, tear retraction, and muscle atrophy. These factors help to formulate and implement a treatment algorithm (Table 2-1).

EVALUATION

Clinical History

Patients with rotator cuff problems will typically complain of pain or weakness or both. Many patients cannot recall an injury, and in others, symptoms may have begun after a trivial trauma (e.g., catching their balance by holding onto a railing). However, more-severe injuries may play a role, especially when a cuff tear occurs in association with a dislocation in an older patient. The pain is usually described as anterior, down the anterior humeral region, or lateral, down to the deltoid tuberosity. Pain in the back of the shoulder or trapezius or that which radiates down past the elbow into the hands is more consistent with a cervical radiculopathy, although there are exceptions. Pain from rotator cuff pathology is usually increased with use of the arm, particularly for overhead activities, and is often most severe at night, frequently interrupting sleep.

Because splinting from pain may simulate weakness, lack of strength does not always indicate a large tear unless a subacromial anesthetic injection has been given first. Furthermore, patients with massive tears may still

TABLE 2-1
ALGORITHM FOR SURGICAL DECISION MAKING

Pathology		Treatment
Subacromial bursitis/ tendonitis	⇒	Arthroscopic subacromial bursectomy
If prominent/worn coracoacromial ligament	⇒	Arthroscopic coracoacromial ligament excision
If prominent acromion, bone spur, <50% thickness partial rotator cuff tear	⇒	Arthroscopic acromioplasty, cuff débridement
If >50% thickness partial rotator cuff tear, small or medium full-thickness rotator cuff tear	⇒	All-arthroscopic or arthroscopically assisted ("mini-open") rotator cuff repair
Large or massive rotator cuff tear	⇒	All-arthroscopic or open rotator cuff repair, coracoacromial ligament preservation Possible muscle transfer in selected patients

have remarkably good motion and function. However, it is more common for patients with large or massive tears to report weakness and fatigue with overhead use and difficulty in raising their arm. If the onset of weakness is sudden after an injury, especially one causing a glenohumeral dislocation, then not only a rotator cuff injury needs to be suspected, but also a neurologic injury (most commonly suprascapular or axillary nerve) should be ruled out.

Physical Examination

Inspection and Palpation

In the presence of a rotator cuff tear, crepitus on passive motion can be palpated by placing a hand over the superior aspect of the shoulder. Visual inspection for shoulder symmetry will not reveal variations between shoulders in the presence of acute rotator cuff tears. However, in the presence of long-standing cuff tears, atrophy of the supraspinatus or infraspinatus will often be present. Asymmetry of the biceps muscle belly may be a clue to a partial recession or dislocation of the tendon of the long head of the biceps.[37] A complete rupture, not infrequently, is seen with large cuff tears and can be identified by deformity of the long head of the biceps.

Range of Motion

Range of motion of the shoulder should be performed actively and passively and compared with the asymptomatic side.[1] Patients with less active than passive range of motion may indicate the presence of a rotator cuff tear. This limitation of active range of motion may be caused either by weakness or pain (Fig. 2-1). Subacromial injections with 1% lidocaine (Xylocaine) can be used to help distinguish between the two. If there is limited range of motion, but no difference between active and passive, then adhesive capsulitis or arthritis should be suspected. It is surprisingly infrequent for patients with rotator cuff tears to have decreased passive range of motion, although this can occur. Patients with rotator cuff tears most commonly exhibit posterior capsular stiffness with a loss of the ability to reach up the back or adduct across the body. Loss of

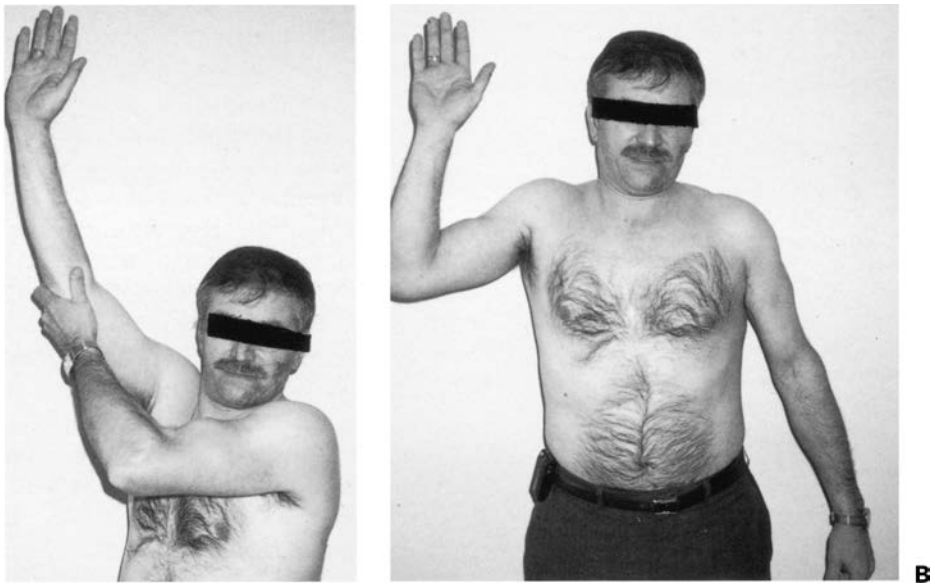


Figure 2-1 **(A)** This patient with a massive cuff tear has full passive elevation when assisted with the other arm. **(B)** Active elevation, however, is severely impaired.

internal rotation can be measured with the arm in abduction and compared to the opposite asymptomatic shoulder. This method of measurement is very accurate particularly when the examiner holds the top of the shoulder to detect and restrict scapula rotation. Excessive passive external rotation in the injured shoulder compared to the opposite side is a good indicator of a full-thickness complete or near complete tear of the subscapularis. These signs should be systematically evaluated in all patients suspected of having a rotator cuff tear.

Strength

Strength of the shoulder should be examined in elevation, abduction, external rotation, and internal rotation. Patients with rotator cuff tears may exhibit weakness or pain on resistance to muscle testing. Isometric strength testing can be used to attempt to isolate specific cuff muscles. The supraspinatus can be tested with the arm internally rotated and elevated 90 degrees in the plane of the scapula, the infraspinatus with the arm at the side in neutral rotation and varying degrees of external rotation with the elbow flexed 90 degrees, and the subscapularis with the elbow flexed 90 degrees and the hand behind the waist or against the abdomen. A handheld or wall-mounted dynamometer has been demonstrated to be a reliable and discriminatory means for assessing strength of the rotator cuff in symptomatic individuals.[20] Patients with partial-thickness tears usually demonstrate more pain on muscle testing than individuals with full-thickness lesions. For large or massive tears, strength may be reduced when compared with the contralateral side. Weakness of external rotation is a common finding, particularly when the infraspinatus is involved in the tear. Patients with large or massive tears involving the infraspinatus will often be unable to maintain their arm in external rotation.

Hertel and coworkers[21] have quantified these findings by describing external rotation and internal rotation "lag signs," which measure the difference between passive and active terminal rotation. If the patient sits with the arm at the side and the elbow bent 90 degrees, the arm may be fully externally rotated passively, but once the arm is let go it will "fall off" into internal rotation[4] (Fig. 2-2A–C). The similar inability to actively externally rotate the abducted arm has been termed the "signe de clairon," since it puts the arm in the position used to blow a bugle[13] (Fig. 2-2D). This pathologic sign usually indicates a massive tear with involvement of the lower head of the infraspinatus and teres minor tendons. The degree of apparent weakness of external rotation in the abduction position may be measured differently if the arm is placed in the plane of the scapula versus the coronal plane. When the arm is in the coronal plane the posterior deltoid can act as a weak external rotator, giving the impression of greater external rotation strength of the rotator cuff. As the arm is brought into the plane of the scapula or sagittal plane, the external rotation effect of the posterior deltoid is diminished.

Weakness of terminal internal rotation indicates involvement of the subscapularis. Internal rotation strength can be evaluated with the "lift-off" test[14] (Fig. 2-3A–C). Patients are asked to reach behind their back and place their hand on the trunk. The test is considered positive if the patients are unable to lift and maintain their hand away from their trunk. The position of the arm for the "lift-off" test in maximum internal rotation may be difficult to accomplish for a patient with a large rotator cuff tear.[12] Another method to test the strength of the subscapularis is to have the patients place their hands on their abdomen, internally rotate their shoulders, bringing their elbows in front of their torso, and then push their hands into their abdomen (Fig. 2-3D). When the subscapularis is deficient the patient's elbow will fall back behind the coronal plane of the body or the patient will not be able to initiate active internal rotation with the palm flat on the abdomen. With mild weakness of the subscapularis the patient will not be able to compress the abdomen with terminal internal rotation and will attempt to use the posterior deltoid to achieve abdominal compression, resulting in pull-back of the arm. When the shoulder is weak in internal rotation, the patient will also compensate for his or her inability to bring the elbow forward by bringing the palm of the hand off the abdomen. In all cases a true positive test must allow for passive internal rotation to achieve these maneuvers. When there is a tight posterior capsule preventing passive internal rotation to the coronal plane, then these tests for subscapularis function are not reliable.

Provocative Testing

Provocative tests are used to elicit symptoms of impingement by maneuvering the biceps and rotator cuff under the coracoacromial arch. The Neer impingement test entails elevation with the arm internally rotated,[34] whereas Hawkins' test for impingement is elevation to 90 degrees, adduction across the chest, and internal rotation[18] (Fig. 2-4). Both of these tests bring the biceps, rotator cuff, and greater tuberosity directly under the coracoacromial arch. However, these maneuvers may also cause pain in other shoulder conditions such as stiffness, calcium deposits, and arthritis. In patients with classic impingement syndrome, pain will not only be produced by these impingement maneuvers, but should be nearly completely eliminated following a subacromial injection of 10 mL of 1% lidocaine. A subacromial lidocaine injection has been found to be helpful in differentiating weakness secondary to a rotator cuff tear from weakness that is due to pain inhibition.[3]

Posterior internal glenoid impingement can result in partial tears of the supraspinatus and anterior half of the infraspinatus when abnormal contact exists between these

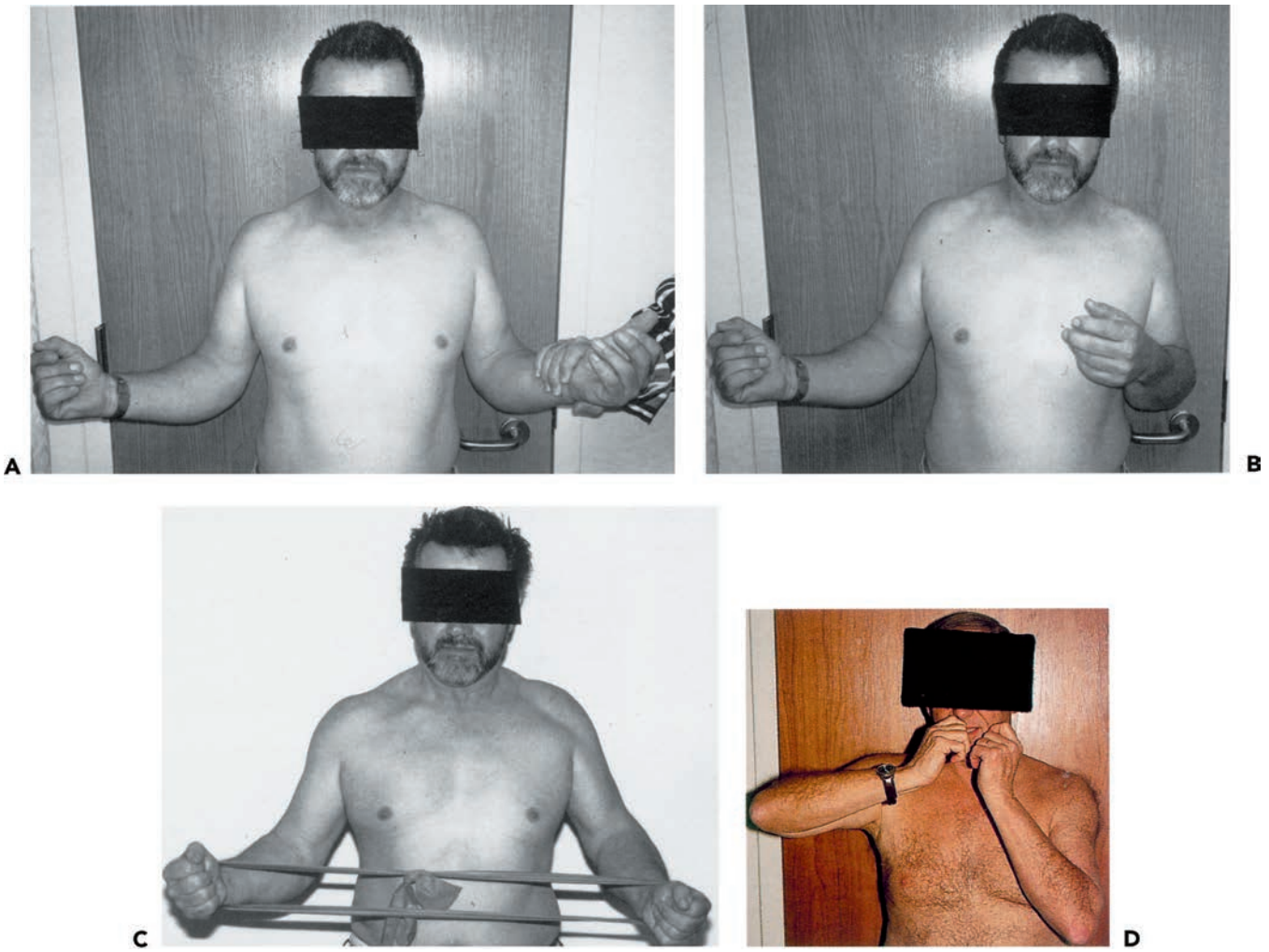


Figure 2-2 **(A)** This patient with a massive tear involving the posterior cuff can be passively externally rotated. **(B)** When the arm is let go it "falls off" into internal rotation, demonstrating severe weakness. **(C)** One year after rotator cuff repair he has regained active external rotation, although his strength is not full. **(D)** "Signe de clarion": Right arm must be elevated higher than left to reach mouth since active external rotation is impaired.

tissues and the posterior superior glenoid. This type of impingement occurs when the arm is brought into the extremes of external rotation and abduction (cocking arm position of throwing or anterior apprehension position). In these cases placing the arm in this position will cause pain in the posterior glenohumeral joint line. With a slight change in arm position, either bringing the arm in less external rotation or less coronal plane extension or placing a posterior directed force along the anterior aspect of the shoulder (relocation test), the pain is substantially diminished. These maneuvers are helpful in the diagnosis of posterior glenoid impingement. Anterior internal glenoid impingement occurs when the undersurface of the subscapularis, the medial biceps pulley complex, and the biceps tendon contact the anterior superior aspects of the glenoid labrum and rim. In these cases cross-body adduction and internal rotation and resisted forward flexion with the arm in this position will differentially illicit a greater amount of pain compared to the same cross-body adduction with external rotation (O'Brien's sign). With both posterior and anterior glenoid impingement, superior labral tears (SLAP lesions) can occur and are part of this pathology and symptoms complex. Subcoracoid impingement may also occur with cross-body adduction and internal rotation.

The acromioclavicular (AC) joint is often a source of pain; it should be inspected for prominence and palpated for tenderness. Maneuvers to elicit signs of impingement can also exacerbate pain from the AC joint. AC joint pain is often increased with internal rotation and cross-body adduction. Pain with these maneuvers should be at the top of the shoulder and should not be confused with posterior or lateral arm pain, which is more commonly associated with rotator cuff disease or posterior capsular tightness.

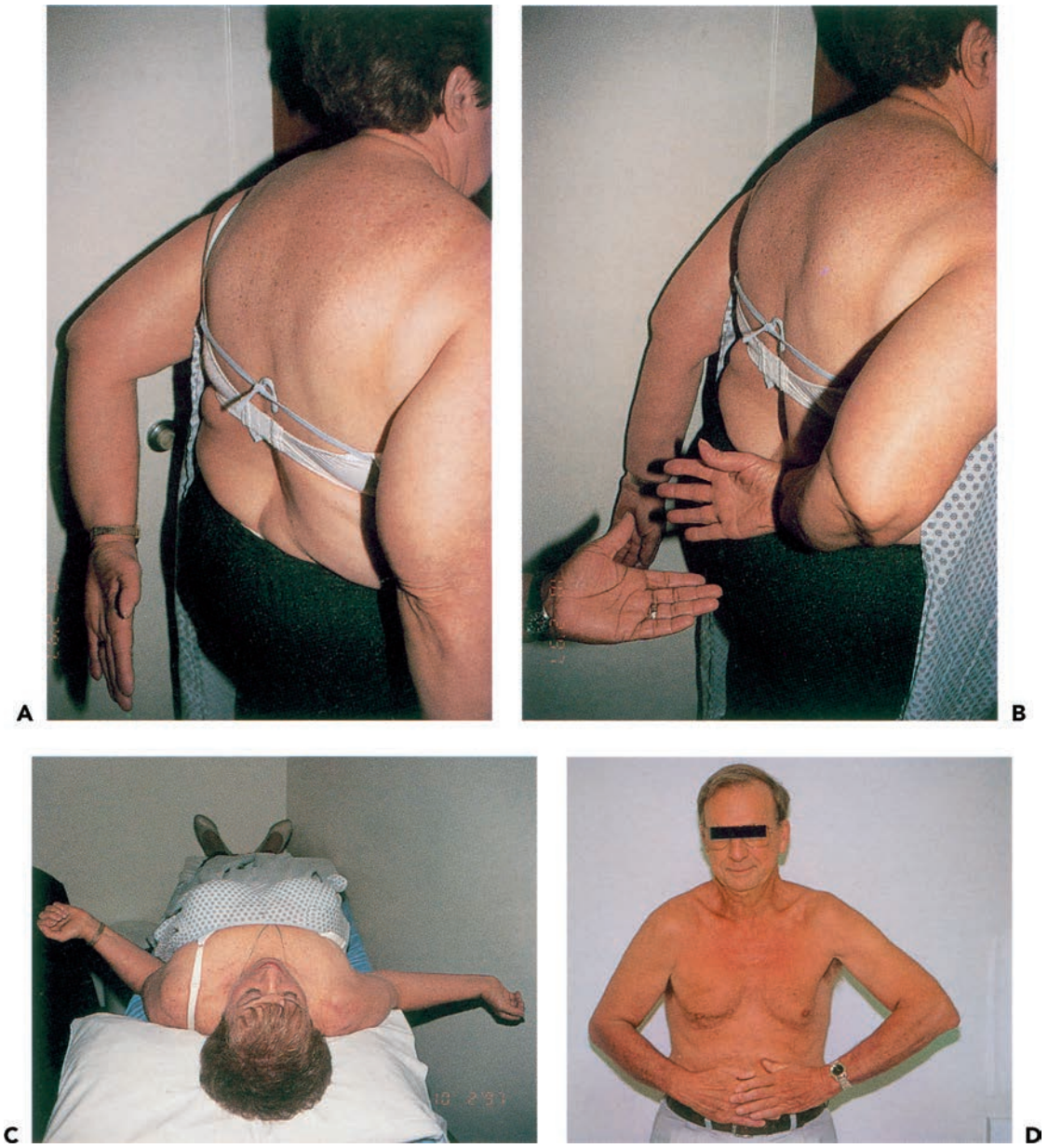


Figure 2-3 **(A)** The lift-off test is used to evaluate the integrity of the subscapularis tendon.[16] Although originally it was described as asking the patient to lift her hand off the small of her back, it can be more sensitive if the examiner holds the hand to maximally internal rotate the arm and then lets go. This patient is able to maintain maximal internal rotation with her left shoulder. **(B)** Her right shoulder, which has an isolated subscapularis tear, has an internal rotation lag because she cannot maintain maximal internal rotation. **(C)** Her right shoulder has increased external rotation, another finding in subscapularis ruptures. **(D)** Abdominal compression test: The right elbow falls back when the patient tries to compress his abdomen.

When there is uncertainty about the source of shoulder pain, serial injections, first into the subacromial space followed by an injection into the AC joint, can be performed and patient responses recorded. It is not uncommon for patients to have prominent, arthritic AC joints that are asymptomatic. Resection of the distal clavicle should be performed when the AC joint itself is tender and painful. Finally, instability testing should be performed in those patients, especially throwing athletes, in whom the relative contributions of instability and rotator cuff pathology are uncertain. Instability and partial cuff tears, particularly those associated with posterior internal glenoid impingement, are not an uncommon combination of overlapping pathologies in the younger athletic patient population.

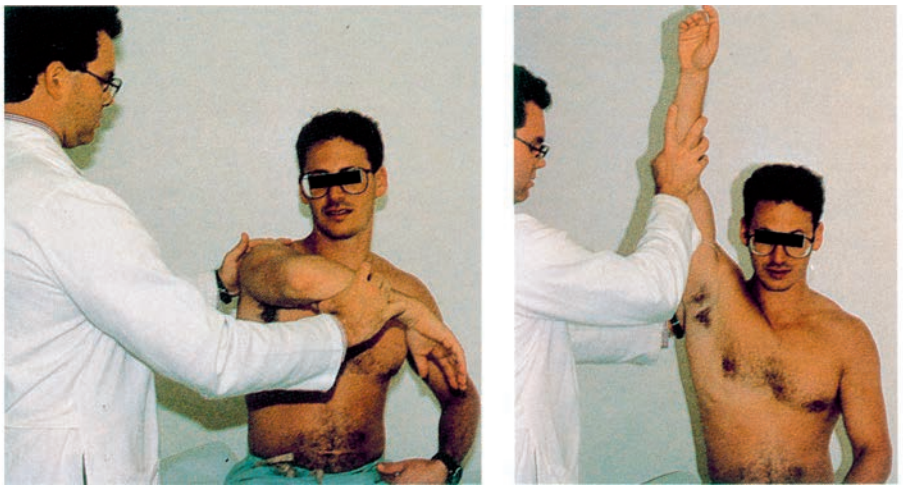


Figure 2-4 **(A)** Hawkins impingement sign. **(B)** Neer impingement sign.

IMAGING

Radiographs

Plain radiographs are used to determine the bony morphology of the acromion and to evaluate the position of the humeral head relative to the glenoid fossa and acromion. Plain radiographs are also important to rule out other sources of shoulder pain such as calcific tendinitis, glenohumeral osteoarthritis, or destructive bone lesions. Patients with rotator cuff pathology are evaluated with five views of the shoulder. Anteroposterior (AP) views in the plane of the scapula in neutral, internal, and external rotation are obtained to visualize the glenohumeral joint and greater and lesser tuberosities, and to bring small calcium deposits into relief. The AP view may also reveal an excrescence or cysts on the greater tuberosity, suggestive of rotator cuff disease. A double-density sign within the acromion on a standard anteroposterior radiograph of the shoulder has been described as highly suggestive of an os acromiale.[27] The os acromiale is best seen on a well-performed axillary view (Fig. 2-5), but can be also seen on magnetic resonance imaging (MRI) (Fig. 2-6). The acromiohumeral interval can also be assessed on the AP views and, if less than 7 mm, may be an indication of a torn or nonfunctioning rotator cuff.[47]

On the axillary view, the glenohumeral joint and tuberosities can be inspected. The acromion is usually well visualized on the axillary view and can be evaluated for the presence of an os acromiale, or unfused anterior acromial epiphysis. The most common type of os acromiale is a meso type and is seen at the level of the posterior margin of the clavicle (see Fig. 2-5). The axillary view is also useful to look for subtle joint space narrowing seen in early arthritis.

Finally, a supraspinatus outlet view will show the subacromial space and the coracoacromial arch. To obtain this view, a lateral x-ray film of the scapula is obtained while the x-ray beam is angled downward 10 degrees.[6,36] The supraspinatus outlet view will reveal any spurs encroaching on the subacromial space from the AC joint or anteroinferior acromion (Fig. 2-7). It is also the view used to determine the acromial morphology (Fig. 2-8).

Tendon Imaging

Arthrography is an extremely accurate method for the detection of full-thickness rotator cuff tears (Fig. 2-9). However, it is an invasive procedure that does not give accurate information on tear size or the condition of the rotator cuff muscles. Also, partial-thickness tears are less reliably assessed.

In recent years, high-resolution dynamic shoulder ultrasound has gained popularity as a noninvasive, accurate method to evaluate the rotator cuff (Fig. 2-10).[10,22,23,29,31,32,39,42,45,50]

The advantages of this technique are that it is noninvasive, is less expensive than MRI, uses no radiation, can be

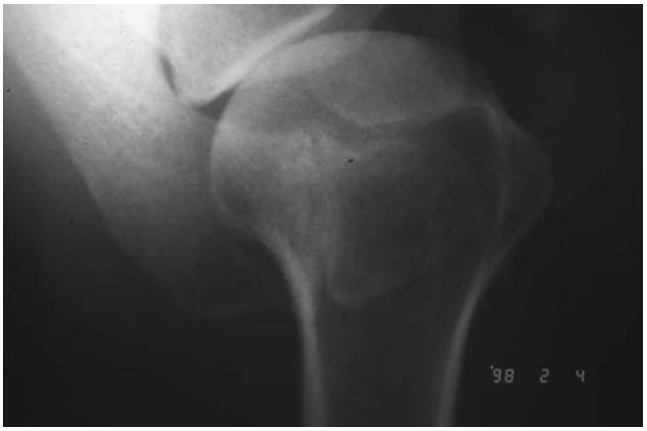
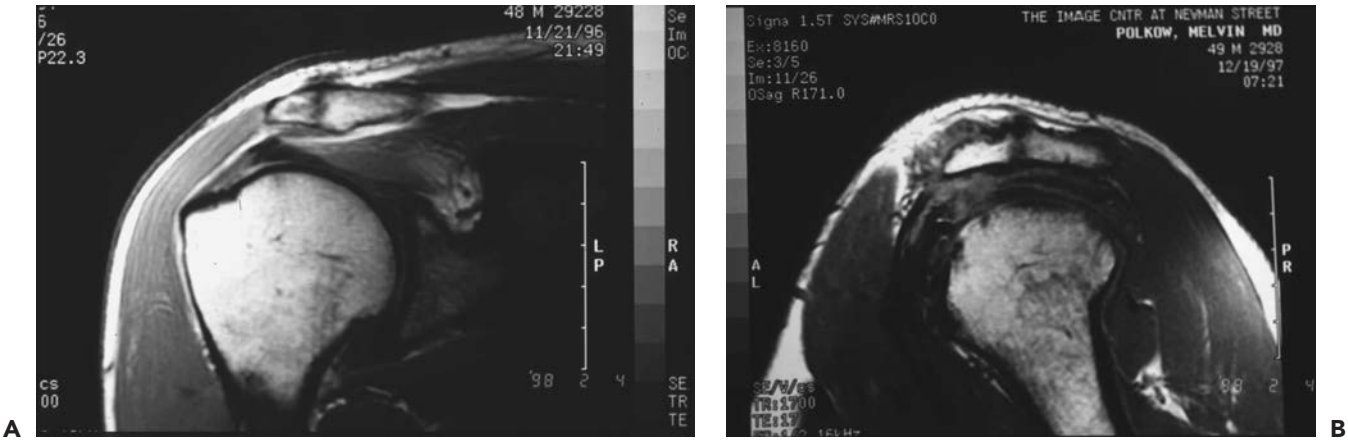


Figure 2-5 Axillary view showing a meso os acromiale.



A B

Figure 2-6 **(A)** Coronal anteroposterior view showing a meso os acromiale. **(B)** Sagittal oblique magnetic resonance imaging view showing a meso os acromiale.

performed in the clinic setting, allows for dynamic examination, and can be performed on both shoulders without undue time or cost. Ultrasonography and MRI have been demonstrated to have comparable accuracy for identifying full-thickness and partial-thickness rotator cuff tears.[29,39,42,45]

Improvements in the quality, portability, and cost have led orthopaedic surgeons to incorporate dynamic ultrasound in the clinic setting.[23,50] This approach may allow for a more efficient management of rotator cuff pathology. It is quick, inexpensive, safe, and tolerated by claustrophobics. However, accuracy is operator-dependant;[22,29,32] therefore, ultrasound has not replaced MRI as the imaging modality of choice in most clinics or centers to assess the rotator cuff. In addition, MRI remains a more accurate method to detect muscle atrophy and associated intraarticular pathology, including glenoid labrum tears and glenohumeral ligament injuries.

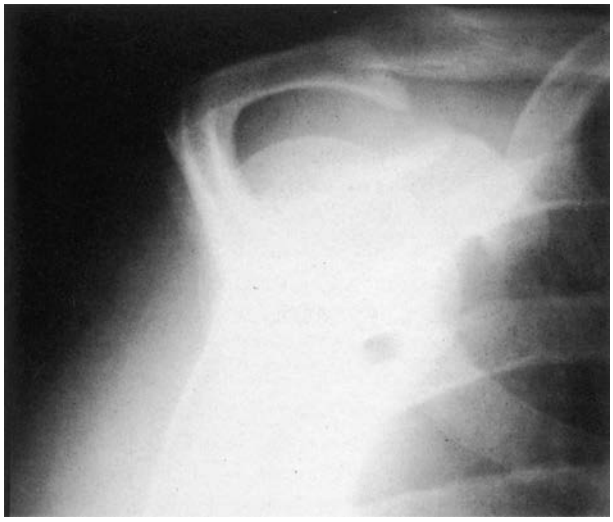


Figure 2-7 Supraspinatus outlet view demonstrates a prominent anterior acromion in a 31-year-old former college outfielder. This can be a clue that despite the patient's young age, extrinsic impingement may be involved.

MRI is the imaging study of choice to assess the rotator cuff in many centers. The accuracy in detecting full-thickness cuff tears has been reported between 93% and 100% (Figs. 2-11 and 2-12). Partial-thickness tears are less accurately detected and are more dependent on the

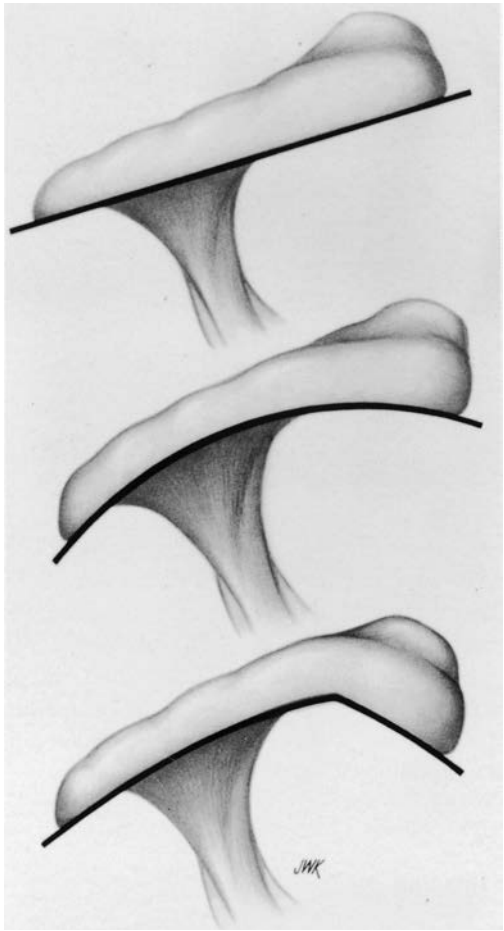


Figure 2-8 Acromion morphology as described by Bigliani et al.[6]: *type I* flat, *type II* curved, and *type III* hooked.

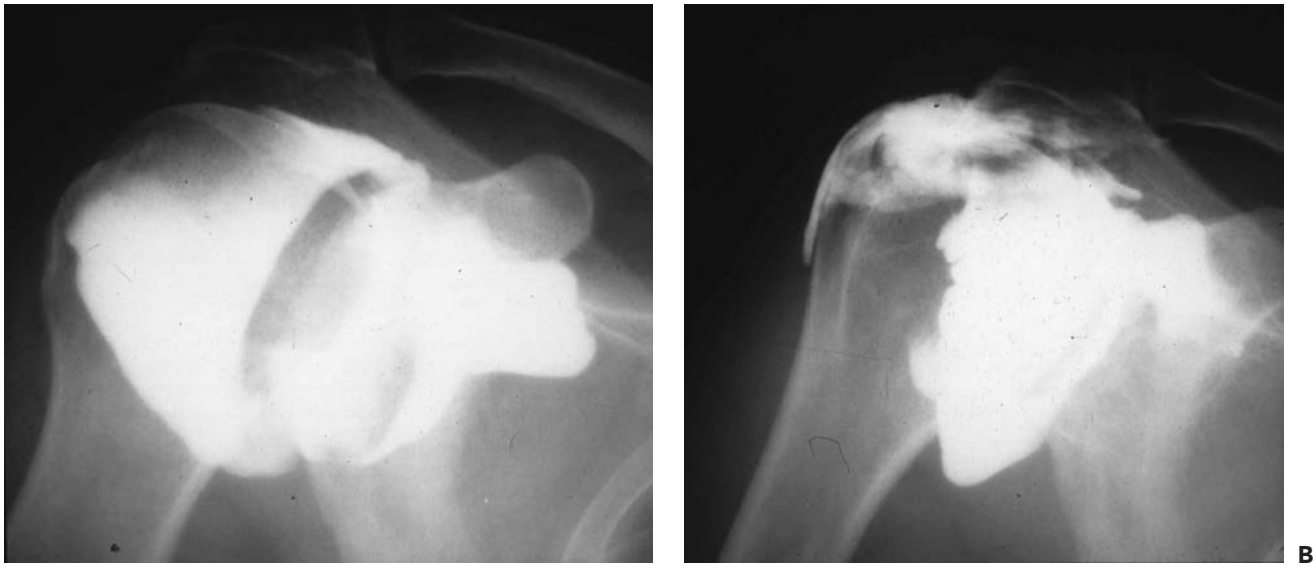


Figure 2-9 **(A)** Normal and **(B)** abnormal single contract arthrogram demonstrating a full-thickness tear of the rotator cuff.

technique used[12] (Fig. 2-13). The main advantage of MRI, however, is the wealth of information gained. The quality of the rotator cuff muscles, size of the tear, amount of retraction, involvement of the biceps tendon, and partial-thickness cuff tears can clearly be determined (Figs. 2-11, 2-12, 2-13, and 2-14). Because many surgeons use different approaches depending on tear size and muscle quality, this information is of value. Even when the approach will not change, many patients wish realistic projections of time in the hospital, postoperative restrictions, and length of rehabilitation to prepare themselves. Finally, some patients base their decision whether to undergo an operation at all on what functional gains can reliably be expected, and an MRI scan of cuff muscle atrophy can help the surgeon give an accurate prognosis. The degree of muscle atrophy has been found to correlate with functional result after rotator cuff repair, with worse function associated with greater atrophy.[15,16,26]

In the experience of the authors, a two-tendon tear with moderate to severe atrophy with retraction medial to the midlevel of the humeral head is often not repairable, and when repairable has a very high rate of incomplete healing or not healing at all of the repaired tendon. In these cases when the patient still has reasonable active elevation and

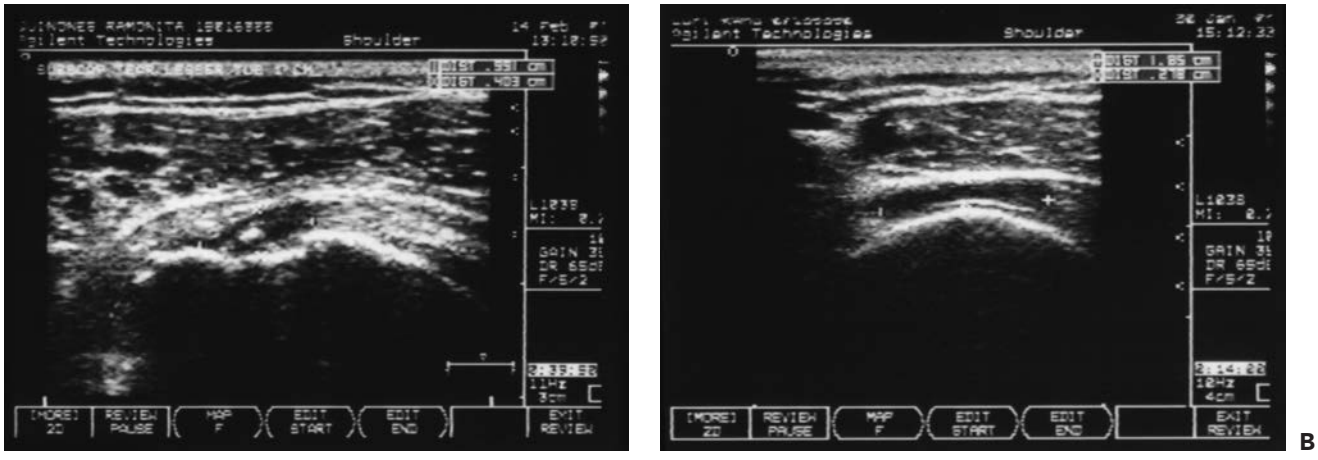


Figure 2-10 **(A)** Ultrasonographic examination in coronal plane (longitudinal) showing full-thickness rotator cuff tear of the supraspinatus tendon (*asterisk*). **(B)** Ultrasonographic examination in the sagittal plane (transverse) showing the same full-thickness rotator cuff tear of the supraspinatus (*asterisk*).

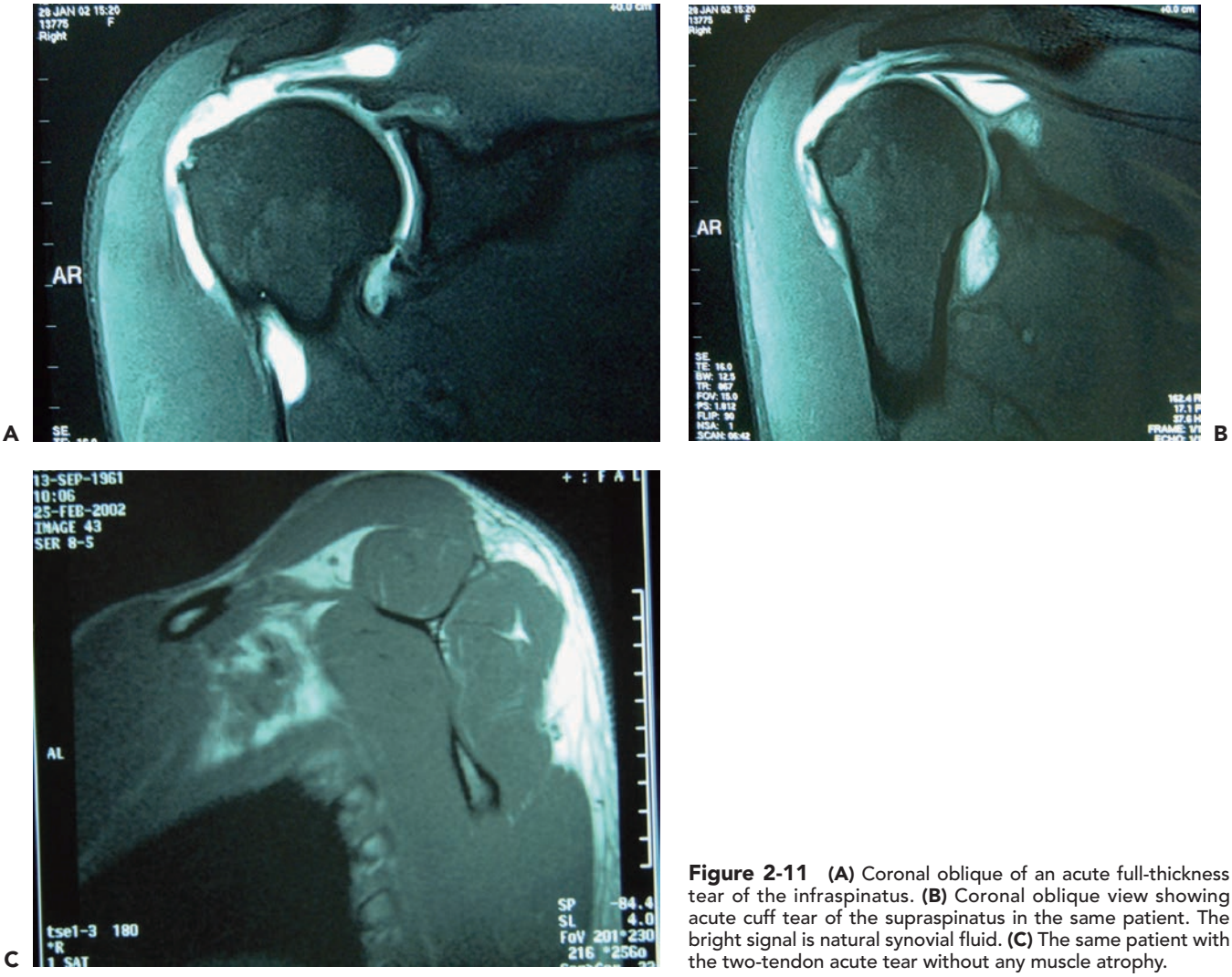


Figure 2-11 **(A)** Coronal oblique of an acute full-thickness tear of the infraspinatus. **(B)** Coronal oblique view showing acute cuff tear of the supraspinatus in the same patient. The bright signal is natural synovial fluid. **(C)** The same patient with the two-tendon acute tear without any muscle atrophy.

lower functional demands, a more limited-goals arthroscopic débridement surgery or an arthroscopic repair may be a better option and more extensive open procedure. On the other hand, when these MRI findings exist and there is poor function in a more youthful and active patient, a muscle transfer surgery should be discussed when an open surgical repair is planned. When the tear is found to be not fully repairable, or if fully repairable under some tension and with a thin tendon, a primary muscle transfer is recommended.

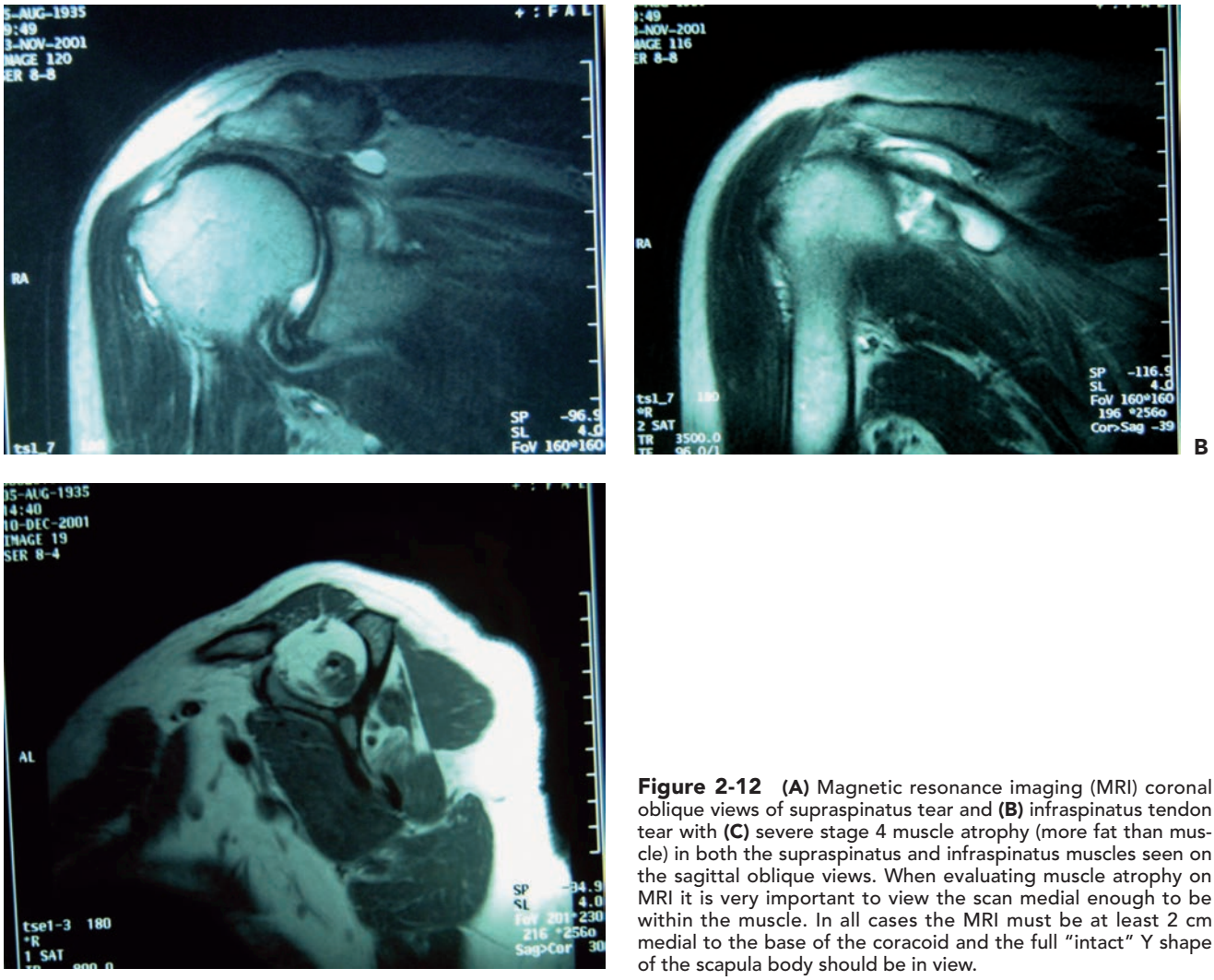
The main disadvantages of MRI are its cost, patient tolerance, the inability to utilize it in patients with a pacemaker, metal in the eye, aneurysm clips, and the difficulties posed to patients with claustrophobia.

The use of arthrogram in conjunction with MRI can increase the sensitivity of detecting full-thickness rotator cuff tears in the postoperative setting. However, the routine use of MRI arthrogram for diagnosis in the primary setting may not be justified because of its invasive nature and in this setting does not significantly improve the accuracy of a well-performed MRI.

The decision regarding whether to utilize dynamic ultrasound versus MRI should be based on the experience of the examiner with both imaging techniques, the importance of gaining additional clinical information regarding muscle atrophy and lesions of the glenoid labrum, patient tolerance, and cost.

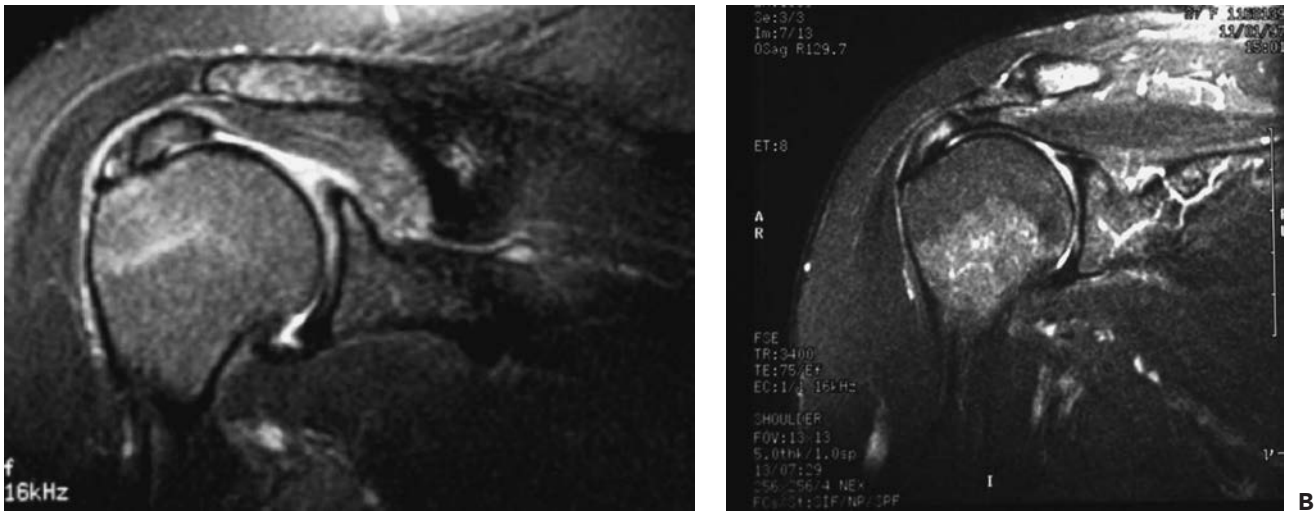
NONOPERATIVE TREATMENT

Rotator cuff pathology is a common cause of shoulder pain, with a reported incidence of rotator cuff tears ranging from 5% to 40%, with an increased incidence in older patients.[11,17,26,28,34,38,41,48] Clearly not all patients with rotator cuff tears are disabled by this condition. McLaughlin noted approximately 25% of cadavers studied had a rotator cuff tear and hypothesized that not all of



A B C

Figure 2-12 **(A)** Magnetic resonance imaging (MRI) coronal oblique views of supraspinatus tear and **(B)** infraspinatus tendon tear with **(C)** severe stage 4 muscle atrophy (more fat than muscle) in both the supraspinatus and infraspinatus muscles seen on the sagittal oblique views. When evaluating muscle atrophy on MRI it is very important to view the scan medial enough to be within the muscle. In all cases the MRI must be at least 2 cm medial to the base of the coracoid and the full "intact" Y shape of the scapula body should be in view.



A B

Figure 2-13 **(A,B)** Magnetic resonance imaging coronal oblique views showing a high-grade partial-thickness rotator cuff tear.

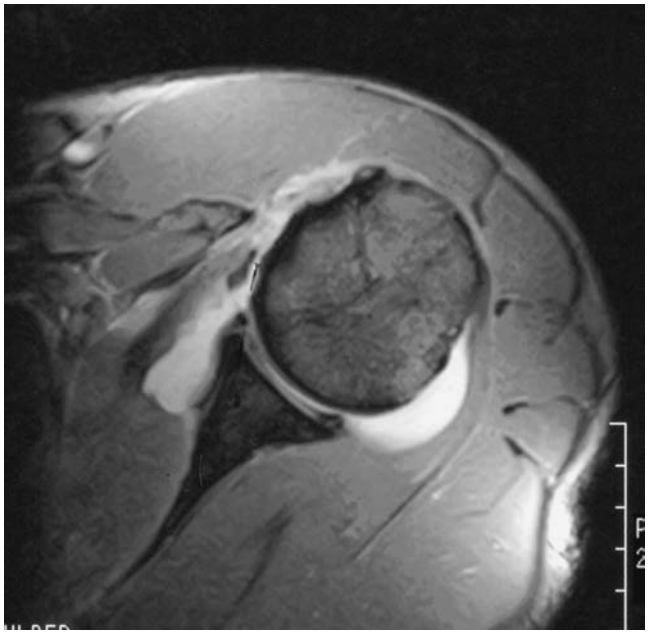


Figure 2-14 Axial magnetic resonance image showing an acute full-thickness tear of the subscapularis with a dislocated biceps tendon. Whenever there is subluxation or dislocation of the biceps there should be a strong suspicion for a partial- for full-thickness tear of the subscapularis. Both abnormalities are best viewed on the axial T2 weighted images.

these had been symptomatic in life,[30] as has been shown in more recent MRI studies of asymptomatic patients.[40] The aim of nonoperative treatment, then, is to help a patient with symptomatic rotator cuff disease become asymptomatic.

A review of the literature suggests that nonoperative treatment of rotator cuff tears is successful in 33% to 92% of cases[7,8,19,24,43,44,48] with most studies reporting a satisfactory result in approximately 50% of patients. Boker and coworkers[7] reported on 53 patients with documented rotator cuff tears undergoing nonoperative treatment at an average follow-up of longer than 7 years. Seventy-five percent of patients had satisfactory pain relief, particularly those presenting after an acute injury. Patients with long-standing pain (over 6 months) did not respond well to nonoperative therapy. Wirth and coworkers[49] reported on 60 patients with documented rotator cuff tears, and at a minimum of 2 years follow-up, only 62% of patients had a satisfactory result. Furthermore, only 4% of the patients were rated as excellent.

Authors' Preferred Treatment

In the authors' experience the patient that is most suitable and responsive to nonoperative management are those patients with chronic attritional rotator cuff tears that are limited to one to one and a half tendons. The onset of these patients' symptoms is not associated with a significant traumatic event. Generally these patients are over the age of 60 and are less active than their younger or more active counterparts. Patients particularly suited for nonoperative management have pain as the primary reason for functional weakness and will often be able to actively elevate their arm to at least shoulder height after a lidocaine injection to the subacromial space. In patients that are active with higher functional demands, regardless of age, and a documented acute full-thickness cuff tear, greater than 2 cm without atrophy on MRI and a clear history of trauma are best treated with earlier (less than 3 months from injury) surgical intervention.

Our own approach would be to recommend nonoperative treatment for patients with rotator cuff disease who present with pain without dramatic or progressive weakness. Treatment would be instituted after a history, physical examination, and plain radiographs. Unless there was diagnostic confusion, we would not image the cuff initially, but rather would consider an MRI later if this approach had failed, once surgery was being considered. Patients with weakness, especially if the onset was sudden after an injury, are imaged more expeditiously.

Patients are taught a home exercise program and sent to physical therapy for supervision and education.[24,33] Also instituted is a course of oral nonsteroidal antiinflammatory medications, heat application, and modification of activities to eliminate offending motions. Initial exercises aim at eliminating any subtle stiffness (especially posterior capsular tightness, which can exacerbate impingement) and strengthening the rotator cuff, initially in nonimpingement arcs of motion, and parascapular muscles. The patient's progress is monitored after 4 to 6 weeks. If adequate progress has not been made, a subacromial injection may be considered, especially if pain is limiting the patient's ability to perform exercises. We prefer a mixture of 3 mL of lidocaine (1%), 3 mL of bupivacaine (0.25%), and a depot corticosteroid (usually 8 mg of dexamethasone acetate in 1 mL). This can be very helpful,[6] even though pathologic studies have shown that true inflammation is rarely present in degenerative rotator cuff disease.[52] This injection is occasionally repeated after a few months, but repeated injections are not used.

For patients with pain as their chief complaint, rather than weakness, and who fulfill the criteria noted previously, this type of program is continued for 4 to 6 months before an MRI is obtained and surgical options are discussed. When weakness is prominent or progressive and fulfills the criteria noted previously, this process is accelerated and MRI or ultrasonographic imaging may be performed at the time of the initial evaluation. A common presentation is that of sudden loss of strength after a relatively trivial injury. Because splinting from pain (e.g., from hemorrhagic bursitis) can simulate weakness, we would generally start with a therapy program if the lag signs are minimally positive or negative and the apparent weakness is improved with a lidocaine

injection to the subacromial space. However, if there has not been dramatic improvement after a few weeks, an MRI is obtained. If a cuff tear is found of sufficient size to explain the weakness, timely repair is considered,[2] for it is likely that this is an acute tear, or at least an acute extension of a prior small, more chronic tear. In these cases the MRI will often show an inconsistency between the size of the tear and the muscle atrophy (e.g., the tear involves the supraspinatus and infraspinatus but only the supraspinatus as significant atrophy, suggesting that the infraspinatus tear is more recent, representing an acute extension of the chronic tear). If the tear size seems inconsistent with the physical findings, other causes of weakness, especially a nerve injury, should be considered. Occasionally an older patient will present with dramatic weakness after a dislocation, and both a cuff tear and a brachial plexopathy will be present, and their relative contributions to the patient's weakness may be unclear. However, because waiting for the nerve lesion to resolve would likely allow irreversible atrophy of the cuff muscles to develop, it seems prudent to recommend early cuff repair in most of these patients.

SURGICAL MANAGEMENT

Surgical Indications

The presence of a rotator cuff tear is not necessarily an indication for surgery. As previously mentioned, MRI and cadaver studies have shown asymptomatic patients to have cuff tears.[40] The indications for surgical repair of rotator cuff tears are, therefore, the presence of pain or functional deficits that interfere with activities and have not responded to conservative measures. Most surgeons would continue nonoperative treatment for at least 3 to 4 months before considering repair; when weakness is prominent or progressive, more timely repair may be considered.

More recently, a more proactive approach has been advocated in patients with full-thickness rotator cuff tears. (This is particularly the case in younger patients who put greater functional demands on their shoulders and who desire restoration of full shoulder strength.) The rationale for early repair stems from the fact that nonoperative management can lead to tear progression and irreversible changes to the muscle–tendon unit, including muscular atrophy, tendon retraction, and tissue thinning. A natural history study demonstrated that symptoms can develop in patients with previously asymptomatic rotator cuff tears. There was a risk for tear size progression over time with a significant increase in pain and decrease in the ability to perform activities of daily living.[49] Bassett and Cofield reported that in patients who have an acute injury and a full-thickness rotator cuff tear, repair within the first 3 weeks resulted in the best surgical outcome.[2]

Another indication for early repair is an acute subscapularis rupture. Patients older than 40 years who sustain a shoulder dislocation are at increased risk for rotator cuff tear.[37] These patients should be examined for subscapularis injury. If the patient exhibits internal rotation weakness, increased passive range of external rotation compared to the opposite arm, or a positive lift-off sign or abdominal compression test, he or she should be imaged to rule out a subscapularis injury. After the subscapularis ruptures, it can retract medially under the brachial plexus. It is easier to dissect the subscapularis, and an arthroscopic repair may be more feasible during the first several weeks after rupture. Scarring of the tendon to the anterior joint capsule and axillary nerve makes late repair technically more challenging and increases the risk of nerve injury.

Surgical treatment should also be considered in patients with large or massive tears that are symptomatic despite conservative management. While these tears are at greatest risk for structural failure of the repair, an attempt at rotator cuff repair can decrease pain and improve function and strength.[25] In cases of tears that involve two or more rotator cuff tendons, healing of the anterior and posterior cuff musculature can restore a balanced force couple and help reestablish the ability to raise the extremity.

CONCLUSION

Accurate diagnosis of rotator cuff pathology is essential to formulate a treatment algorithm. The mechanism and chronicity of injury and physical examination findings aid in determining the severity of the injury. Imaging studies can assess the specific tendon involvement and identify prognostic factors for success of operative or nonoperative treatment. An accurate diagnosis can assist in the determination of the best surgical or nonsurgical approach and guide rehabilitation.

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3 Management of Rotator Cuff Disease: Intact and Repairable Cuff

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Laurent Lafosse *Evan L. Flatow*

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INTRODUCTION

In the 19th century the cause of chronic shoulder pain was poorly understood, and most cases were lumped together and vaguely ascribed to "periarthritis."[64] In a series of papers beginning in 1904, Codman shifted attention away from the glenohumeral joint and toward the subdeltoid bursa and its contents, especially the supraspinatus tendon.[37–50] In 1911 he reported his first repair, performed in 1909, of a full-thickness supraspinatus tear.[44] Although scattered reports of procedures that included repair of the tendons of the short rotators have been identified in the older literature,[29,177] Codman's reports and lectures eloquently presented the clinical and pathologic findings of rotator cuff injuries and influenced a generation of orthopedic surgeons on the importance of these disorders. The field was further advanced by McLaughlin, who published a series of practical reports on the treatment of rotator cuff tears over a 30-year period.[140–143]

Codman emphasized the role of trauma in causing rotator cuff tears, and disputed Meyer's theory of attrition.[48,146] Other authors recognized that the acromion could pinch underlying structures and advocated complete or lateral acromionectomy[6,103,216] for a variety of painful conditions, usually when the cuff was intact. Neer's report in 1972 described the impingement syndrome and its role in rotator cuff disease.[154] He identified the anteroinferior acromion as the principal area of pathology, and advocated anterior acromioplasty to enlarge the subacromial space and decompress the rotator cuff.[154,155] Neer argued that total acromionectomy was unnecessary, and in fact, was deleterious to shoulder function.[158] He thus advocated a reshaping (without removal) of the anteroinferior acromion, preservation of the deltoid, and mobilization and repair of the tendons. These

principles led to a revolution in rotator cuff surgery. The results of rotator cuff repairs had been unpredictable and often disappointing before 1970, with unsatisfactory results reported in as many as 26% to 46% of patients.[11,23,44,48,49,60,94,112,221] More recent experience, using techniques that follow the principles established by Neer, have documented predictably satisfactory results for pain relief and function.[1,10,16,35,51,53,54,69,76,110,149,155–157,165,173,179,183,184]

The advent of arthroscopy has had a dramatic effect on the evaluation and treatment of rotator cuff pathology.[30,70,71,72] The combination of the arthroscope's ability to routinely visualize the glenohumeral joint as well as the undersurface of the rotator cuff tendon and the information gained from the widespread use of magnetic resonance imaging (MRI) has uncovered a bewildering array of "lesions." Diagnostic accuracy has been improved and the new information has led to a better understanding of the pathogenesis of rotator cuff injuries, the frequency of asymptomatic lesions, and the causes of pain in the absence of a full-thickness rotator cuff tear. Clear value has been demonstrated for the therapeutic value of arthroscopic techniques.[87,226] Arthroscopic anterior acromioplasty, arthroscopic rotator cuff repair, and arthroscopic-assisted "mini-open" repair are new tools available to the orthopedic surgeon for the care of rotator cuff disorders.

The anatomy, biomechanics, pathoetiology, and classification of rotator cuff tears are discussed in Chapter 1. Chapter 2 discusses the diagnosis of rotator cuff tears, so these areas will be mentioned only when necessary to place surgical principles in context. This chapter will focus on the reparable (or intact) cuff tendon. The management of irreparable tears will be discussed in Chapter 4, as will biceps lesions in Chapter 7, complications of cuff repairs in Chapter 5, and arthritis with cuff deficiency in Chapter 23.

NONOPERATIVE TREATMENT

Rotator cuff pathology is a common cause of shoulder pain, with a reported incidence of rotator cuff tears ranging from 5% to 40%, with an increased incidence in older patients.[62,99,125,133,155,178,201,222] Clearly not all patients with rotator cuff tears are disabled by this condition. McLaughlin noted approximately 25% of cadavers studied had a rotator cuff tear and hypothesized that not all of these had been symptomatic in life,[141] as has been shown in more recent MRI studies of asymptomatic patients.[198] The aim of nonoperative treatment, then, is to help a patient with symptomatic rotator cuff disease become asymptomatic.

A review of the literature suggests that nonoperative treatment of rotator cuff tears is successful in 33% to 92% of cases,[19,21,24,91,96,99,174,175,187] with most studies reporting a satisfactory result in approximately 50% of patients. Boker and coworkers[21] reported on 53 patients with documented rotator cuff tears undergoing nonoperative treatment at an average follow-up of longer than 7 years. Seventy-five percent of patients had satisfactory pain relief, particularly those presenting after an acute injury. Patients with long-standing pain (over 6 months) did not respond well to nonoperative therapy. Wirth and coworkers[222] reported on 60 patients with documented rotator cuff tears, and at a minimum of 2 years follow-up, only 62% of patients had a satisfactory result. Furthermore, only 4% of the patients were rated as excellent.

Bartolozzi et al. reported on 136 patients treated with nonoperative modalities with an average 1.5-year follow-up.[9] Seventy percent of the patients had excellent or good results. Prognostic factors for success included tear size less than 1 cm and symptom duration less than a year. Goldberg et al. treated 46 patients with a full-thickness tear with nonoperative modalities.[96] After an average follow-up of 2.5 years, 59% of the patients experienced an improvement in their symptoms, 30% of patients got worse, and 5% stayed the same. The ability to sleep on the affected side and the ability to place the hand behind the head were significantly improved.

Authors' Preferred Treatment

Our own approach would be to recommend nonoperative treatment for patients with rotator cuff disease who present with pain without dramatic or progressive weakness. Ideally patients treated nonoperatively have chronic tears limited to the supraspinatus tendon or have irreparable tears with at least shoulder-level active elevation and lower demands for lifting and reaching activities of more than a few pounds above shoulder level. Treatment would be instituted after a history, physical examination, and plain radiographs. Unless there was diagnostic confusion, we would not image the cuff initially, but rather consider an MRI later if this approach had failed, once surgery was being considered. Patients with weakness, especially if the onset was sudden after an injury, are imaged more expeditiously. In these cases an acute full-thickness tear is suspected and in most healthy patients a repair of the acute tear is preferred over prolonged nonoperative treatment, which in some cases may result in a much more difficult repair, less likelihood for healing, and sometimes an irreparable tear.

Patients are taught a home exercise program and are sent to physical therapy for supervision and education.[121,152] Also instituted is a course of oral nonsteroidal antiinflammatory drugs, heat application, and modification of activities to eliminate offending motions. Initial exercises aim at eliminating any subtle stiffness (especially posterior capsular tightness, which can exacerbate impingement) and strengthening the rotator cuff and parascapular muscles. The patient's progress is monitored after 4 to 6 weeks. If adequate progress has not been made, a subacromial injection may be considered, especially if pain is limiting the patient's ability to perform exercises. We prefer a mixture of 3 mL of lidocaine (1%), 3 mL of bupivacaine (0.25%), and a depot corticosteroid (usually

8 mg of dexamethasone acetate in 1 mL). This can be very helpful,[19] even though pathologic studies have shown that true inflammation is rarely present in degenerative rotator cuff disease.[213] This injection may be repeated after 2 to 3 months, if the initial injection was helpful, but more than two injections a year are discouraged.

For patients with pain who still have good functional use of the arm with minimal weakness, this type of program is continued for 4 to 6 months before an MRI is obtained and surgical options are discussed. When weakness is prominent or progressive, this process is accelerated. A common presentation is that of sudden loss of strength after a relatively trivial injury. Because splinting from pain (e.g., from hemorrhagic bursitis) can simulate weakness, we would generally start with a therapy program. However, if there has not been dramatic improvement after a few weeks, an MRI is obtained. If a cuff tear is found of sufficient size to explain the weakness, timely repair is considered,[10] for it is likely that this is an acute tear, or at least an acute extension of a prior small tear. If the tear size seems inconsistent with the physical findings, other causes of weakness, especially a nerve injury, should be considered. Occasionally an older patient will present with dramatic weakness after a dislocation, and both a cuff tear and a brachial plexopathy will be present, and their relative contributions to the patient's weakness may be unclear. However, because waiting for the nerve lesion to resolve would likely allow irreversible atrophy of the cuff muscles to develop, it seems prudent to recommend early cuff repair in most of these patients.

SURGICAL MANAGEMENT

Surgical Indications

The presence of a rotator cuff tear is not necessarily an indication for surgery. As previously mentioned, MRI and cadaver studies have shown asymptomatic patients to have cuff tears.[198] The indications for surgical repair of a chronic rotator cuff tear are the presence of pain or significant functional deficits that have not responded to 2 or 3 months of conservative measures. In a younger and active (work, sports, or hobbies) patient, an acute full-thickness tear is an indication for early surgery without any trial of nonoperative treatment. Bassett and Cofield reported that in patients who had an acute injury and a full-thickness rotator cuff tear, repair within the first 3 weeks resulted in the best surgical outcome.[10] Specific indications should be tailored to the pathology and individual patient factors.

Surgery for Subacromial Impingement, Cuff Intact

Subacromial impingement results from irritating contact between the rotator cuff and biceps tendon and the coracoacromial arch. The various causes of this syndrome, as well as the pathomechanics involved, are reviewed in Chapter 1. Whether or not impingement is thought to be primary or secondary in a particular patient, if a prominent anterior acromion is felt to be contributing to pain and tendon injury at the time that surgery is considered, then an acromioplasty is considered. Open acromioplasty[154] has been an effective procedure, with long-term satisfactory results ranging from 80% to 90% in most series.[102,108,155,183]

In 1985 Ellman introduced the technique of arthroscopic subacromial decompression.[66] His preliminary findings were that the results of arthroscopic subacromial decompressions are comparable with open decompressions. In a follow-up study,[67] he demonstrated an 88% satisfactory outcome for arthroscopic subacromial decompression. Similarly, in a prospective, randomized study, Sachs and associates[194] found that patients having an arthroscopic acromioplasty did better in the first 3 months following surgery than did patients undergoing an open procedure. After 3 months the two groups were equal. Long-term follow-up showed no difference between the two procedures, with an overall success rate of 90%. These findings are consistent with other reports in the literature.[3,71,71,86,175,192,193,205]

Open Acromioplasty

Open acromioplasty should follow the principles as described by Neer.[154] The area of greatest impingement is along the undersurface anterior and lateral portions of the acromion. An anterior–inferior acromioplasty as described by Neer is therefore the preferred technique. In this technique, the anterior deltoid is sharply dissected from the anterior acromion with the underlying coracoacromial ligament (CAL). It is important to preserve the CAL length so that when the deltoid is reattached to the acromion the CAL is sutured back to the anterior acromion margin. The principle of an anterior–inferior acromioplasty is to preserve the normal anterior–posterior dimension of the acromion. When a large acquired osteophyte is formed within the CAL, this abnormal portion is removed but the natural anterior–posterior dimension of the acromion is preserved. Preserving the normal acromion dimension and suturing the CAL back to or near the anterior acromion is believed to decrease the incidence of superior escape of the humeral head, which can occur when there is a large or massive rotator cuff defect and loss of the depression and containment function of the rotator cuff. It should also be understood that some patients with rotator cuff tears (full thickness or partial thickness) do not have acquired spurs, nor do they need acromioplasty.

Arthroscopic Acromioplasty

Arthroscopy may be performed in either the lateral or beach-chair position. The beach-chair position allows for

scalene anesthesia (awake patients do not tolerate the lateral position well), provides a standard anatomic orientation (same as open cases), facilitates conversion to an open procedure, and avoids the risks of fixed traction.[202] The torso is angled approximately 60 degrees from the horizontal plane.[182,226] A head rest that allows access to the superior and posterior aspects of the shoulder is used. The arm is draped free, allowing shoulder rotation, extension, and elevation. Two small towels are placed under the scapula to elevate the shoulder off the table.

Regional interscalene anesthesia avoids the morbidity of general anesthesia, allows improved relaxation, and facilitates outpatient surgery.[25,180] There is a misperception among some surgeons that patients are loath to undergo a scalene block. Although it is true that patients often express unease with being awake during surgery, they generally become highly accepting when reassured that they will be as sedated as they wish, especially if reminded about the risks of general anesthesia.

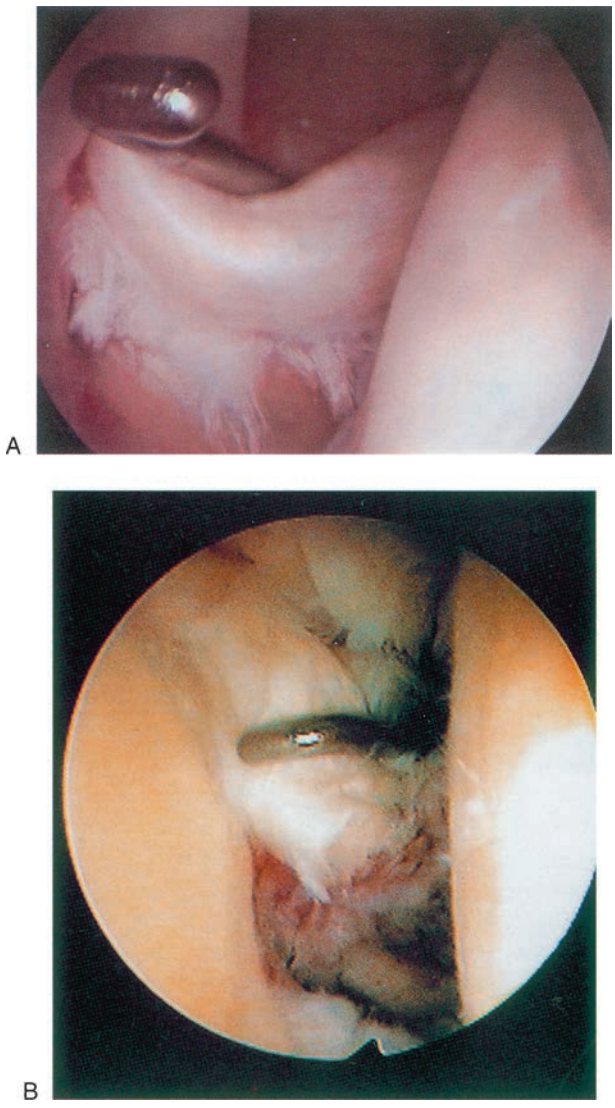

Figure 3-1 **(A)** Partial biceps tear seen on glenohumeral arthroscopy as the biceps is pulled down into the joint. This will be débrided. **(B)** Proximal stump of a torn biceps tendon. This will be resected back to the base.

Surgical Technique for Arthroscopic Acromioplasty

We prefer to perform routine decompressions arthroscopically in all patients when there is a significant subacromial impingement lesion and an intact or partial-thickness cuff tear. The procedure begins with glenohumeral joint inspection. Any inflamed synovium should be débrided and glenohumeral pathology addressed as indicated[57] (Fig. 3-1). Attention is then focused on the undersurface of the rotator cuff, and any irritation or tears evaluated.

Once the glenohumeral joint has been examined, the subacromial space is entered with the arthroscope.

Typically a thick bursitis is encountered, and this is removed with a 5.5-mm full-radius soft tissue motorized shaver. Once there is good visualization of the subacromial space, the undersurface of the acromion and CAL are inspected. Although there may be a synovitic or inflamed appearance to the ligament, the usual finding is hypertrophy of the ligament, with a degenerative, attritional lesion of the anteroinferior acromion, including frayed fibers hanging down (Fig. 3-2). In young, athletic patients there may be no bone abnormalities, but just scarring of the bursa and thickening of the coracoacromial ligament.[8] In these patients, a soft tissue decompression with débridement without release of the ligament, combined with

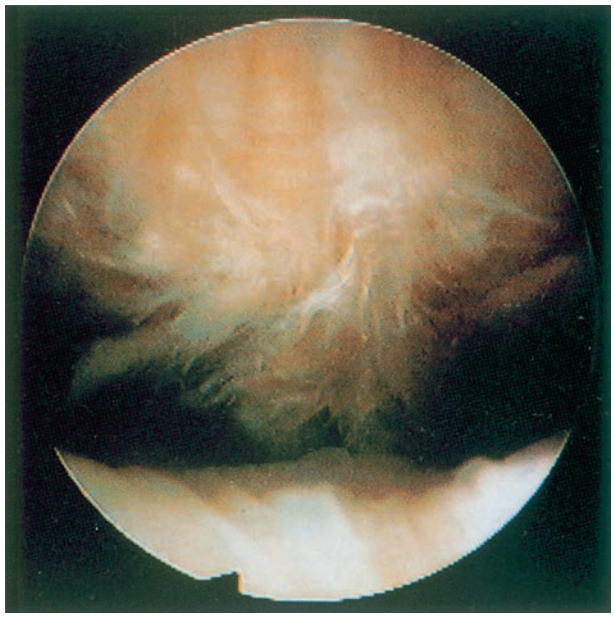

Figure 3-2 Attritional lesion of the anteroinferior acromion at the insertion of the coracoacromial ligament.

renewed rehabilitation to treat any subtle underlying instability, may be helpful.

In older patients with a more degenerative profile, the ligament is removed only from the undersurface of the acromion to visualize the acromion and its anterior margin, and an acromioplasty performed. The CAL is sequentially removed from the undersurface of the acromion using electrocautery and then freed at its margins laterally and medially. It is important to visualize the entire acromion to the junction between the anterior two-thirds and posterior one-third of its anteroposterior dimension, from the acromioclavicular (AC) joint to the lateral margin to perform an adequate acromioplasty. We routinely use the electrocautery to expose the anterolateral and lateral aspect of the acromion to better appreciate the acromial morphology and spur size. Bipolar cautery devices are the best device for this part of the procedure.

The bony acromioplasty is performed with a 5.0– to 6.0-mm tapered burr. The thickness and morphology of the acromion as well as the size of any bone spur will dictate the amount of bone removed in an individual patient. A popular technique for determining the amount of bone removal is the "cutting-block" technique[33] (Fig. 3-3). In this method the burr is brought in from the posterior portal, applied to the posterior acromion, and then advanced to plane down the anterior acromion, thereby flattening the entire acromion. This approach aims to convert the acromion to a type I after Bigliani et al.[18] However, this may remove an excessive amount of bone if the burr is brought into the subacromial space at an acute angle to the posterior surface of the acromion. In the past, the coracoacromial arch has been often thought of as a purely harmful structure, causing impingement and good for little else. Indeed, in 1984, Rockwood advocated the routine removal of the CAL at the time of any shoulder operation.[187] This encouraged, in past years, a "more is better" approach to bone removal at acromioplasty. However, contact by the acromion on the underlying rotator cuff and humerus in normal shoulders has a passive, stabilizing function.[77,79,130,151,167,215,220] In a later study simulating different amounts of bone removal at acromioplasty, smoothing the anterior third of the acromial undersurface removed all focused contact on the supraspinatus insertion, which was termed "impingement." Total flattening of the acromion was found to be not only unnecessary to relieve impingement, but also this technique destroyed much of the broad contact between the bone and rotator cuff, which was likely important for stability of the humeral head in a superior direction.[75]

Figure 3-3 Cutting block technique for arthroscopic acromioplasty. The burr is entering from posterior and the remaining acromion bone is at the anterior margin.

In addition to removing excessive bone, the arthroscopic cutting block technique may risk injuring the deltoid origin, especially in curved acromions in which a line drawn up against the posterior acromial undersurface essentially transects the anterior acromion. Indeed, some surgeons who purport to use this technique will depart from it when they notice a thin, curved acromion. We prefer to aim for opening up the subacromial space by resculpting the anteroinferior acromion so that it curves gently away from the underlying humerus and cuff. The amount of bone removal may be estimated from the preoperative outlet view and measured intraoperatively by comparison with the known diameter of an instrument (Fig. 3-4). The transition to the posterior acromion is then smoothed. All debris is removed, and the bursal space is irrigated. After decompression, the instruments are removed and portals are closed with absorbable suture. The patient is placed in a removable sling for 1 to 2 days for initial comfort. Postoperative motion exercises are generally started immediately, and progressed as tolerated.

The major advantage of arthroscopic over open decompression is that deltoid detachment is avoided.[2] Use of the arthroscope also allows inspection of the glenohumeral joint, as well as the undersurface of the rotator cuff, and any pathology encountered can then be addressed. Finally, arthroscopic decompressions are less invasive and are routinely performed on an outpatient basis. In most clinical practices it is rare to perform an acromioplasty as an isolated procedure as in the vast majority of cases there is cuff damage requiring débridement or repair.

Acromioclavicular Joint Resection

AC joint resection is performed when there is significant tenderness over the AC joint on preoperative examination, pain over the AC joint with activities, and anatomic evidence of arthritic changes on preoperative radiographs. The techniques for open or arthroscopic distal clavicle resection are detailed in Chapter 32. The authors prefer the arthroscopic approach for AC joint resection; it is performed after the acromioplasty in three steps:

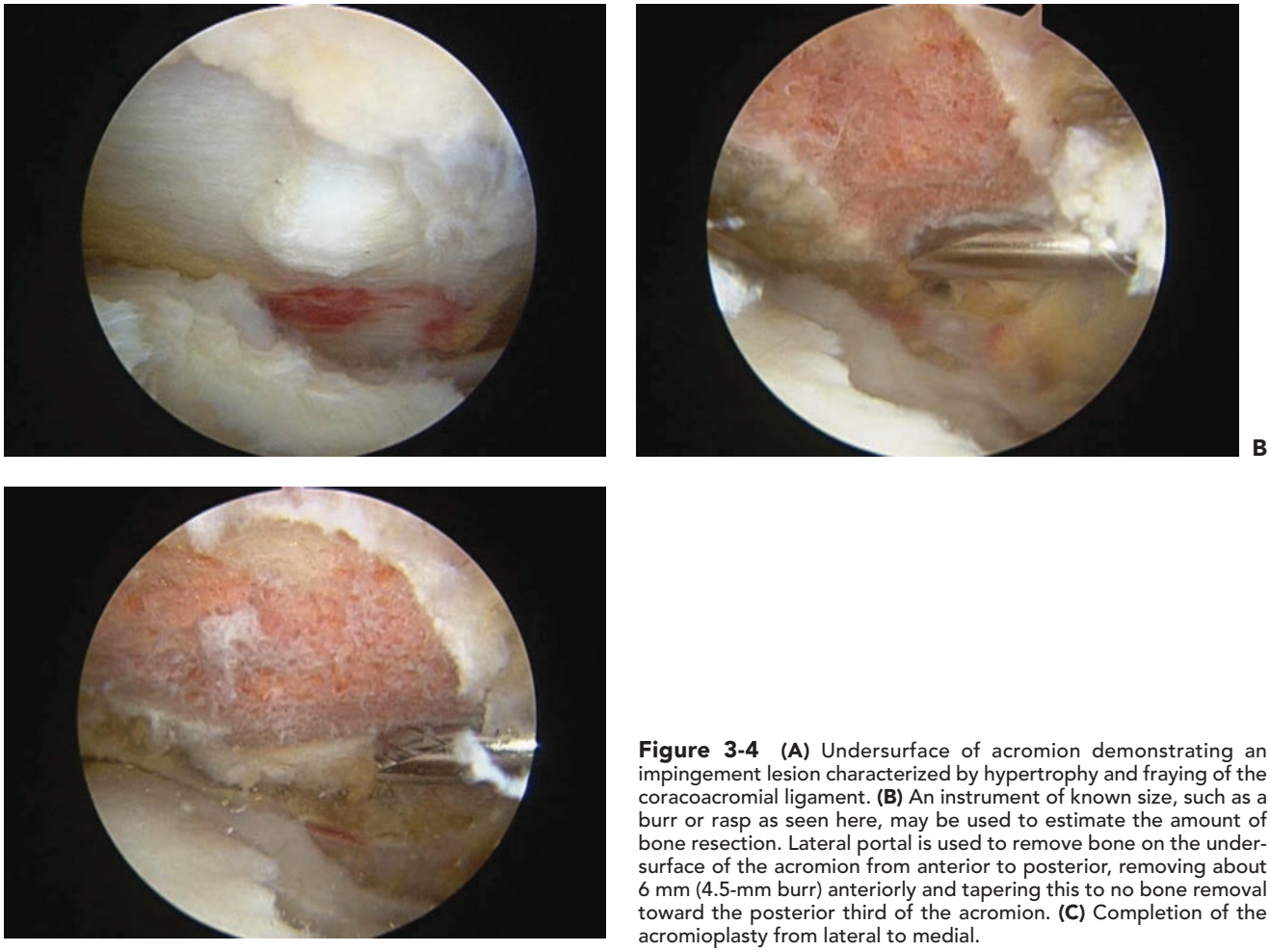

Figure 3-4 **(A)** Undersurface of acromion demonstrating an impingement lesion characterized by hypertrophy and fraying of the coracoacromial ligament. **(B)** An instrument of known size, such as a burr or rasp as seen here, may be used to estimate the amount of bone resection. Lateral portal is used to remove bone on the undersurface of the acromion from anterior to posterior, removing about 6 mm (4.5-mm burr) anteriorly and tapering this to no bone removal toward the posterior third of the acromion. **(C)** Completion of the acromioplasty from lateral to medial.

- First, using the posterolateral portal for visualization, the soft tissue and medial portion of the acromial facet of the AC joint is removed, thereby exposing the distal clavicle. The inferior osteophyte of the distal clavicle is removed.
- An anterior–superior portal is placed in the AC joint to introduce the burr. Bone is removed from both the acromion and clavicle with visualization from the lateral portal. Special care must be taken to remove the posterior and superior clavicle spur while preserving the attachment to the posterior and superior AC capsular ligaments.
- Finally the modified posterior portal is inspected to check the anterior clavicle, ensuring that the superior AC ligament is intact (Fig. 3-5).

Surgery for Partial-Thickness Rotator Cuff Tears

The literature remains unclear and somewhat confusing on the treatment of partial-thickness rotator cuff tears. Recommendations range from conservative therapy to open rotator cuff débridement and repair. Prior to arthroscopic surgery, excision and repair of significant partial cuff tears seemed logical and added little morbidity to an open procedure.[82,157] However, after the advent of arthroscopic acromioplasty, but before techniques of arthroscopic tendon repair had been developed, simple débridement with or without decompression became more widely used for partial tears and even many full-thickness tears.[58,68,72,117] Later, as data accumulated that indicated poor results after this procedure,[18,217] primary excision of the damaged tendon followed by primary tendon repair came back into vogue. This procedure became more popular thanks to the development of mini-open and arthroscopic techniques, because it avoids conversion to a full open procedure.[73] Currently, recommendations for the operative management of partial tears vary between investigators. Essentially, there are three surgical options: débridement alone, decompression and débridement, and excision of damaged tendon with primary repair (usually along with decompression). Let us examine each of these in turn.

Andrews reported good success with débridement,[4] but his population was young and athletic. It is likely that

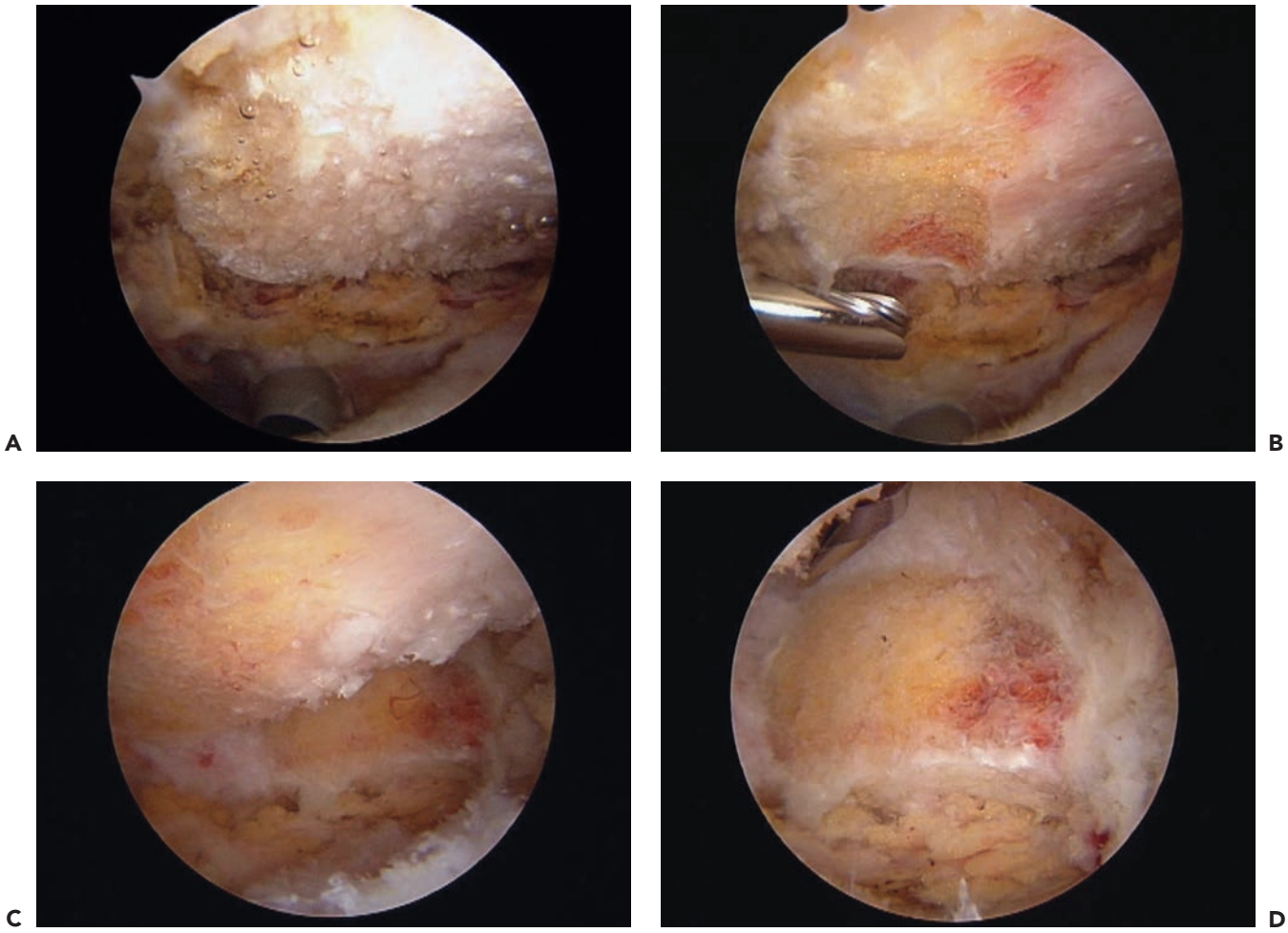

Figure 3-5 **(A)** Arthroscopic view of acromion after removal of soft tissues and coracoacromial ligament. **(B)** Lateral removal of bone (acromioplasty). **(C)** Completed acromioplasty and distal clavicle resection. **(D)** Distal clavicle resected with 360-degree view of the resected clavicle showing complete resection with intact superior and posterior acromioclavicular capsule. The burr is entering the space at the anterior-superior corner of the capsule. The inferior capsule has been removed and the arthroscope is in the subacromial space from a posterior-lateral portal.

overuse and tensile failure were involved, rather than any acromial abnormalities. Snyder and coworkers[205] reported their results on arthroscopic cuff débridement with or without subacromial decompression. They had 85% satisfactory results, with similar results between those patients having a decompression and those not having a decompression. However, patients were not randomized for decompression, but were selected on clinical criteria, likely correctly identifying patients in whom impingement was prominent. Arroyo and coworkers noted that young, overhead athletes frequently develop subacromial scarring and bursitis owing to overuse and instability, and that soft tissue cleanout of the subacromial space may be helpful.[8] Altchek and Carson studied 50 throwing athletes with anterior shoulder pain, which was refractory to nonoperative treatment,[2] and found that most had fraying of the articular surface of the cuff. Débridement of this area, combined with débridement of bursitis and CAL hypertrophy when noted, was associated with favorable results in 80% of cases.[2]

The role of internal impingement, in which the cuff undersurface abuts the glenoid rim in abduction and external rotation,[122,123,213] also adds complexity to decision making. This mechanism and other causes of cuff failure are discussed in Chapters 1 and 2. When deep-surface cuff partial tears are associated with internal impingement, most surgeons have employed simple débridement;[73] however, little data are available on the effectiveness of this approach. In some cases, anterior subluxation has been thought to play a role, and capsulorrhaphy recommended.[123] Indeed, one author has suggested that derotational humeral osteotomy be considered.[213] In any event, this group of younger patients, who are often involved in throwing sports, is a different group from that of older patients with degenerative tendon failure.

In some patients, simple débridement has been far less satisfactory. Ogilivie-Harris treated 57 partial tears with

arthroscopic débridement alone, and found that only half achieved satisfactory results.[169] In this group, acromioplasty appears to improve results.[24,68,86,170,226] Arthroscopic decompression has also been effective. Gartsman noted that 33 of 40 patients (83%) with partial-thickness tears had major improvements in their shoulders at an average of 29 months after arthroscopic acromioplasty.[86] However, not all patients do well with this approach. Altchek and coworkers noted that the results of débridement and decompression of partial tears were not as favorable as those from decompression in shoulders with intact cuffs.[3] But which subgroups of partial tears need more than decompression?

Weber felt that the degree of tendon involvement was important.[217] He reported inferior results in patients undergoing débridement and decompression of partial cuff tears that involved more than half the tendon's thickness, as compared with a mini-open approach in which the damaged tendon was excised and healthy tissue repaired side to side.[217] Interestingly, his recommendations are the same as those of Neer et al.[157] (done open), although less aggressive than Fukuda and coworkers, who performed excision of damaged tissue on most partial tears[82] and achieved a 92% success rate. Further study is needed to make definitive recommendations for treatment.

Authors' Preferred Treatment

We treat minor degrees of tendon injury in a young, athletic patient with rehabilitation. If that fails, arthroscopy is considered. This population will often have subtle degrees of instability, but if cuff and bursal pathology are the dominant findings and anteroinferior labral detachment is not found, then we would not perform a capsulorrhaphy. We would débride the partial tear and any labral fraying, and then inspect the bursa. If there is bursitis and CAL hypertrophy, then we would débride those areas, performing in effect a soft tissue subacromial decompression.

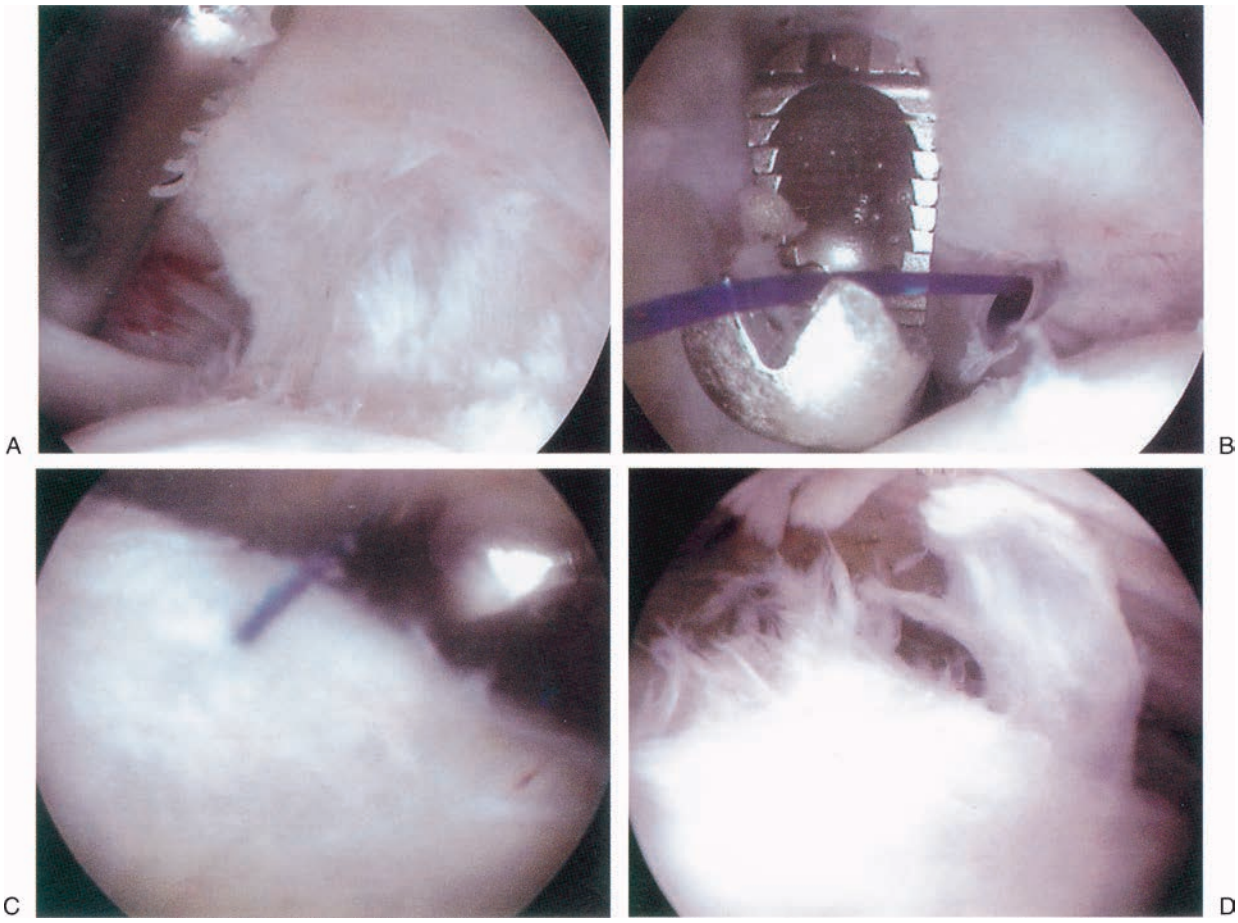

Figure 3-6 Partial-thickness tears of the rotator cuff can be treated with débridement if the tear is less than 50% of the thickness of the tendon. **(A)** Arthroscopic view from the posterior glenohumeral portal of an articular-sided partial-thickness tear of the supraspinatus. **(B)** An absorbable suture is placed through the tendon with a spinal needle to allow bursal-sided inspection. **(C)** Subacromial view of the bursal side of the tendon showing that the tear does not extend to the bursal surface. **(D)** Subacromial view of a different patient who has a partial bursal-sided tear.

For older patients with degenerative partial cuff tears who fail nonoperative treatment, we will perform a shoulder arthroscopy to determine the depth and size of the tear. If the tear is less than 50% in thickness, then we will perform an arthroscopic cuff débridement followed by an anterior acromioplasty with CAL excision (Fig. 3-6). The retracted edge of the "rim-rent" can be bulky, and may cause the tendon to buckle and jam in the subacromial space. Débridement includes trimming this edge to decrease its bulk. A colored absorbable (in case a piece breaks off) suture is passed through the partial tear with a spinal needle to aid in identifying the corresponding bursal surface of the tendon later. This will help to avoid missing a full-thickness perforation and to find cases in which the tendon is thinned from both sides. If the tear involves more than 50% of the tendon's thickness in an active patient, we will perform the arthroscopic anterior acromioplasty and arthroscopic rotator cuff repair.

SURGICAL TECHNIQUE

Partial Tear

Partial Bursal Surface Tear: Guy Rope technique

The guy rope technique is currently used only for partial superficial tears. A posterior subacromial portal and a lateral instrumentation portal are used. The greater tuberosity is abraded and a hole is drilled as lateral as possible into the border of the greater tuberosity. A metallic screw anchor loaded with a double suture is placed laterally through the strong cortical bone of the great tuberosity

Through an anterior portal a pig-tail suture passer (Linvatec) equipped with a 45-degree curved hook (left-rotated for a right shoulder and vice versa) is loaded with a single strand of PDS #0. The instrument is passed through the cuff from the superficial to the deep layer and then back again, twisting the hook, resulting in a U-suture (Fig. 3-7A). The

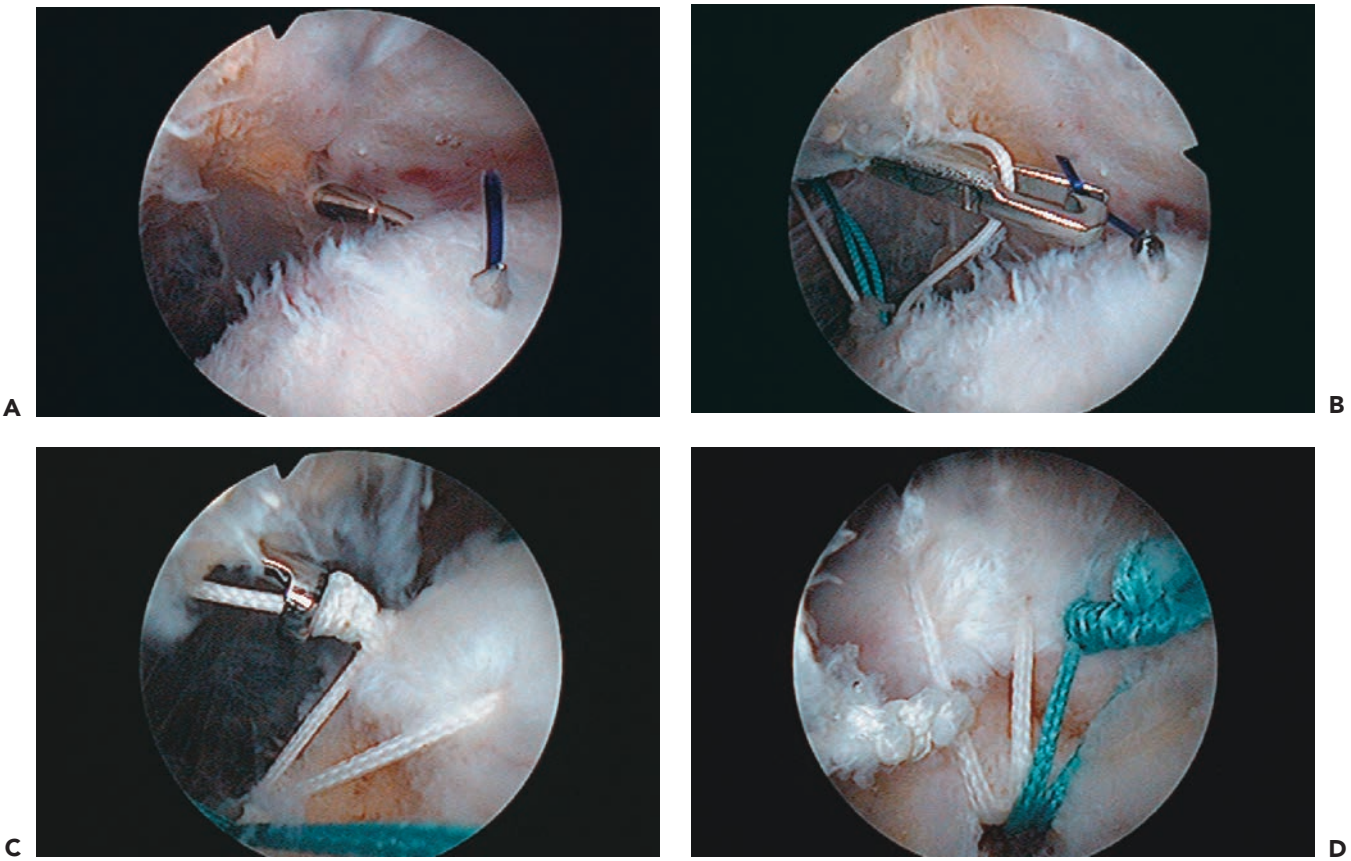

Figure 3-7 Superficial cuff tear: Guy rope suture. **(A)** Placement of PDS suture for suture relay (double pass of suture through the tendon). **(B)** After placing the anchor lateral in the greater tuberosity, one permanent suture is taken with the PDS and passed from superficial to deep within the tendon. **(C)** The same limb of the suture is passed from the acromial to articular surface and then back to the acromial surface of the tendon and the knot is tied. Both sutures are passed in this manner. **(D)** The final repair has a double grasp of the suture in the tendon (mattress suture) but the suture is passing over the top of the tendon. This method allows for greater surface area of contact between the tendon and greater tuberosity than what would be achieved with a mattress suture where both limbs of the suture are tied over the top of the tendon and both limbs contact bone from the deep side of the tendon.

PDS end is grasped at the same time as one of the two sutures is loaded on the anchor by a forceps and pulled out through the lateral portal. The PDS is used as a shuttle relay for passing the nonabsorbable suture. (Fig. 3-7B,C). Alternatively the sutures can be passed directly with suture punch instruments or grasped directly with a tissue penetrator. A sliding knot on the anterior strand of the thread is pushed on the cuff and pulled laterally to the anchor to bring the tendon to the greater tuberosity. Then the knot is secured by changing traction on the threads, and two additional half loops are pushed along the posterior thread. The second suture is passed through the tendon in the same way to obtain a double suture technique (Fig. 3-7D).

Intratendinous Tear

Only an acromioplasty is performed for this lesion because it is thought that relief of the impingement prevents further tendon damage.

Partial Articular Surface Tear

Articular surface partial tears most often affect the supraspinatus, and they can be associated with secondary biceps tendon instability when the tendon tear is located at the level of the posterior biceps pulley. This lesion is best visualized with internal rotation of the shoulder as the biceps tendon subluxates over the lateral wall of its groove (Fig. 3-8A,B).

The aim of the supraspinatus repair is to rebuild the juxtacartilaginous tendon's insertion. To reattach the supraspinatus, after cleaning the bursa and acromioplasty, the repair is managed by the intraarticular visualization "inside out" technique. A small anterosuperior portal close to the anterior corner of the acromion is placed near the rotator interval at the anterior part of the supraspinatus above the biceps groove. A 5-mm incision is made in the cuff in the midportion of the partial cuff tear. All anchors and instruments can be placed through this cuff defect, and then this defect is closed with suture at the end of the repair. In this way the intact superficial bursal surface portion of the cuff remains intact and the footprint of the cuff is reestablished medially at the margin of the articular cartilage. A metal anchor is inserted in the previously abraded area of the tuberosity, close to the cartilage and to the posterior border of the biceps groove (Fig. 3-8C).

Two techniques are possible to pass the four sutures through the tendon. A suture passer can be introduced by the lateral portal used for the acromioplasty, perforating the cuff from outside to inside, and either a PDS suture can be used as a suture relay or the suture on the anchor can be passed directly (Fig. 3-8D–G). The four threads are successively passed and pulled through the tendon. Visualization of the knot may be done on subacromial space, but secure tension is necessary to ensure good application of the cuff over the bone (Fig. 3-8H).

It is important to avoid strangulation of the biceps with the suture fixation. When the biceps pulley reconstruction is not secure or anatomic or when biceps degenerative changes are visible, then a tenodesis of the biceps is performed.

Full-Thickness Rotator Cuff Tears

History

Codman performed his first cuff repair in 1909,[44] and in 1934 he noted that 20 of 31 patients followed after repair of full-thickness rotator cuff tears obtained a good result.[48] Four years later Outland and Shepherd published a series of 12 rotator cuff repairs; satisfactory results were obtained in 67%.[171] McLaughlin, using a transacromial approach, reported 94% pain relief and 100% return to manual labor in 32 patients after cuff repair.[140] In the 1950s and 1960s, overall satisfactory results after cuff tendon repair were seen in 77% of Moseley's cases,[153] 74% of Godsil and Linscheid's cases,[94] 60% of Heikel's cases,[112] and 66% of Debeyre et al.'s cases.[60]

In 1972, Neer advocated routine anterior acromioplasty at the time of rotator cuff repair.[154] Of 20 patients with full-thickness tears treated by acromioplasty and repair, 19 (95%) achieved satisfactory results (patient satisfied, no significant pain, less than 20 degrees of limitation of elevation, and at least 75% of normal strength). In this and in other writings,[154,156–158] Neer argued for certain principles in rotator cuff surgery:

1. Reshaping rather than removing the acromion, avoiding procedures that damage the deltoid origin
2. Restoring motion
3. Releasing, mobilizing, and repairing the torn tendons
4. Surgeon-directed individualized rehabilitation

Although most modern surgeons have employed an approach similar to Neer's, there have been exceptions. Repair of the tendons without acromioplasty has been advocated,[95,168,200] as has acromioplasty without tendon repair.[189] Some have even continued to advocate acromionectomy.[23] Nevertheless, decompression and repair remains the most common treatment for full-thickness tears requiring surgery. With recent advancements in arthroscopy, many authors are advocating arthroscopic repair of rotator cuff tears. Preliminary results appear to be promising and equal to those for mini-open repairs.[87,203,210] Arthroscopic repair follows the same principles as those for open repairs, including subacromial decompression, cuff mobilization, and repair of the tendon back to the tuberosity. Technically, this can be very challenging, particularly on the upslope of an individual surgeon's learning curve.

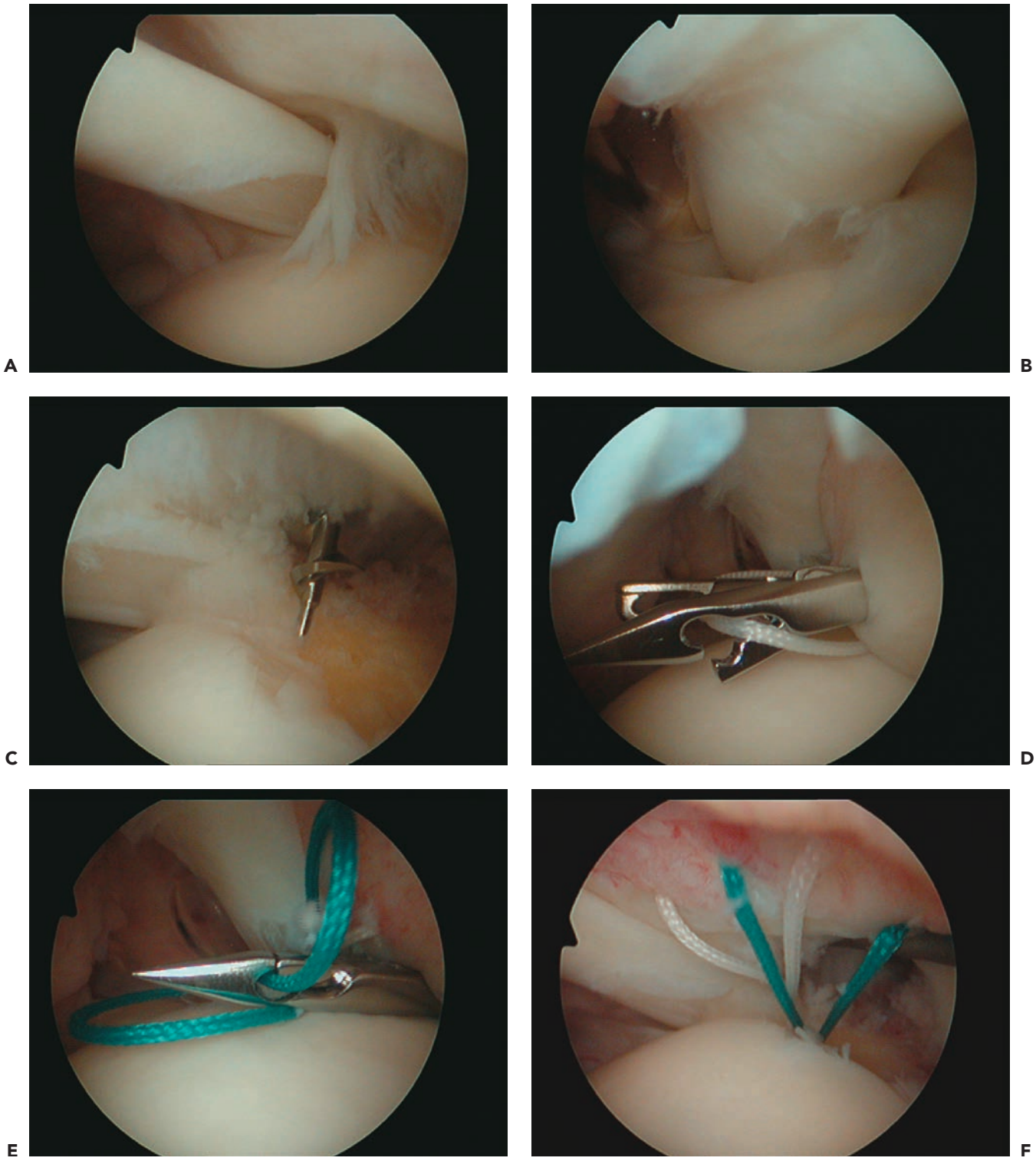

Figure 3-8 Articular cuff tear: Parachute technique. **(A)** Partial cuff tear near the biceps tendon. **(B)** Long head of the biceps with minor damage on the lateral side near the supraspinatus cuff tear. **(C)** A small defect is made in the cuff, which is placed through the defect into the lightly decorticated greater tuberosity near the articular margin. **(D)** A tissue penetrator is used to grasp the suture. **(E)** All the sutures passed. **(F)** The sutures are arranged so that when they are tied **(G),** the articular margins of the tendon are placed at the articular margin of the cuff. **(H)** Subacromial view of the repair.

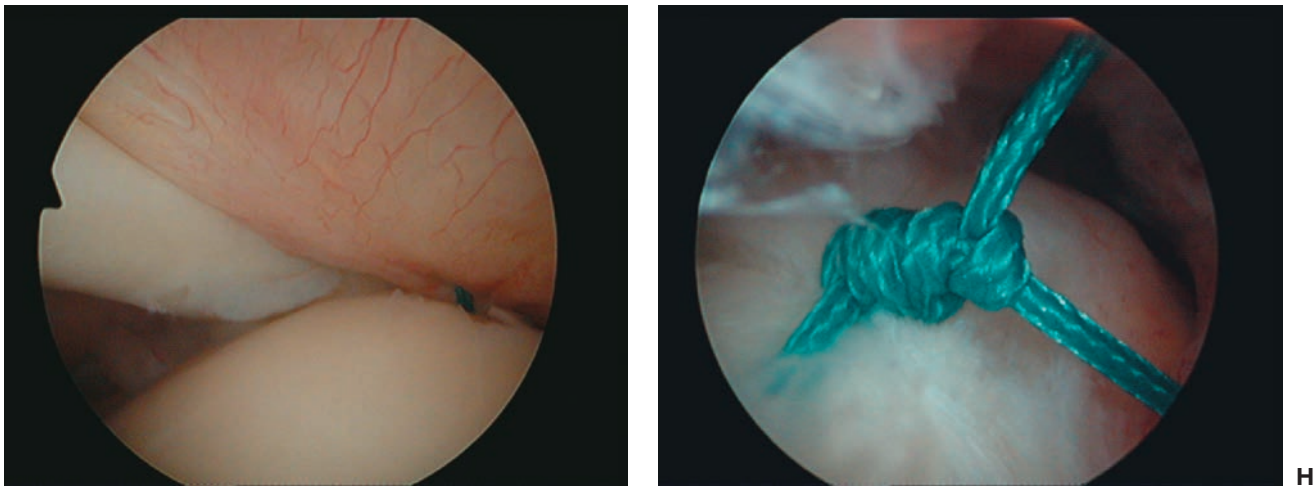

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Figure 3-8 (*continued*)

Principles of Open Rotator Cuff Repair

Approach

Although stiffness is surprisingly infrequent in shoulders with large cuff tears, it can occur.[157] Even if superior structures may be released during an open repair, the inferior capsule and axillary recess are difficult to reach through an open anterosuperior approach. Consequently, stiff shoulders are gently manipulated at the beginning of the procedure.

A variety of surgical approaches for cuff repair have been reported.[138] Norwood et al. have described a posterior approach,[168] whereas Leffert and Rowe preferred an anterior, deltopectoral exposure,[131] also advocated by Gerber et al. for subscapularis ruptures.[91] Most authors, however, have employed an anterosuperior approach through a split or takedown of the proximal deltoid.[138] In an attempt to visualize large tears with retraction, takedown of the anterior or lateral deltoid origin has been used, as have acromion-splitting approaches. However, Codman,[48] after trying various techniques, came to prefer a deltoid split combined with rotation of the head beneath the split to expose the involved area of the cuff. Diamond, in 1964, described an extensile approach for acromionectomy in which a deltoid split was taken up over the acromion, which was then exposed subperiosteally.[63] Neviaser et al. employed a split taken into the AC joint with subperiosteal reflection of the flaps to expose the tear, made easier because the distal clavicle was routinely resected.[165] However, exposure of a retracted posterior tear may be difficult with an anterior split, occasionally even requiring a supplemental posterior incision.[157] For this reason Bigliani et al. have shifted the split posteriorly by beginning it at the anterolateral corner of the acromion; this affords excellent posterior exposure[16,56] (Fig. 3-9).

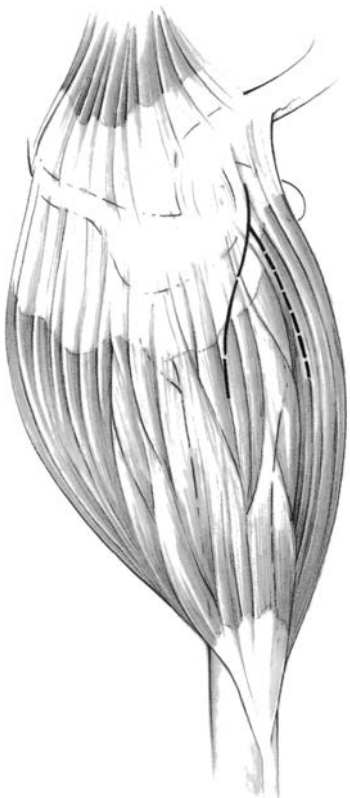


Figure 3-9 The posterior curve of the deltoid incision allows the exposure to be centered over the greater tuberosity for better access to the cuff. The dotted line demonstrates the less desirable anterior exposure provided by the older and more anterior type of deltoid incision. (From Bennett WF. Arthroscopic repair of massive rotator cuff tears: a prospective cohort with 2- to 4-year follow-up. *Arthroscopy* 2003;19(4):380–390, with permission.)

Decompression

Traditionally, decompression at the time of cuff repair has consisted of anterior acromioplasty, resection of the coracoacromial ligament, and, if needed, resection of downward-projecting acromioclavicular osteophytes.[154] It has become increasingly appreciated that the coracoacromial arch has a normal buffering role in passively resisting superior humeral translation,[75,77,79,130,151,167,215,220] especially when the dynamic stabilizing function of the cuff muscles has been lost. Wiley reported that anterosuperior subluxation could result from decompression without repair of massive cuff tears,[220] but we have noticed this also after decompression and repair when the repair fails or the muscles, although reattached, are too atrophied to generate the force necessary to center the head. Indeed, Watson, noting that his cases of cuff repair did better when the coracoacromial ligament was incised rather than resected, conjectured that coracoacromial ligament "removal may allow the strong deltoid muscle to pull the humeral head proximally," stretching and potentially damaging the cuff repair.[215]

Preservation of the coracoacromial arch for stability has been employed at the time of arthroplasty for cases of end-stage rheumatoid arthritis and cuff-tear arthropathy.[7,181] In 1991, one of us (ELF) began to preserve the coracoacromial ligament when repairing massive tears, and reported on the initial experience a few years later.[80] This has not appeared to result in postoperative impingement or inadequate pain relief. Whether this should be used for all tears or only large ones, and if the latter, what size cutoff there should be, remains unanswered.

Tendon Mobilization and Repair: Open Technique

The aim of tendon mobilization is not only to allow repair, but to free the cuff muscle–tendon units so they can glide, and to prevent postoperative stiffness.[156,157] It is unfortunate that the cuff literature has emphasized concepts such as "coverage of the head," as if cuff repair were a plastic surgery procedure. No hand surgeon would sew a graft into an old, scarred flexor tendon laceration and say that he or she had "covered" the proximal interphalangeal joint. The goal should be to restore the cuff's dynamic function as best possible. Therefore, the tendons must not only be repaired, but also freed from adhesions to surrounding structures so that the muscle–tendon unit can glide and function. Also, passive shoulder motion may be normal, despite extensive cuff scarring, because the capsule is detached from the humerus along with the cuff tendons, so motion occurs "through the tear." If the tendons (and the attached capsule) are repaired without being adequately released, a stiff shoulder may result, and stretching exercises will improve motion only by pulling out the repair.

Arthroscopic techniques have progressed to the point where experts report that the same tendon releases traditionally performed open may be accomplished arthroscopically.[81,87] A possible exception may be chronic subscapularis tears, which may have extraarticular adhesions (especially including the axillary nerve). More recently, the techniques for arthroscopic repair of subacute and chronic subscapularis tendon tears are reported to be safe and successful.

Repair of the tendon is performed with the arm at the side. Performing the repair with the arm in abduction and then placing the patient in an airplane splint will usually lead to pullout of the repair when the brace is discontinued.[163] However, protection of a repair of a large tear (performed with the arm at the side) with a brace was employed by Neer,[157] and has been more recently advocated by Gerber.[93] Recently developed braces are more comfortable than earlier models, are less likely to rotate into extension, and allow for more intermediate positions, rather than wide abduction and external rotation. It is our impression from clinical experience that bracing, or use of an abduction pillow, for at least 4 weeks after repair of chronic posterior-superior two-tendon cuff tears results in better healing. During this time it is still important and necessary to begin passive range-of-motion exercises.

Traditionally, many surgeons preferred repairing the tendon to a trough in bone.[142] Many techniques have been reported in which the tendon edge is pulled into a deep cancellous trough with mattress sutures. Several factors have contributed to a trend away from cancellous troughs. First, use of mattress sutures often allows some mobility of the free edge of the tendon, or at least some discontinuity between the repair and the tuberosity. We have preferred simple or Mason-Allen sutures that hold the tendon edge flat against the tuberosity and present a smooth surface. Second, concern for the holding power of sutures in osseous tunnels, and especially for the pullout strength of suture anchors, has prompted a desire to preserve the cortical bone at the articular–tuberosity junction. Finally, doubts have been raised as to whether, in fact, cancellous bone is a better healing bed for tendon than cortical bone—a recent study in sheep found no difference in repair strength.[206] Most surgeons freshen the bone at the articular–tuberosity junction to remove nonhealing bursal tissue and scar, but do not necessarily expose the cancellous bone. We believe that removing the degenerative soft tissue is important; then removal of some bone without creating a trough or exposing the cancellous bone of the greater tuberosity yields a bone area that will allow healing and preserve the mechanical strength of the cortical bone.

Numerous suture techniques have been described. An influential study recently suggested that modified Mason-Allen sutures provided the best holding power in a weak tendon while minimizing strangulation.[92] However, simple sutures elongate least under load, and thus gap less in a strong tendon. The most accepted arthroscopic techniques involve placing suture anchors, threading the sutures through the edge of the torn tendon, and then arthroscopically tying knots to secure the repair. Alternatively, a

transfixing implant such as a tack or staple may be used, or a suture anchor with attached suture may be threaded directly through the tendon edge.

Deltoid Repair

In open surgery the deltoid must be securely repaired; indeed, some of the worst complications in cuff surgery involve damage to or detachment of the deltoid. The deltoid may be repaired back to bone, a cuff of soft tissue, or both. Some surgeons detach a portion of the deltoid with a sliver of acromial bone, so that it can be repaired bone to bone. When repairing the deltoid after open cuff repair, it is necessary to use heavy (#2) nonabsorbable suture to the deltotrapezius fascia and through the acromion bone.

The major advantage of arthroscopic repair is believed to be the preservation of the deltoid origin. However, Rockwood and Lyons have suggested that subperiosteal elevation of the deltoid, as is performed with arthroscopic acromioplasty, may detach a large proportion of the Sharpey fibers of the deltoid origin, causing substantial weakening.[189] Indeed, with the advent of mini-open cuff repair, surgeons have been able to examine the deltoid after an arthroscopic acromioplasty, and many have noted (anecdotally) occasional thin areas or even frank detachment of the anterior fibers. It is therefore important when performing an arthroscopic acromioplasty to keep the anterior and superior attachments of the deltoid to the acromion.

Rehabilitation

Passive motion begun early after repair is used by most rotator cuff surgeons, and active motion is generally deferred until tendon healing can be expected. The limits of motion must be based on the surgeon's impressions of the security of the repair and the quality of the tissues. These factors are highly variable. Stiffness is unusual after repair of massive tears, because most of the capsule was off with the tendon and both are often thin and insubstantial. Pulling out of the repair is the more likely complication, so slow, gradual, passive motion is generally the core of the postoperative program. Conversely, patients with small tears, with thick, robust tendon (and attached capsule), not infrequently may become stiff after repair, whereas repair dehiscence is far less frequent than after repair of massive tears.[105] For these patients, a more aggressive mobilization program is used. In any event, the program must be directed by the surgeon who performed the repair, and progress must be closely monitored.

Mini-Open Approach for Full-Thickness Rotator Cuff Tears

Arthroscopic approaches generally involve three bursal portals: anterior, lateral, and posterior. Whereas a decompression and, if indicated, a distal clavicle resection involve visualizing the anteroinferior and anteromedial acromion, the cuff tear and tuberosity are generally quite lateral to the acromion, and the lateral portal must not be placed too high. Mini-open approaches are considered to be those that employ only a split of the deltoid, without any takedown of the origin. The actual skin incision may range from a 3-cm portal extension in the skin creases[182] to a fairly large longitudinal incision.[176]

In an arthroscopic-assisted approach, the initial procedure is the same as with an arthroscopic repair, usually including an arthroscopic subacromial decompression. Then a small incision is used to directly repair the tear through a deltoid split without detachment. This approach is especially useful for small and medium cuff tears. Although some authors have made quite large skin incisions and still called it a "mini-open" approach, if only a deltoid split is used, most surgeons have kept the incision small and in the skin creases. We have preferred a "portal-extension" approach in which the anterolateral portal is extended to a length of 3 cm, anterior to posterior[182,226] (Fig. 3-10). Flaps are elevated, and the deltoid is split in the direction of its fibers to just posterior to the anterolateral corner of the acromion. The deltoid split should be no longer than 5 cm from the acromion and should incorporate the small defect from the arthroscopic portal. None of the deltoid is detached from the acromion. A stay suture is then placed in the deltoid to prevent propagation of the split and possible damage to the axillary nerve. By maneuvering the arm, the entire extent of the tear can be seen. This approach can provide adequate exposure of the supraspinatus and the upper portions of the infraspinatus tendons, but access to the subscapularis and lower portions of the teres minor is difficult.

When a mini-open approach is performed, it is best suited for the small (less than 1 cm) and medium cuff tears (1 to 3cm). In these size tears retraction of the tendon should be limited to the midhumeral head. In many cases use of a mini-open technique for tendon repair can be a useful method of advancing to a full arthroscopic repair. The steps of the arthroscopic-assisted mini-open procedure are as follows: (1) diagnostic arthroscopy of the glenohumeral joint; (2) diagnostic arthroscopy of the subacromial space; (3) arthroscopic bursectomy, removal of soft tissue undersurface of the acromion, and acromioplasty; (4) cuff tendon edged débridement and tendon mobilization; (5) decortication of the greater tuberosity; (6) placement of bone suture anchors; (7) passage of sutures into the tendon; and (8) knot tying. These surgical steps involved can be performed with an arthroscopic technique, and when the procedure becomes more difficult for the skill level of the surgeon, it can be safely and effectively converted to the mini-open technique to complete the procedure.

When this procedure is performed, we generally recommend limiting this technique to isolated supraspinatus tears because the releases for large, retracted tears seem

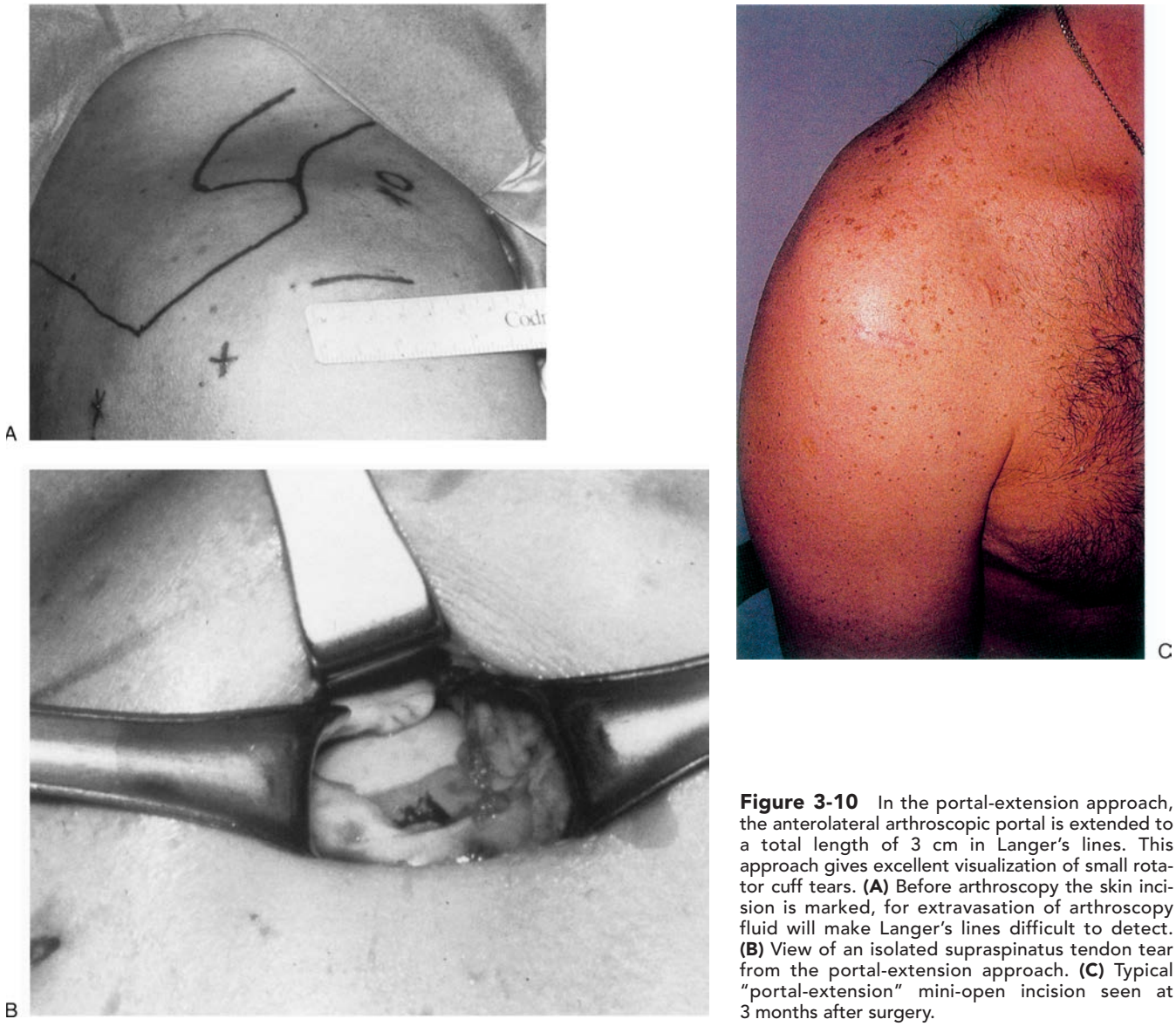


Figure 3-10 In the portal-extension approach, the anterolateral arthroscopic portal is extended to a total length of 3 cm in Langer's lines. This approach gives excellent visualization of small rotator cuff tears. **(A)** Before arthroscopy the skin incision is marked, for extravasation of arthroscopy fluid will make Langer's lines difficult to detect. **(B)** View of an isolated supraspinatus tendon tear from the portal-extension approach. **(C)** Typical "portal-extension" mini-open incision seen at 3 months after surgery.

more reliably and more rapidly performed using a full open or arthroscopic technique. Also, the limited exposure of the mini-open technique does not allow for adequate visualization of the tear configuration for placement of anchors, tendon-to-tendon repair, or tying of the sutures compared to a full open or all arthroscopic technique. With limited exposure excessive retraction of the deltoid may result in increased morbidity, which includes deltoid injury, detachment of the deltoid origin, and increased postoperative pain. Although the mini-open technique is useful for the smaller tears, it is also true that these smaller tears with minimal retraction are easily treated by full arthroscopic techniques. We currently view the value of the mini-open technique as an option for a surgeon to transition from a full open to a full arthroscopic cuff repair.

Technique for Standard Full Open Superior Approach for Posterior Superior Cuff Tears

Many skin incisions can be used to gain access to the rotator cuff, but in our experience the most versatile and cosmetic incision (6 to 10 cm) is one extending from the middle of the acromion anteriorly to approximately 2 cm lateral to the coracoid in the lines of Langer (Fig. 3-11). This incision can be moved medially if access to the AC joint is necessary or laterally if a large tear is anticipated and the AC joint does not need to be addressed. We use this approach for all large and massive tears.

Following the skin incision, subcutaneous flaps are raised. A 3- to 5-cm deltoid split is made from the anterolateral corner of the acromion distally in the direction of the deltoid fibers for tears limited to the supraspinatus

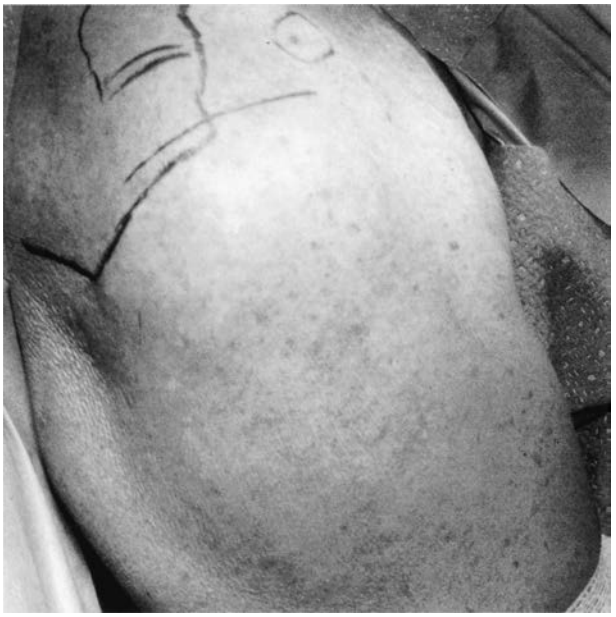


Figure 3-11 Skin incision in Langer's lines at the superoanterior aspect of the shoulder extending from the lateral aspect of the anterior third of the acromion inferiorly to the lateral aspect of the coracoid.

tendon. A stay suture is placed at the end of the split to avoid extension of the split and possible damage to the axillary nerve, which generally lies 5 to 6 cm from the tip of the lateral acromion. For massive posterior tears, the deltoid split is made more posteriorly, starting at the middle of the acromion to allow for greater exposure of the back of the cuff.[16] The deltoid origin is then elevated over the anterior acromion to the anterior aspect of the acromioclavicular joint, traveling 2 to 3 mm posterior to the anterior edge of the acromion. The dissection is then continued around the anterior edge of the acromion underneath the anteroinferior acromion, so that the entire coracoacromial ligament is subperiosteally elevated and stays as one flap with the anterior deltoid (Fig. 3-12). This keeps a thick flap of tissue with the anterior deltoid, allowing secure repair to the acromion with transosseous sutures.

Once the coracoacromial ligament is detached, there is excellent exposure of the anterior acromion and any spurs that may have developed. An acromioplasty is then performed. A 1-in. wide sharp osteotome or an oscillating micropower saw is used to remove the anteroinferior aspect of the acromion from the AC joint to the lateral edge of the acromion. The amount of bone removal depends on the thickness of the acromion, the degree of anteroinferior acromial prominence, and the size of any spurs. The emphasis should be on contouring a smooth undersurface of the acromion. Rongeurs, rasps, and a burr are used to obtain a perfectly smooth surface. The wedge of bone excised should, however, consist of the full width of the acromion from the medial to the lateral border. If the AC joint is tender preoperatively, a distal clavicle excision will be performed. This is performed from the undersurface using either a rongeur or a burr. The superior and posterior AC ligaments are left intact for distal clavicle stability. If the AC joint is not tender preoperatively, then the AC joint is left undisturbed, even if it is arthritic. If it is felt that inferior AC osteophytes are contributing to impingement, then they are removed, and the undersurface of the AC joint is smoothed. Some surgeons have found a high incidence of postoperative AC pain after such undersurface trimmings, and have suggested that perhaps it is better to either leave the joint unviolated or to completely resect the distal clavicle. However, this has not been our experience.

Once the acromioplasty is performed, the bursa is removed for easy rotator cuff visualization. Mobilization of the rotator cuff is the first step in repairing the tendon. Stay sutures are placed in the retracted rotator cuff tendon, beginning anteriorly and working posteriorly, which can be used for traction while mobilizing the tendon. Clamps (which might crush the tissue) are not employed. To mobilize the tendon, all adhesions are freed, beginning on the bursal side, bluntly separating the tendons from the undersurface of the acromion and deltoid. After complete bursal surface release and exposure, the posterior tissues are assessed to determine the full extent of the cuff tear. Usually a portion of the posterior cuff remains attached to the humeral head (Fig. 3-13).

Releases are performed systematically. First the tendon edges are freshened to remove nonsticking bursal tissue and to stimulate healing, and to remove a tapered edge to yield a thick edge that will hold sutures. This generally means removing 1 to 2 mm of tissue; resection of tendon edges to bleeding tissue is not advocated, as, despite a white, nonbleeding appearance, the edges of cuff tears are, in fact, usually well vascularized. The plane between the cuff tendons and the overlying acromion and deltoid is bluntly developed. The retracted tendons are often scarred to the coracoid base, with fixed shortening of the coracohumeral ligament.[160] This ligament is divided (Fig. 3-14), and the coracoid base is freed anteriorly, laterally, and posteriorly, but not medially (to avoid injury to the suprascapular nerve).

As the tendons retract, the capsule may shorten until the tendons are tenodesed to the glenoid rim. To release them, the capsule is divided external to the labrum. Care must be taken to avoid going more than 1 cm medial to the glenoid rim, lest injury to the suprascapular nerve ensue. The use of a self-retaining laminar-type device placed between the humeral head and acromion, to sublux the humeral head inferiorly, can be extremely helpful in performing these releases (Fig. 3-15).

The tendons commonly retract variable amounts. A frequent pattern is that the supraspinatus is retracted medially, whereas the subscapularis is out to length. In such a situation the anterior margin of the supraspinatus, being

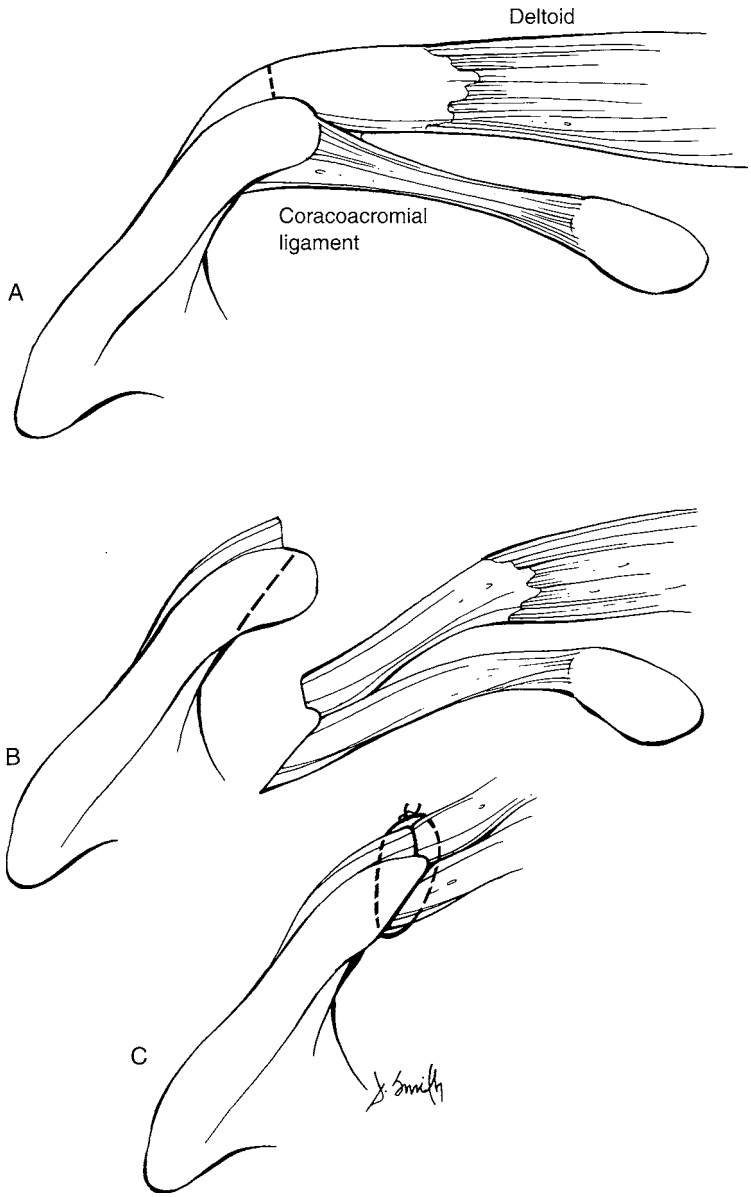


Figure 3-12 **(A)** An incision is made just behind the anterior acromion. **(B)** This is then skived under the acromion to elevate the deltoid and coracoacromial ligament as one flap. The dotted line shows the amount of the acromioplasty. **(C)** The coracoacromial ligament is repaired with transosseous sutures.

medially retracted, is scarred to the medial aspect of the subscapularis. To realign it, an "interval release (slide)"[16] may be helpful. The interval between the supraspinatus and subscapularis is divided to the coracoid base (Fig. 3-16). If there is differential retraction between the supraspinatus and infraspinatus posteriorly, it can also be useful to free the interval between these two tendons (Fig. 3-17). Not only do interval releases allow realignment of retracted tendons, but they also function as "relaxing incisions"[215] so that the tendons, scarred into a circle with the attached capsule at the glenoid rim, may be brought out over the larger-diameter humeral head.

Next the articular surface of the tendon is mobilized. Usually, when the tendon is retracted, the capsule shortens, so that the tendon is essentially tenodesed to the glenoid rim. This may be released by incising the capsule external to the labrum (Fig. 3-18). Care must be taken to avoid injury to the suprascapular nerve at the base of the scapular spine or to the biceps tendon origin. Once the undersurface is freed, the excursions of the tendons are assessed.

If the biceps tendon is intact and gliding in its groove, it is preserved. It is often enlarged and may have minor degrees of fraying. If a significant portion of the biceps tendon is damaged, or if it is subluxed out of its groove (usually in association with a tear or the upper portion of the subscapularis), it is detached from the glenoid and either tenodesed or incorporated into the repair.[31,157]

Intact muscles are generally not rerouted, and tendon transfers for cuff repair are described in Chapter 4. By using the techniques of tissue mobilization described previously,

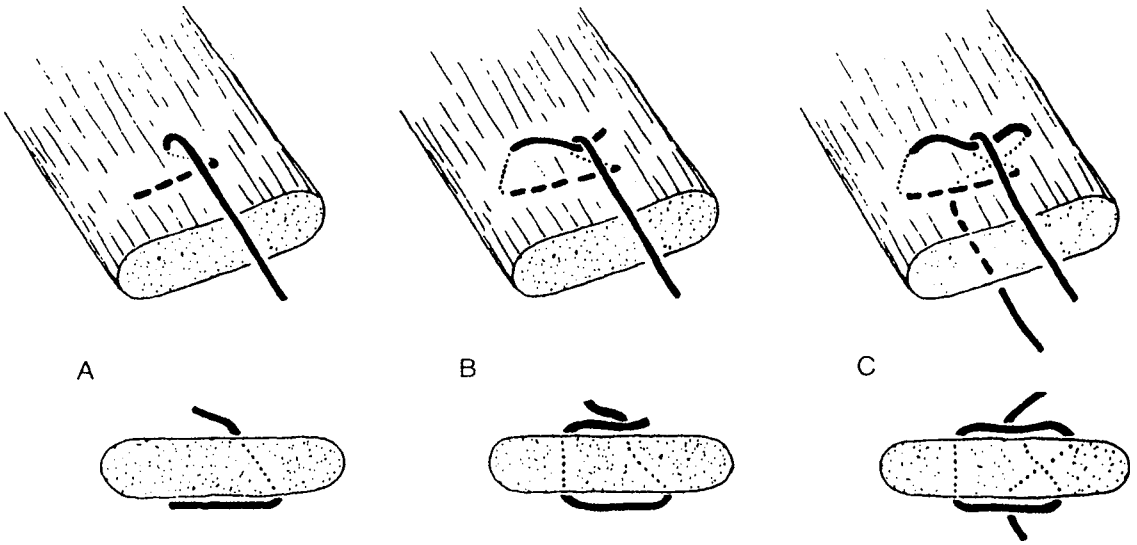


Figure 3-13 Schematic illustration of the Mason-Allen stitch used in rotator cuff repair. **(A)** The stitch is passed obliquely from the superior tendon surface to the inferior tendon surface. **(B)** The suture is then passed directly through the tendon from the inferior surface to the superior surface. **(C)** Finally, the suture is passed in front of the stitch placed in panel A, and brought obliquely through the tendon such that it passes just behind the transverse suture lines present on the inferior tendon surface. This illustration assumes that the suture is now going to be passed through the bone. If the suture has already been passed through a bone tunnel, the steps are inverted (the suture first enters through the inferior surface of the tendon). (From Flatow EL, Weinstein DM, Duralde XA, Compito CA, Pollock RG, Bigliani LU. Coracoacromial ligament preservation in rotator cuff surgery. *J Shoulder Elbow Surg* 1994;3:73, with permission.)

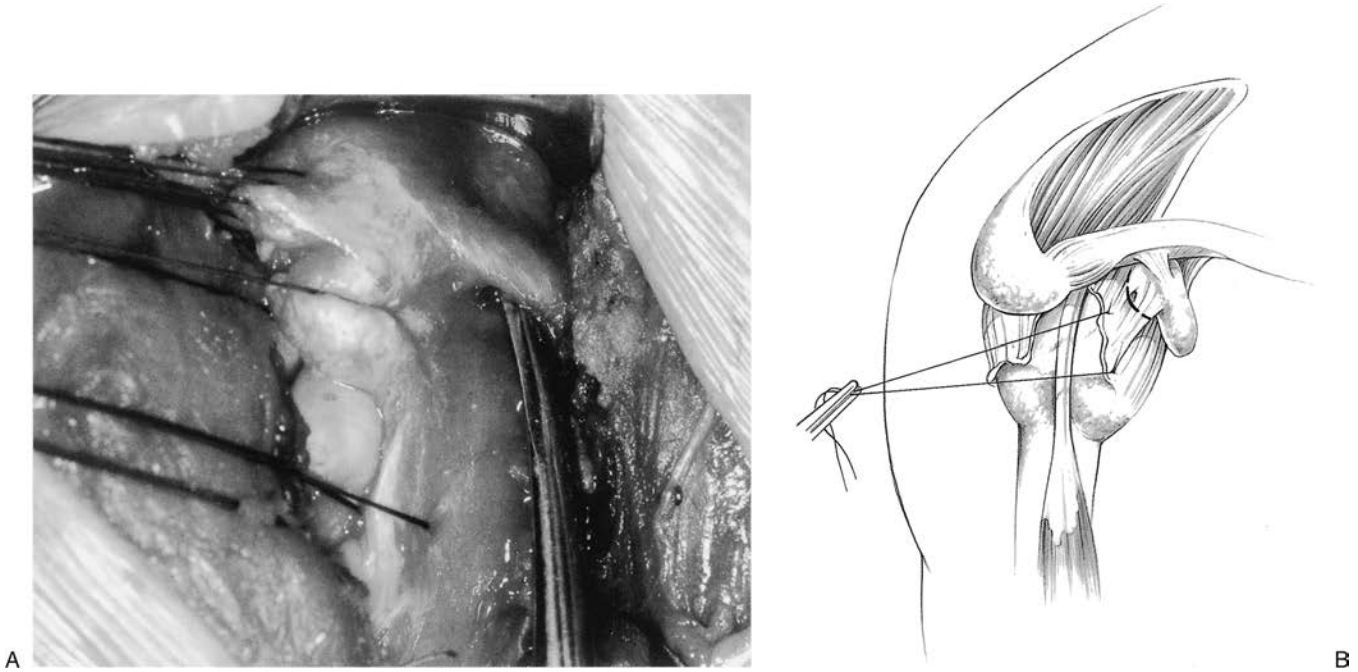


Figure 3-14 **(A)** The coracohumeral ligament (at end of instrument) is contracted, tethering the retracted tear edge to the coracoid. **(B)** This ligament must be divided (*dotted line*) and the coracoid base freed to mobilize the tendon. (From Lazarus MD, Yung S-W, Sidles JA, Harryman DT. Anterosuperior humeral displacement: limitation by the coracoacromial arch. American Academy of Orthopaedic Surgeons, 62nd Annual Meeting, Orlando, Florida, February 1995, with permission.)

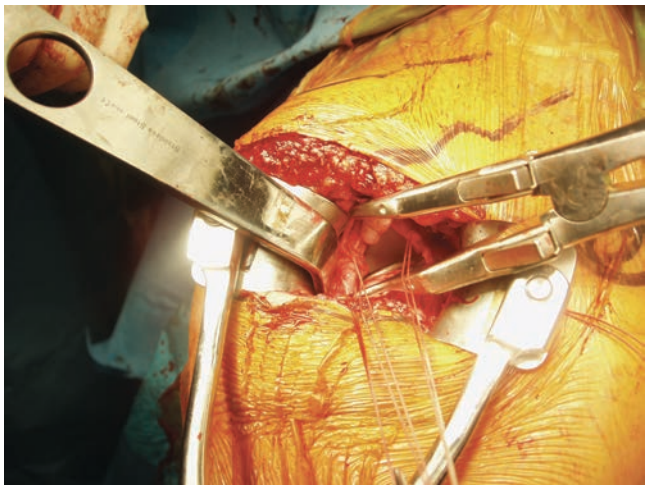


Figure 3-15 Lamina spreader-type device designed by Christian Gerber to subluxate the humeral head from the glenoid to gain access to the superficial and deep portion of the rotator cuff and capsule and to see within the joint.

tendon repair may be achieved in all but a few rare situations. These repairs may sometimes be imperfect, reattaching an atrophied muscle by thin, poor-quality tendon tissue. However, reattachment gives the muscle–tendon units a better chance for functional recovery than leaving them disinserted. If there is significant loss of cuff tissue and full repair is not possible, the anterior and posterior cuff are mobilized cephalad as much as is possible to gain a better fulcrum for head depression.[27]

The greater tuberosity is prepared for tendon repair by removing all soft tissue and smoothing irregular bony prominences with a rongeur or curette. A deep trough is not used, for it requires more tendon mobilization and has not been shown to be necessary to promote tendon-to-bone healing. There are many ways to make a tunnel in bone for passing sutures. Among the techniques that we find useful include the use of a set of reusable curved awls (Link America, Denville, NJ), disposable suture passing set (Linventec), or a custom power tool such as a Curvetec. An inexpensive method is the use of a heavy cutting needle on a heavy needle passer that can be loaded with suture directly and passed through the bone of the greater tuberosity. The tendon is then repaired to the greater tuberosity, with the arm at the side in a neutral position. A combination of simple and Mason-Allen stitches are used at the tendon edge. In all methods the bone holes should remain small and separated by about 1 cm of bone and should be at least 2 cm from the top of the greater tuberosity. Adhering to these principles will minimize the sutures cutting through the bone. When the bone is very weak, use of a bone augmentation device (plate or plastic button) can be used beneath the sutures and the knots tied over the device (Fig. 3-19).

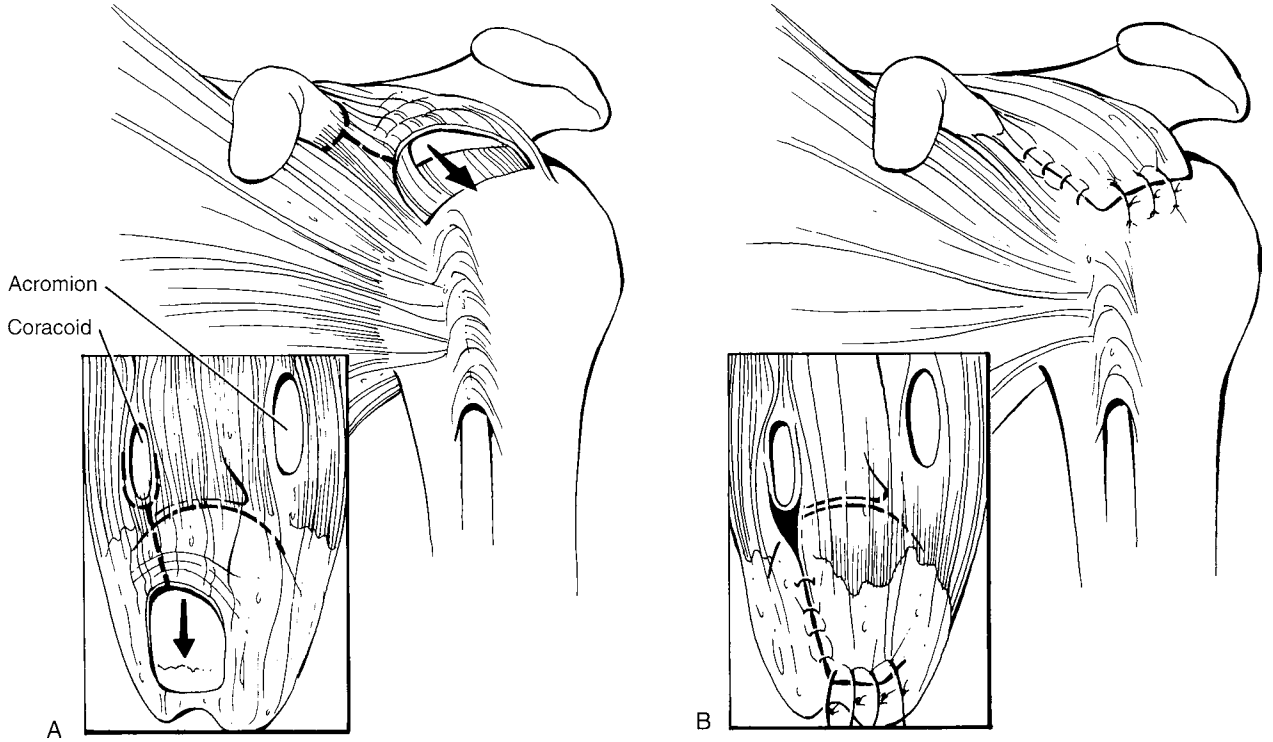


Figure 3-16 **(A)** If there is differential retraction, release of the rotator interval (*dotted line*) can allow realignment of the supraspinatus tendon (*arrow*). **(B)** After the tendon is repaired to the bone, the interval is sutured. (Redrawn from Calvert P, Packer N, Stoker D, et al. Arthrography of the shoulder after operative repair of the torn rotator cuff. *J Bone Joint Surg Br* 1986;68:147–150.)

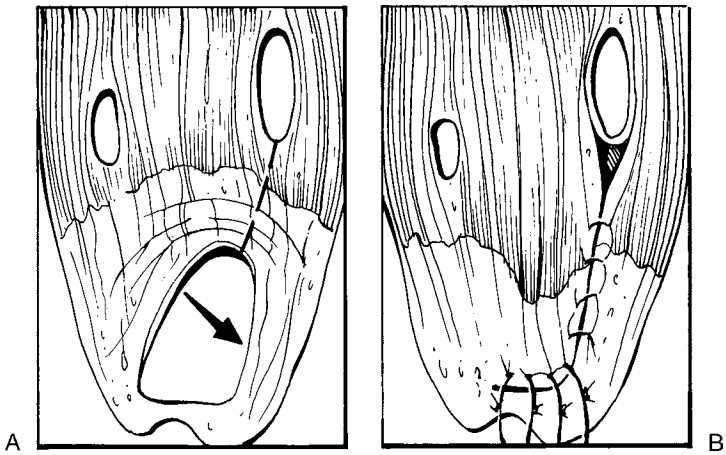


Figure 3-17 **(A)** If the tear is retracted with the greatest differential retraction between the posterior supraspinatus and the infraspinatus, a posterior interval release (*dotted line*) is performed, separating these two tendons so that the supraspinatus tendon may be mobilized (*arrows*). **(B)** After the tendon is repaired to bone the interval is repaired with sutures. (Redrawn from Calvert P, Packer N, Stoker D, et al. Arthrography of the shoulder after operative repair of the torn rotator cuff. *J Bone Joint Surg Br* 1986;68:147–150.)

We still prefer transosseous sutures over metallic suture anchors in open cuff surgery because of the variable strength of the tuberosity bone in these often elderly cuff-disease patients (unlike the uniformly hard bone of the anterior glenoid in instability patients or in younger patients with smaller tears and better bone for arthroscopic cuff repair). Also, the attached sutures tend to hold loose anchors in the superior joint, where articular damage may result (Fig. 3-20). However, technical improvements, including plastic anchors, resorbable anchors, and better fixation techniques, may make implants more attractive in the future.

Following repair of the rotator cuff, the deltoid is repaired, which is as important as the rotator cuff repair itself. A heavy no. 2 nonabsorbable suture is used to reattach the deltoid back to the anterior acromion. An anatomic repair can be achieved by passing one suture through the AC joint capsule and then two sutures through the bone of the acromion. A Mason-Allen suture[92] technique (see Fig. 3-13) is used to hold the deltoid fibers securely. The deltoid split is closed in a side-to-side fashion with simple buried knots. The coracoacromial ligament is repaired together with the anterior deltoid flap to the anterior acromion. This provides a buttress that may provide restraint from superior migration or anterosuperior instability of the humeral head.

Depending on the quality of tissue and repair, postoperative rehabilitation is tailored to the security of the repair and the quality of the tissues. Braces that hold the arm in wide abduction and external rotation are not used. However, occasionally, the posterior cuff is repaired with some tension, and a brace to hold the arm in slight abduction

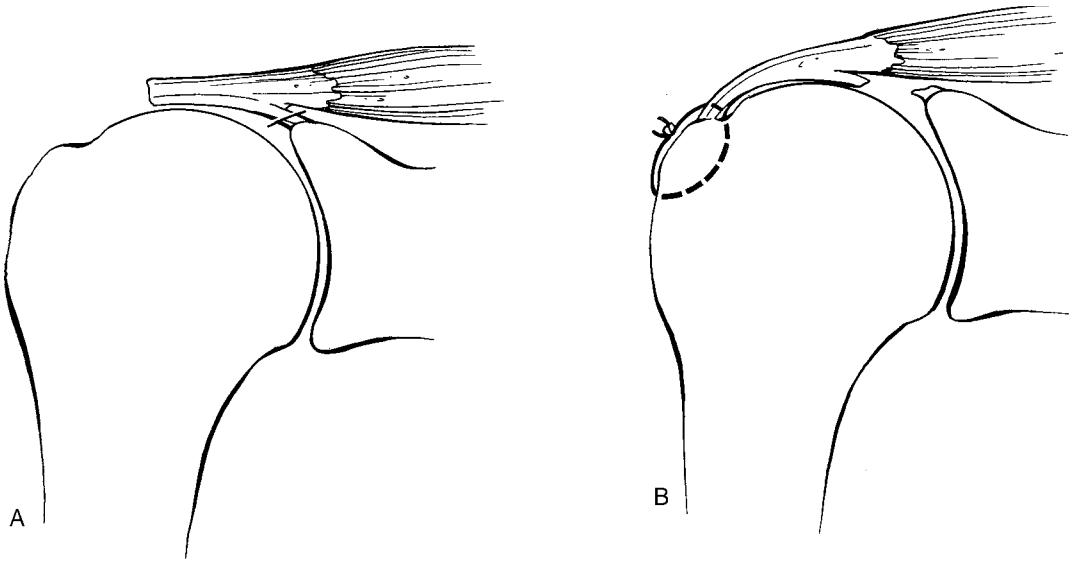


Figure 3-18 **(A)** If the retracted tendon is tethered to the glenoid rim by a shortened, fibrotic capsule, the capsule should be incised (*dotted line*). **(B)** This allows mobilization and repair of the tendon.

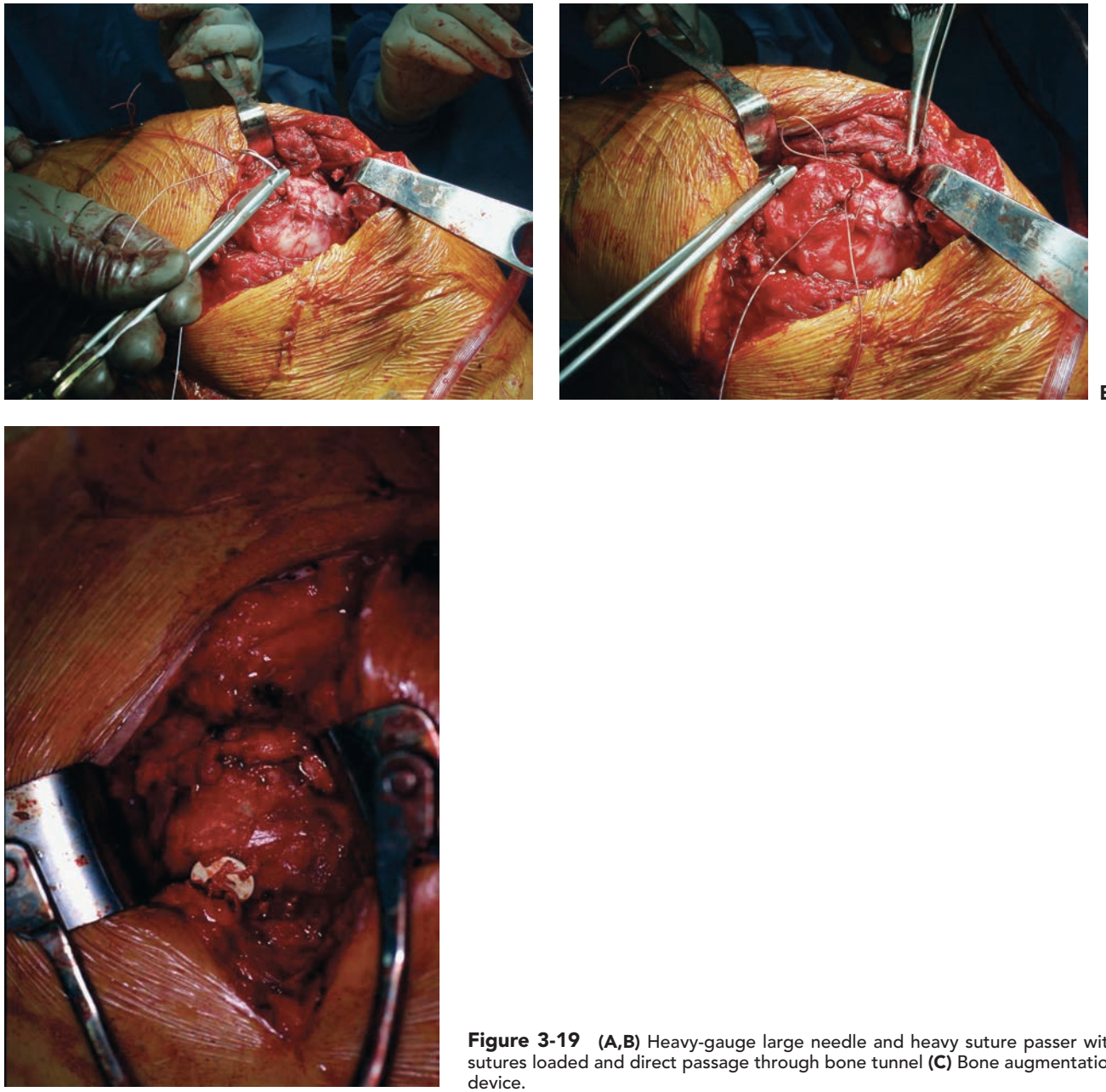


Figure 3-19 **(A,B)** Heavy-gauge large needle and heavy suture passer with sutures loaded and direct passage through bone tunnel **(C)** Bone augmentation device.

and neutral rotation may be used; this avoids the sling position of internal rotation across the body. Passive motion is still begun immediately, with the therapist or a trained family member raising the arm in the scapular plane above the brace.

Postoperative Care for Open Repair

A physician-directed rehabilitation program is usually begun on the first postoperative day. For very massive tears with poor tissue, only pendulum and passive elevation in the scapular plane by the surgeon, the therapist, or a trained family member are used for the first 6 weeks. Pulley elevation is not used owing to the significant active cuff recruitment that results.[139] Elevation is allowed to a level determined at the time of repair, usually between 120 and 140 degrees. Assistive exercises are begun at 6 weeks, but active elevation and light resistive exercises are not allowed until 3 months after repair of these massive tears. Patients after repair of medium and large tears with good tissue begin an assistive program immediately, and active exercises (progressing slowly to light resistive) are added between 6 and 8 weeks postoperatively.

The use of a standard sling versus a small pillow sling versus an abduction brace or large pillow for large posterior-superior two-tendon rotator cuff tears remains controversial. In cases with two-tendon tear involvement

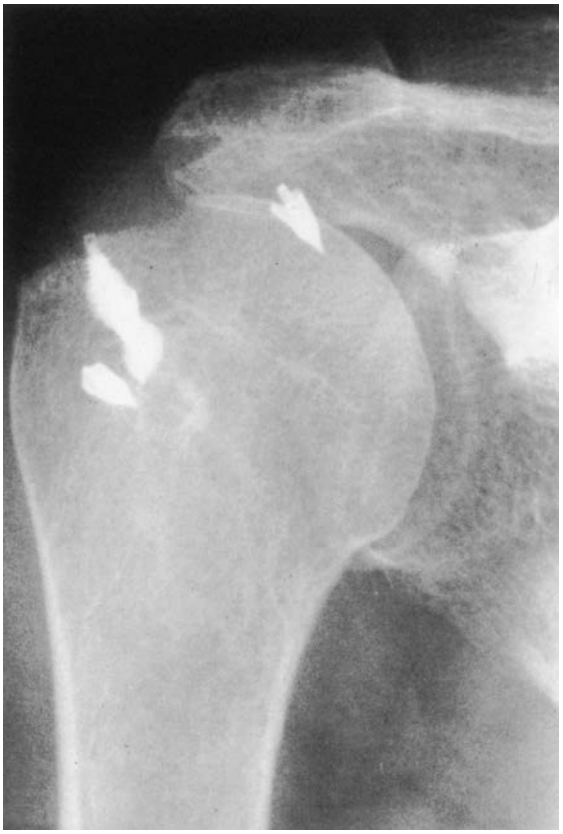


Figure 3-20 This metal anchor has pulled out of the soft bone of the tuberosity. Because it is attached to sutures on the cuff edge, it has stayed adjacent to the articular surface.

(supraspinatus and infraspinatus tendon tears) one of the authors (JPI) will routinely use an abduction brace for the first 4 weeks after surgery. The brace is removed a few times each day for washing, dressing, eating, and doing the exercises noted previously. The patient will otherwise wear the brace during the first 4 weeks after surgery for about 18 to 20 hours each day. Thereafter, the patient is protected in a sling for an additional 2 weeks. Smaller tears treated by either open or arthroscopic means are treated with a sling for 4 weeks after surgery with the same mobilization protocol. The rationale for this different method of postoperative care is based on the increased tissue tension present in large tears with muscle atrophy and fibrous tissue formation within the muscle. This increased tissue tension results in greater tension at the repair site, particularly with the arm by the side. In addition, one of the authors (JPI) believes the use of a brace significantly decreases the patient's overall activity level. With this combination of factors, we believe our results of tendon healing are improved compared to sling immobilization alone.

A study by Reilly et al. showed that supraspinatus tendon tension was reduced by 34 N when the arm was abducted 30 degrees.[186] Tendons repaired in a cadaver that were subjected to 34 N of force over 24 hours gapped an average of 9 mm. Davidson and Rivenburgh showed that the repair tension in 67 rotator cuff repairs correlated with lower Constant scores if it was greater than 8 lb at the time of the repair.[59] Hersche and Gerber measured the tension in a normal supraspinatus tendon with the arm at the side and compared it to the tension in a chronically torn tendon repair with the arm at the side.[115] The normal tendon generated 25 N of force, compared to 59 N of passive tension in the pathologic tendon.

Technique for Anterior Deltopectoral Approach for Isolated Subscapularis and Anterior-Superior Cuff Tears

A deltopectoral approach is considered a preferred technique over a superior approach for open repair for full-thickness tears of a majority of the subscapularis tendon, particularly when the tendon is retracted and the tear is more than 2 months from injury (Fig. 3-21). In these cases the retracted tendon can be more easily and safely found and mobilized. In these cases the axillary nerve should be localized and retracted. After initial exposure there is often a fibrous tissue covering the humeral head and joint, which needs to be excised to expose the joint and find the torn tendon. This tissue can be quite thick and in some cases may seem to be an intact tendon, but when the humerus is rotated this tissue can be seen not to move with the humeral head or lesser tuberosity (Fig. 3-22). With a chronic tear, the retracted tendon is often scarred in a retracted position requiring release of the scarred and contracted coracohumeral ligament and anterior capsule.

After the tendon is mobilized, suture fixation to the decorticated lesser tuberosity is accomplished by suture anchors or with bone tunnels. Mobilization of the tendon

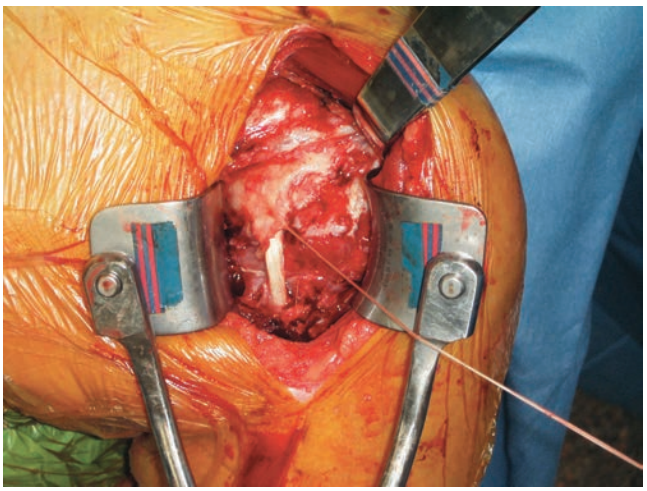


Figure 3-21 Deltopectoral approach for subscapularis tendon tear. This provides excellent exposure and adds a greater margin of safety to the axillary nerve when mobilizing the retracted tendon.

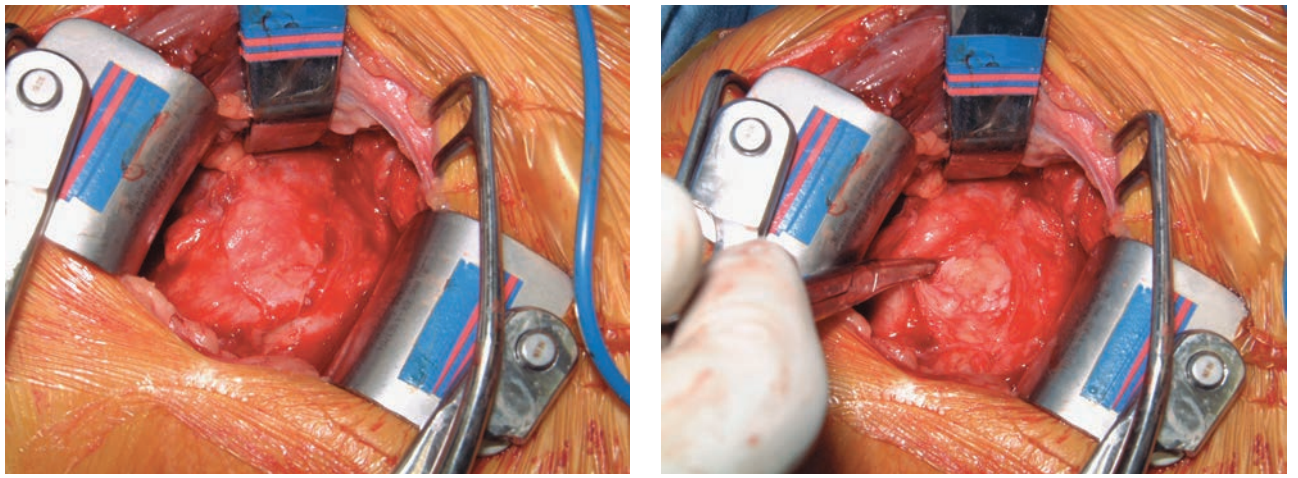


Figure 3-22 **(A)** The thickened bursae over the torn subscapularis can at times make the initial exposure of the tendon seem as if it were intact. **(B)** It is necessary to incise and then excise this tissue to identify and repair the tendon.

should be sufficient to allow the tendon to be repaired to bone with at least 0 degrees of external rotation.

Postoperative rehabilitation should include use of a gunslinger-type brace or small abduction pillow. It is advisable to have the arm in neutral rotation rather than in full internal rotation. Scarring in a full internal rotation (sling) can make rehabilitation to achieve external rotation difficult.

In some patients with massive tears of the subscapularis, supraspinatus, and portions or all of the infraspinatus, there is a need to combine the deltopectoral and superior approaches.

Technique for Arthroscopic Repair of Full-Thickness Tears

Five standard portals are most commonly used (Fig. 3-23) for a full arthroscopic cuff repair. The first is the posterior portal for the evaluation of the glenohumeral joint (Fig. 3-23, portal a). A second posterosuperior portal placed at the posterolateral angle of the acromion and is used for the subacromial space (Fig. 3-23, portal b). Two instrumentation portals are initially localized using a spinal needle working from outside in. One is a portal near the midlateral acromion and another portal is more anterior near the coracoacromial ligament (Fig. 3-23, portal c and d). The fifth approach is anterosuperior at the anterolateral corner of the acromion (Fig. 3-23, portal e).

Diagnostic Arthroscopy

A systematic assessment of the glenohumeral and subacromial space is done both statically and dynamically to assess tissue quality and mobility of the tendon tissue. After a complete visualization, the degree of cuff retraction and mobilization to the insertion site is tested. In many cases the tendon will be under tension when pulled to the greater tuberosity or will not be able to reach the greater tuberosity with the arm adducted to the side of the body. In these cases a periglenoid capsular and subacromial release of bursae, scar, and adhesions is performed so that the tendon can be easily pulled to the tuberosity with the arm by the patient's side. The release is essential to perform the repair without excessive tension. Mobilization is performed from medial to lateral for U-shaped tears (Fig. 3-24A–C) or by reducing with traction on the tendon combined with humeral rotation in L-shaped tears (Fig. 24D,E). Special attention is given

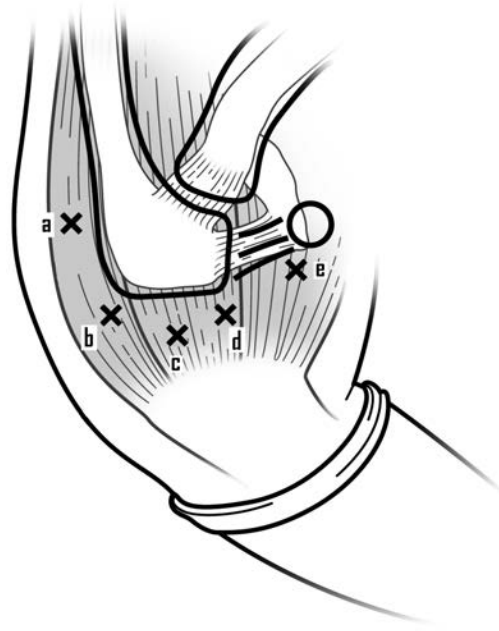


Figure 3-23 Arthroscopic portals for repair of full-thickness rotator cuff tears.

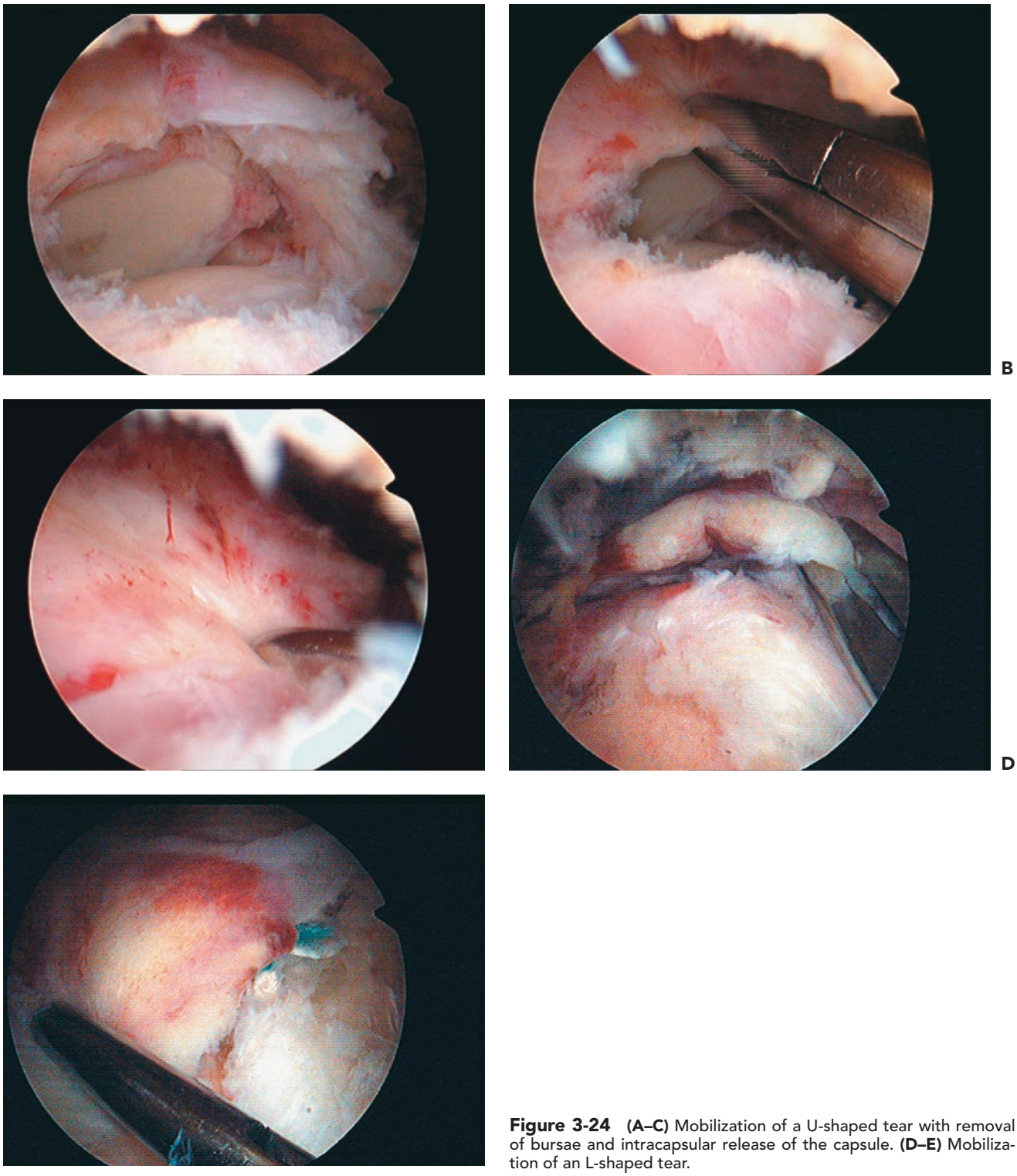


Figure 3-24 **(A–C)** Mobilization of a U-shaped tear with removal of bursae and intracapsular release of the capsule. **(D–E)** Mobilization of an L-shaped tear.

when delamination occurs to repair both deep and superficial leaves of the tear; this is more commonly seen in massive posterior cuff tear.

The stability of the origin of the long head of the biceps tendon is evaluated with a probe. Its stability within the groove is inspected and with internal and external rotation of the arm to check the anterior and posterior pulley. Instability may require repair, tenodesis, or tendon release.

Based on cuff tear size, the ability to mobilize the cuff to the tuberosity and the biceps tendon, and muscle quality, the plan for further treatment is made intraoperatively.

Acromioplasty is systematically performed in addition to the tendon repair, except for massive, partially repairable ruptures where the coracoacromial arch is the last structural element to keep superior stability and avoid anterior-superior escape of the humeral head. If lack of space impedes the exposure or the passage of the instruments, acromioplasty is performed prior to repair of the tendon; otherwise it is done after repair so as to avoid this as a source of bleeding during the repair. The coracoacromial ligament is released with an electrocautery; a burr is used to remove bone form the acromion starting at the anterior lateral corner and proceeding medial to the AC joint to achieve a flat type I acromion (Figs. 3-4 and 3-5).

Mobilization of the Tendon Tear

For large retracted tears the intraarticular capsular and subacromial release is essential to mobilize the tear and decrease the traction on the repair. We used to do it with a bipolar electrocautery device.

For posterosuperior cuff tears, the coracoacromial ligament is released from the base of the coracoid at the anterosuperior edge of the glenoid rim with intraarticular assessment. The capsule is released above the labrum from the anterior-superior to posterior-superior quadrants. Released bursal side scar tissue is removed medially until the cuff muscle is seen.

Special care must be given to the subscapularis release for massive retracted tendons as it usually requires visualization of the axillary nerve and the medial plexus with the axillary artery (Fig. 3-25). This can be a dangerous part of the surgery that should not be done without advanced expertise.

Tendon Fixation

Anchors: The type of fixation depends on the bone quality and influences the technique used for anchor insertion. The use of push-in anchors (vs. thread-in anchors) enables the surgeon to pass a suture through the cuff first and then load the anchor with the suture and place the anchor in bone. When using screw-in anchors or anchors with two preloaded sutures, an anchor-in first technique is required.

Location: Location of the fixation is done according to the lesion, but whenever possible, restoring the normal footprint of the rotator cuff is performed by adding a double row of anchors. One row restores the lateral tendon attachment and the other the medial cartilaginous attachment.

Suture: Suture type #2 or #3 is always a braided nonabsorbable. Absorbable sutures break before tendon healing. Different techniques to pass the braided suture through the tendon using different devices must be available:

- A two-step technique uses a suture passer (Linvatec) equipped with a 45-degree curved hook (left rotated mostly used for a right shoulder and vice versa), loaded with a simple PDS #0 pushed through the hook (Fig. 3-26). The PDS is grasped by a forceps and can be pulled out through the same or a different portal. The PDS is easily used as a shuttle relay for passing a nonabsorbable suture, which is already fixed to an anchor. This avoids any twist or soft tissue interposition when many sutures are used.
- Direct braided suture passing is done in two ways:
  1. An instrument loaded with the suture is grasped by a second retriever (Fig. 3-27); the instrument from a second instrument portal is used to grasp the suture. Alternatively, the same instrument can be used in the same portal by a straight-shaped (Fig. 3-28) or a curve-shaped instrument (Fig. 3-29) once the tendon is perforated. A special shuttle instrument allows for this to be done in one step (Fig. 3-30). The difficulty is in releasing the suture from the instrument without pulling it out of the tendon and grasping it with the same instrument. Lastly, a disposable instrument can pierce the tendon and with a retractable wire can grasp the suture passing this through the tendon (Fig. 3-31).
  2. A suture is retrieved by using a perforating grasper already passed through the tendon. The difficulty with this technique is in anticipating the position of the retriever and preparing to bring the suture to the retriever (Fig. 3-32).

Whatever device or technique is used, the key of the tendon perforation is the access and the direction of the device to perforate the tendon as perpendicular as possible to the direction of the fibers. An in-line device must come from the top or the bottom of the tendon; a curved device should come perpendicular to the tendon (from either the anterior or posterior portal for the supraspinatus tendon),

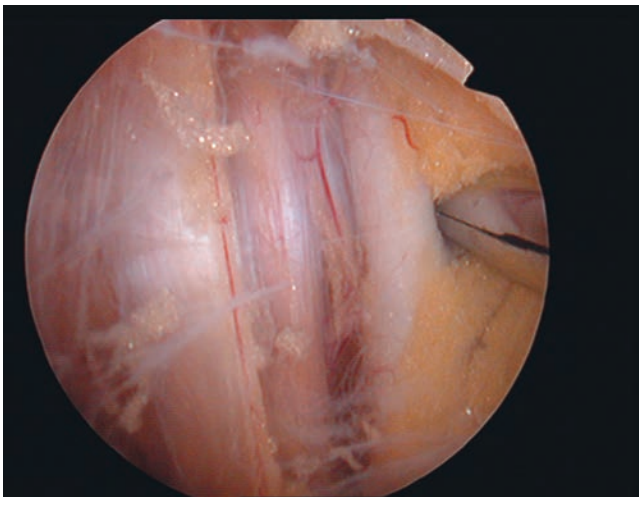


Figure 3-25 Arthroscopic view of the brachial plexus, which often is required for arthroscopic mobilization of a retracted chronic subscapularis tear.

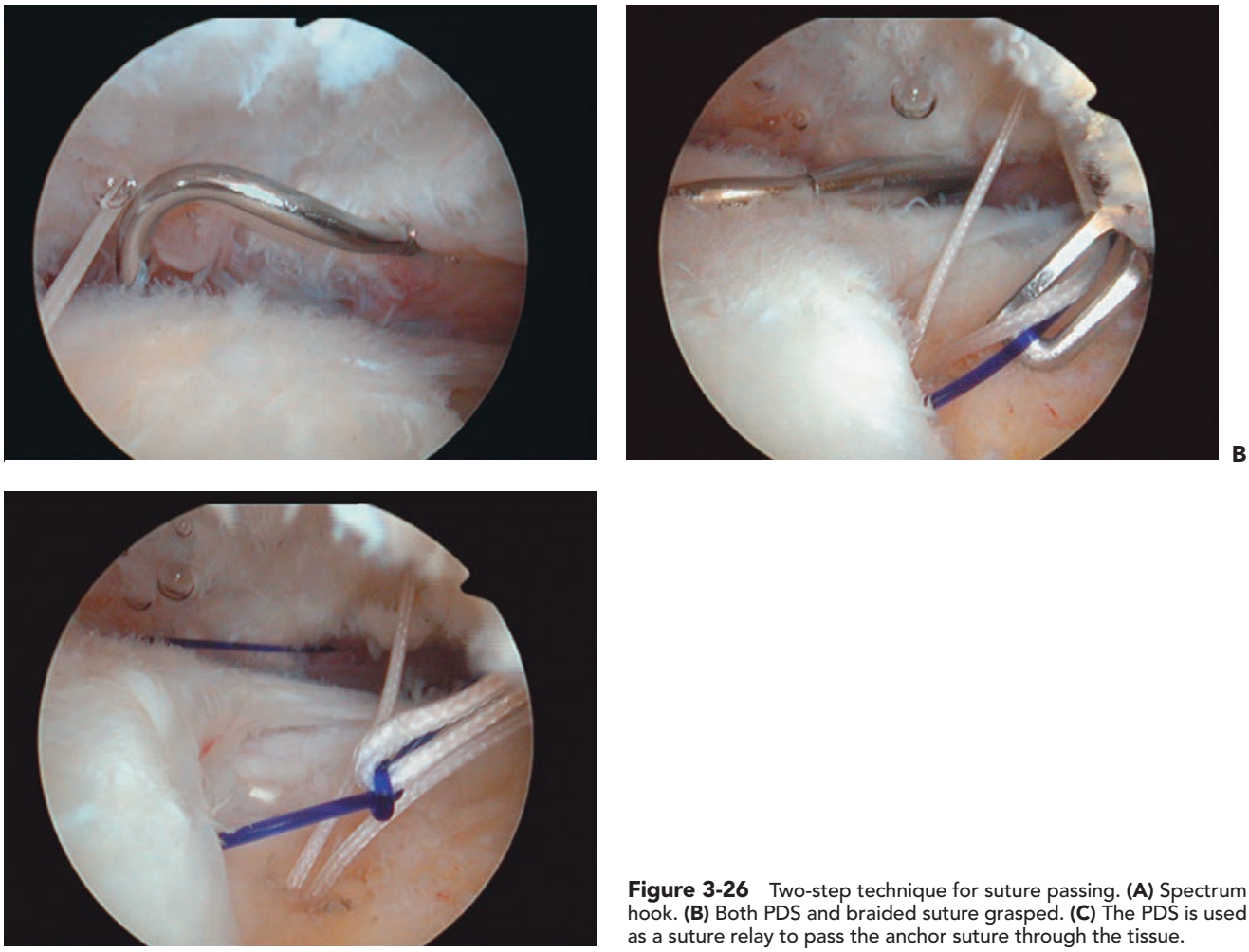


Figure 3-26 Two-step technique for suture passing. **(A)** Spectrum hook. **(B)** Both PDS and braided suture grasped. **(C)** The PDS is used as a suture relay to pass the anchor suture through the tissue.

except for the Express Sew (Arthrex), which has a different shaped needle that can perforate the side of the tendon.

Different Stitches: A few different stitches are possible (Fig. 3-33):

- Simple stitch
- U shape by perforating the cuff from the superficial to the deep layer and back by twisting the perforating hook in one pass of the instrument, to realize a guy rope attachment (see Fig. 3-7)
- A mattress suture by performing successive passage of the suture through the cuff

Side-to-Side Suture: A "lasso" technique was developed by one of us (LL; Fig. 3-34). The aim is to have increasing hold and approximation of the tissue as the suture is pulled and the knots tightened. Once a double braided suture is passed through the two tendons or two edges of the same tendon, then one of the free ends of the same suture is passed through the loop located on the other side. The suture through the loop is tied to the other side of the suture. When the suture is pulled through the loop it gives a perfect method for tightening the suture on the tendons.

Full-Thickness Tears

With a full-thickness tear, reattachment of the tendons establishes an anatomic footprint of the tendon using two rows of suture, one row medially at the cartilage–bone interface and the other row on the lateral aspect of the great tuberosity (Fig. 3-35).

Tears Isolated to the Supraspinatus (Small and Medium-Sized Tears)

After removal of the bursal scar and mobilization of the tendon, the repair starts with medial fixation. The anchor is passed through an accessory small portal (about 2 to 3 mm) situated at the lateral border of the acromion to reach the greater tuberosity as perpendicularly as possible (Fig. 3-36). The repair is done with visualization of the biceps tendon as described for a partial deep cuff tear, or from subacromial if the biceps is torn or fixed to the groove (tenodesis). Bone suture fixation is done with an anchor through the anterosuperior portal. The sutures need to be passed with a mattress

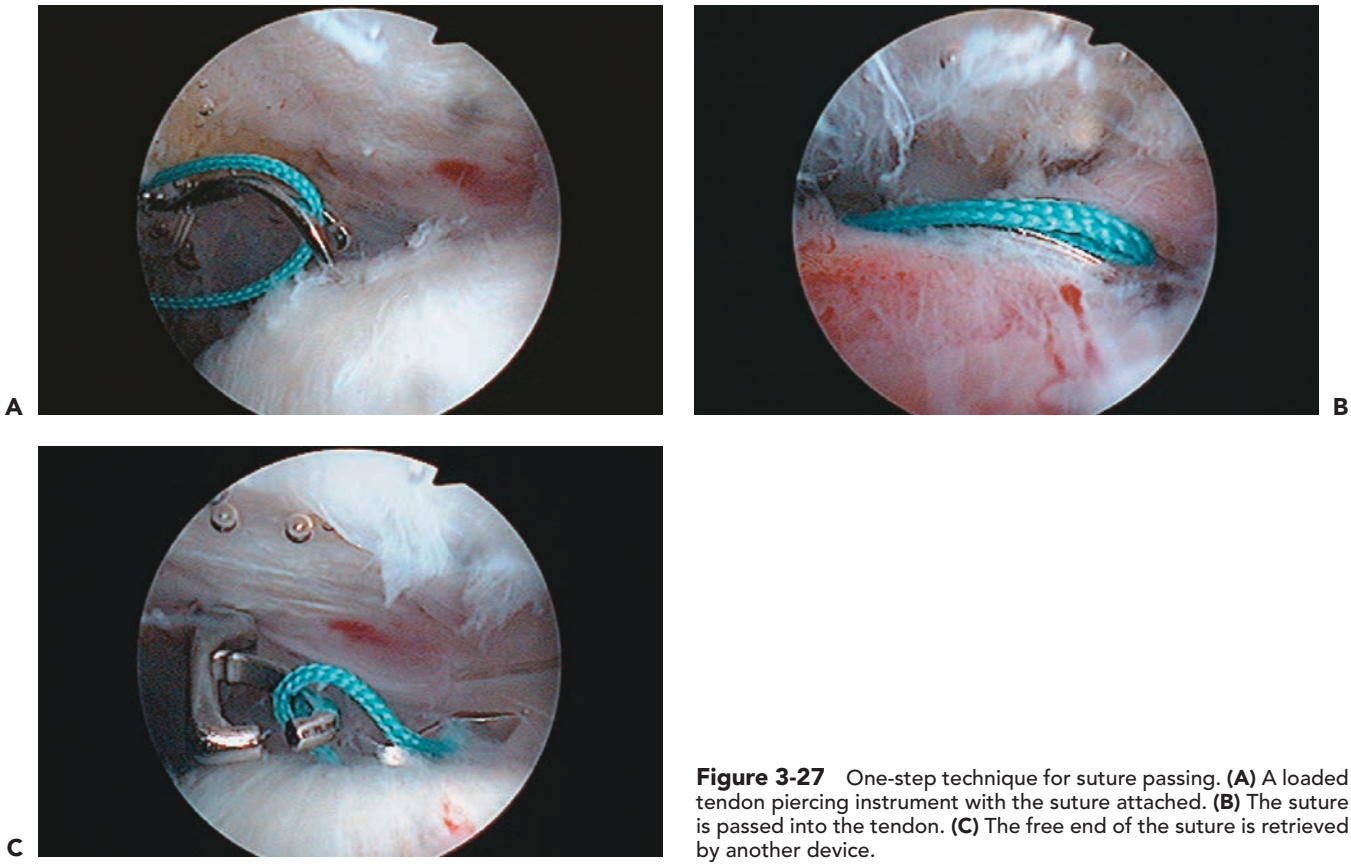


Figure 3-27 One-step technique for suture passing. **(A)** A loaded tendon piercing instrument with the suture attached. **(B)** The suture is passed into the tendon. **(C)** The free end of the suture is retrieved by another device.

technique approximately 15 mm from the edge of the tendon and each limb of the suture approximately 5 mm from one another. There are many ways to pass these sutures, as described previously in this chapter. These sutures are not tied at this point but pulled through an anterior portal to keep them out of the surgical field.

A second anchor is then placed laterally and the sutures passed as a simple stitch or a U stitch for a guy rope technique as described previously. The Spectrum instrument is often used for a U stitch and a grasper may be used for a simple stitch. Knots are tied first with the lateral fixation followed by the medial sutures.

Extension into the Infraspinatus Tendon

Tears that extend into the infraspinatus tendon are often L shaped. These tears require a reverse approach for the tendon-to-bone reinsertion. The arm is placed closer to the side of the body. The visualization is through the anterolateral portal. The tendon is pulled from posterior to anterior and to a lesser degree from medial to lateral. The first anchors are placed at the posterolateral border of the greater tuberosity following the same principles of a supraspinatus repair but using two posterior portals for the instruments. In cases of a laminated tear, the deeper-flap one is fixed first to the medial part of the tuberosity (Fig. 3-37). Before tendon fixation to bone, a tendon-to-tendon suture repair is necessary between the split in the infraspinatus tear to close the "L" portion of the tear. The side-to-side sutures between the supra- and infraspinatus are performed after the tendon to bone to perform an anatomic repair with lateral view and both anterior and posterior portal for instrumentation.

Subscapularis Tendon Tears

Tear of the Upper Third. Subluxation or dislocation of the long head of the biceps tendon (LHB) is often associated with this pathology. When the LHB is in its normal position in the groove, then one needs to be careful neither to create instability by damaging the medial pulley nor to create an impingement between the LHB and the knots tied for subscapularis fixation. The entire operation is performed in the anterior portal and a lateral portal, or alternatively located at the rotator interval near the LHB. The scope is placed near the bicipital groove and is oriented inferior. To expose the subscapularis, the arm must be positioned with more flexion than usual. After abrasion of the lesser tuberosity, an anchor is inserted just in front of the biceps groove and the braided suture is passed through the subscapularis tendon in the same manner as used for the other tendon repairs (Fig. 3-38). The suture retriever may be passed through the canula or the anterosuperior portal

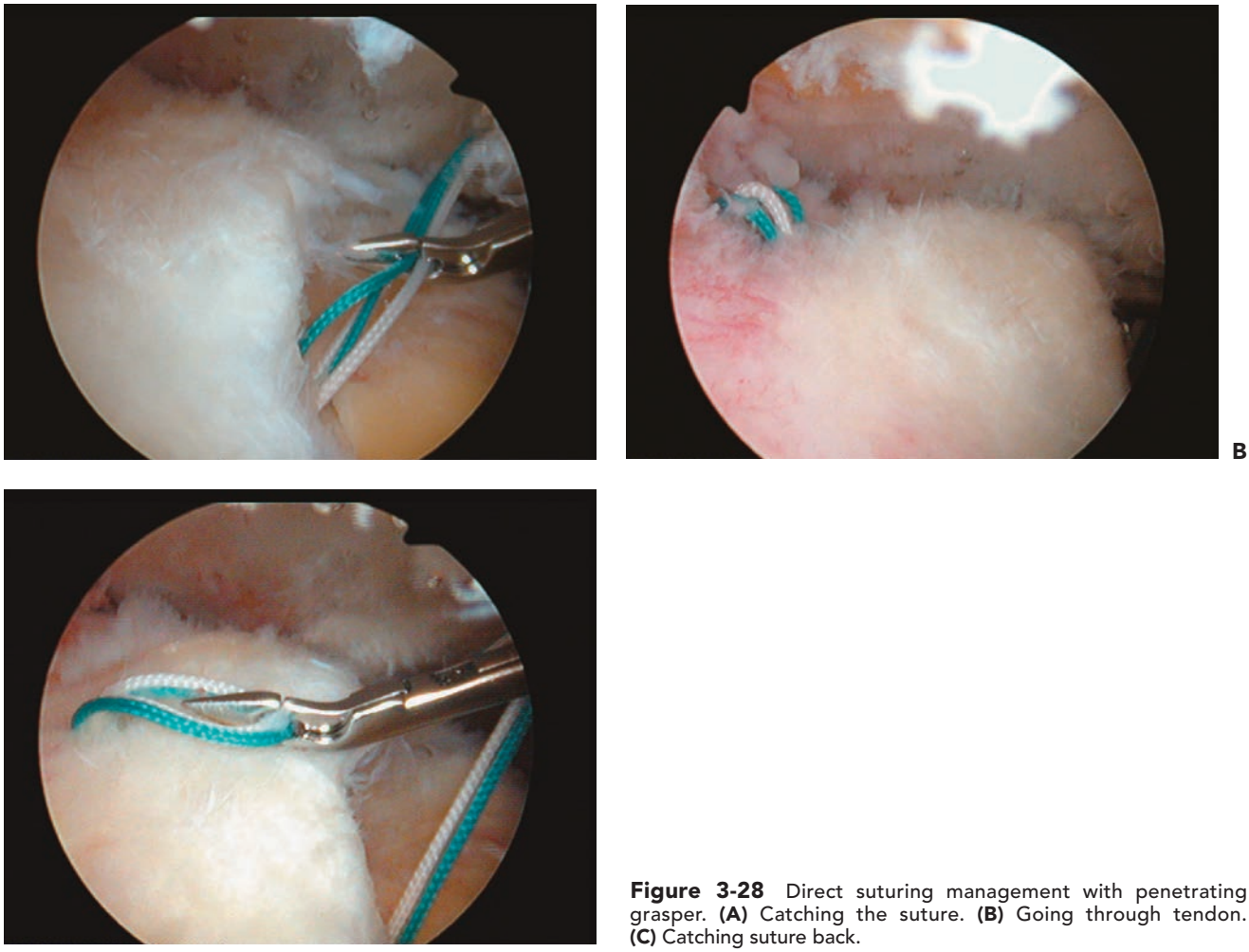


Figure 3-28 Direct suturing management with penetrating grasper. **(A)** Catching the suture. **(B)** Going through tendon. **(C)** Catching suture back.

based on the location of the tendon. A U and simple suture are used on an anchor with a double suture. While pulling at the strands, the knot is tightened and the arm is internally rotating to help reduce the tear.

ROLE OF BIOLOGIC ENHANCEMENT OF ROTATOR CUFF REPAIR

Rotator cuff tears fail to heal in 5% to 90% of cases depending upon the size of the tear, chronicity of the tear, degree of muscle atrophy, amount of fibrofatty degeneration, and method of repair. There is a growing interest in methods to enhance the biologic potential of rotator cuff tendon repair through the use of naturally occurring extracellular matrices (ECMs). Several of the currently available and Food and Drug Administration–approved products are listed in Table 3-1. ECMs are naturally occurring three-dimensional constructs that are harvested from human or animal tissues and are often processed by the manufacturer to remove most of the cellular components. The goal for removal of the cells is to decrease the antigenicity of the material. Cell removal involves different, often patented and proprietary processes that can include saline and mild acidic washes and in some cases the use of enzymes to remove DNA and RNA. The degree and consistency of cell removal varies among the graft materials available for clinical use today. The second major differences in these graft materials is the use of cross-linking agents. Cross-linking of collagen will result in a marked decrease in the ability of the host to resorb the matrix and in these cases the material is a permanent implant.

The purpose of the use of most of the ECM grafts is to apply an absorbable three-dimensional matrix that contains growth factors and an environment that promotes host cell infiltration and subsequent production of neotendon matrices. Over a period of time (6 to 12 weeks), the graft material is absorbed, assuming that the material is not cross-linked. During this time period the graft material loses its mechanical properties, and in most cases it is not sufficient to act as a mechanical tendon substitute of the repair site. Moreover, the decrease in the graft material properties occurs in a time frame that is shorter than the

TABLE 3-1
CURRENTLY AVAILABLE AND FOOD AND DRUG ADMINISTRATION–APPROVED EXTRACELLULAR MATRIX PRODUCTS FOR TENDON REPAIR AND AUGMENTATION

Product Name	Manufacturer	Industrial Source	Tissue Type	Source	Chemically Cross-Linked?
Restore Orthobiologic implant	DePuy Orthopaedics	DePuy Orthopaedics	Small intestine submucosa	Porcine	No
CuffPatch Bioengineered tissue reinforcement	Organogenesis	Arthrotek sports medicine division of Biomet	Small intestine submucosa	Porcine	Yes (carbodiimide)
GraftJacket Regenerative tissue matrix	LifeCell (AlloDerm)	Wright Medical Technology	Dermis	Human	No
TissueMend Soft tissue repair matrix	TEI Biosciences	Stryker Orthopaedics	Dermis (fetal)	Bovine	No
Zimmer Collagen repair patch	Tissue Science Laboratories (Permacol Surgical Implant)	Zimmer	Dermis	Porcine	Yes (diisocyanate)

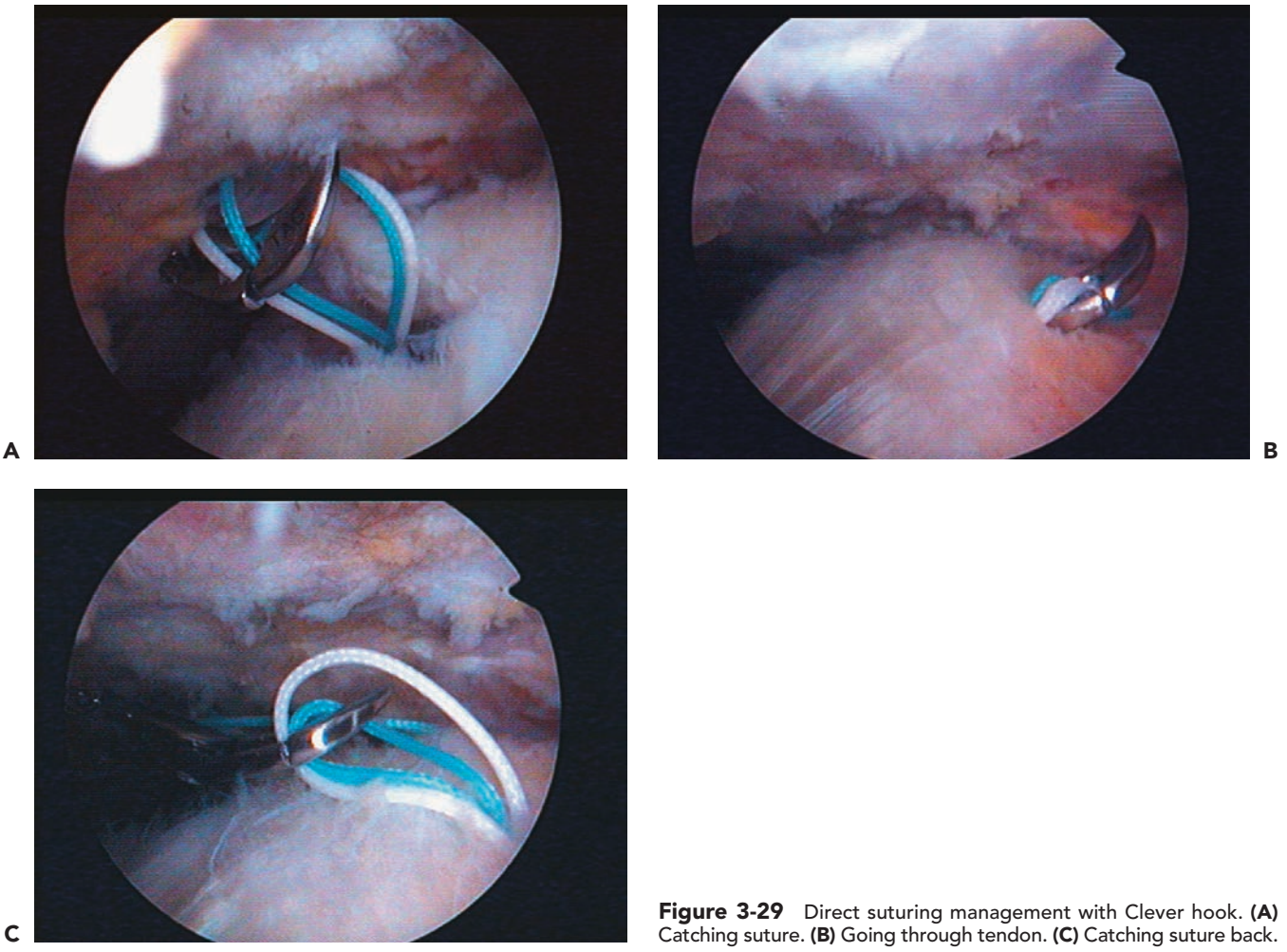


Figure 3-29 Direct suturing management with Clever hook. **(A)** Catching suture. **(B)** Going through tendon. **(C)** Catching suture back.

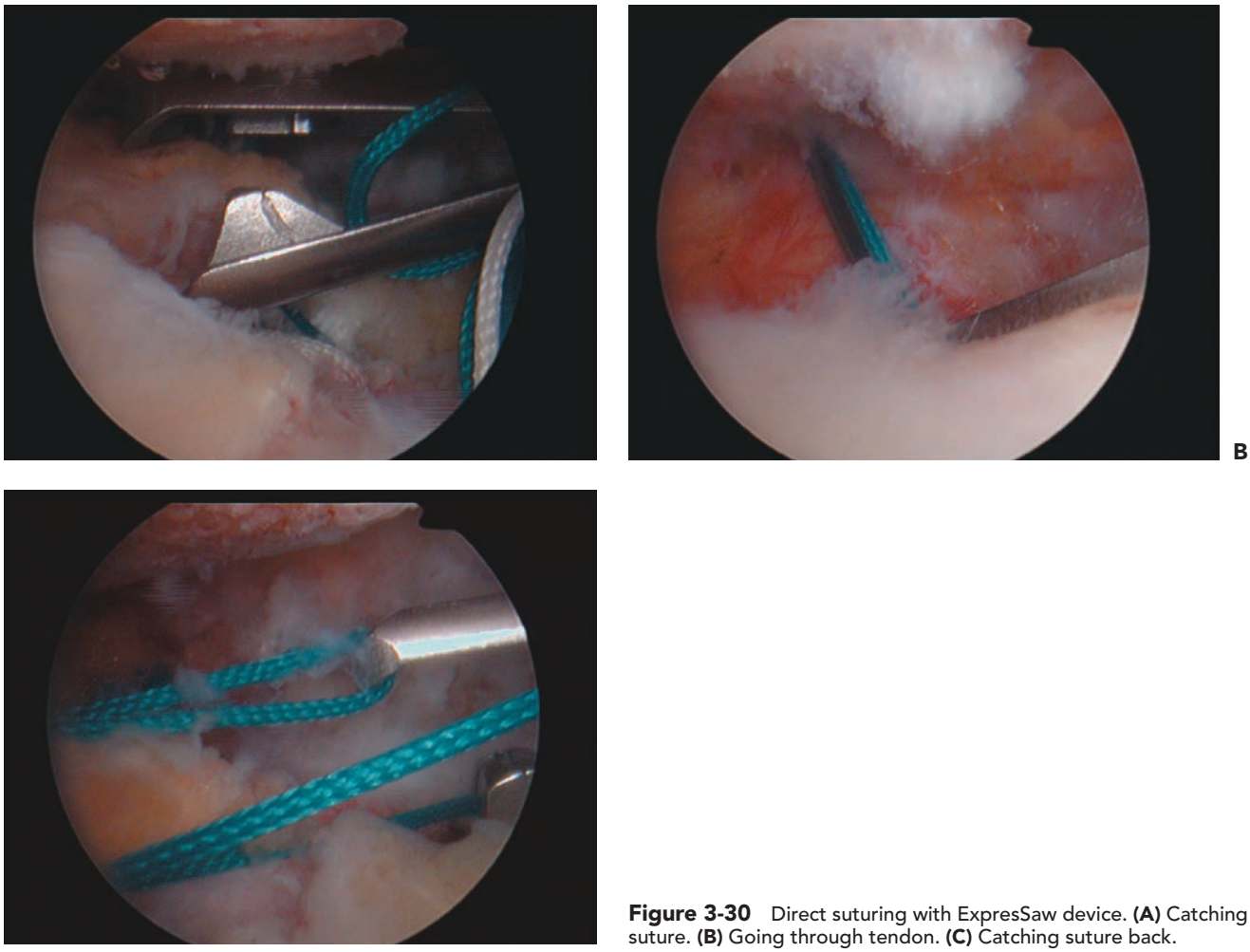


Figure 3-30 Direct suturing with ExpresSaw device. **(A)** Catching suture. **(B)** Going through tendon. **(C)** Catching suture back.

time it takes to produce a strong new tendon host matrix either within the primary tendon-to-bone repair site or within the graft material. For these reasons the absorbable graft materials available today should not be used primarily as a mechanical augmentation device but rather for their biologic properties. The primary repair, quality of the tissues, and postoperative rehabilitation should be mechanically sufficient to allow for optimal healing. Under these circumstances chronic two-tendon tears of the supraspinatus and infraspinatus tendon will fail to heal in 30% of cases when repaired by open surgery, using bone reinforcement, with #2 nonabsorbable sutures and a Mason-Allen suture technique and an abduction brace or pillow for 4 to 6 weeks after surgery.[93] If these ECM grafts are to have a potential biologic benefit in these types of tears, it is likely to be apparent only when the mechanical environment of the repair is sufficient to allow an intact repair in the first 6 weeks after repair.

For smaller tears with less tension at the repair site and better quality of the tissue, the healing rate without biologic enhancement is 85% or greater with open or arthroscopic repair, use of a sling, and early mobilization of the shoulder. In these cases the use of a graft may improve the results of surgery under these surgical and postoperative conditions.

There continues to be rapid progress and advances in this area of tendon repair augmentation and careful prospective randomized clinical trials are needed of the currently available products as well as those that will be developed in the future. All clinicians using these materials are at this point advised to have a thorough understanding of the products' properties and evidence-based data to support their safety and efficacy in each specific clinical application.

Results

The long-term results of rotator cuff repair have been evaluated. Wolfgang[224] in 1974 reported on 65 full-thickness rotator cuff repairs that were first evaluated at 9.8 months and then at 8.2 years postoperatively. In addition to cuff repair, 55% of the shoulders in this series underwent

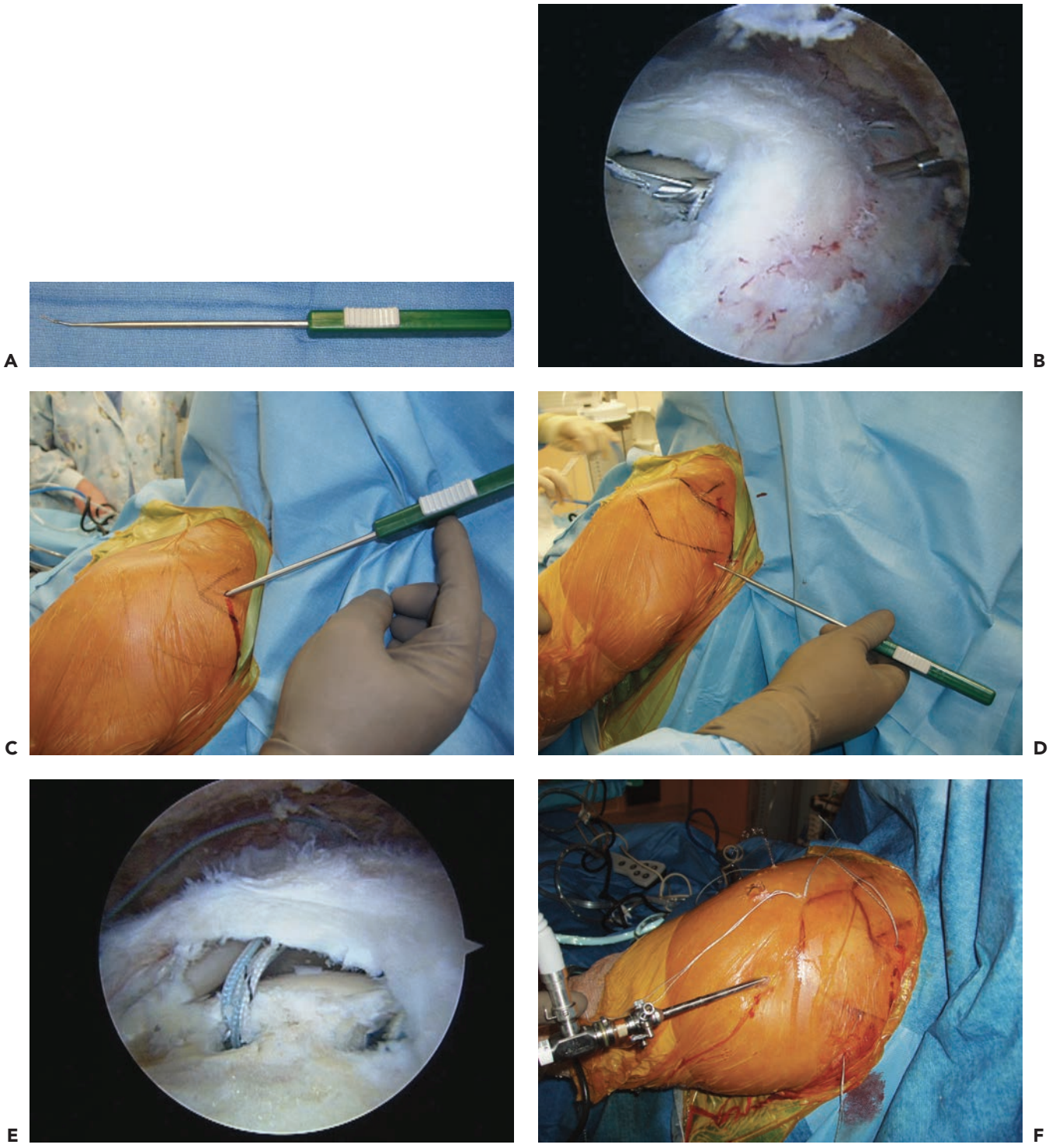


Figure 3-31 **(A,B)** An alternative device has a retractable wire loop that comes from the end of the instrument. The device pierces the tendon and grasps the suture, which is then retrieved from the portal. **(C,D)** Posterior portal for placement of the device. **(E,F)** Sutures in place for the medial row anchors.

lateral acromionectomy. Sixty-nine percent of the shoulders in this series demonstrated good to excellent results at 9.8 months; 46 of these patients were again evaluated at an average of 8.2 years postoperatively, and 74% of these had good to excellent results at that time. Petersson[178] in a different study in 1981 also demonstrated that the results of rotator cuff repair do not deteriorate over time. Of 66 original surgical patients in the study, 43 shoulders were available for reexamination at an average of 14 years after the original procedure. These results were compared with the

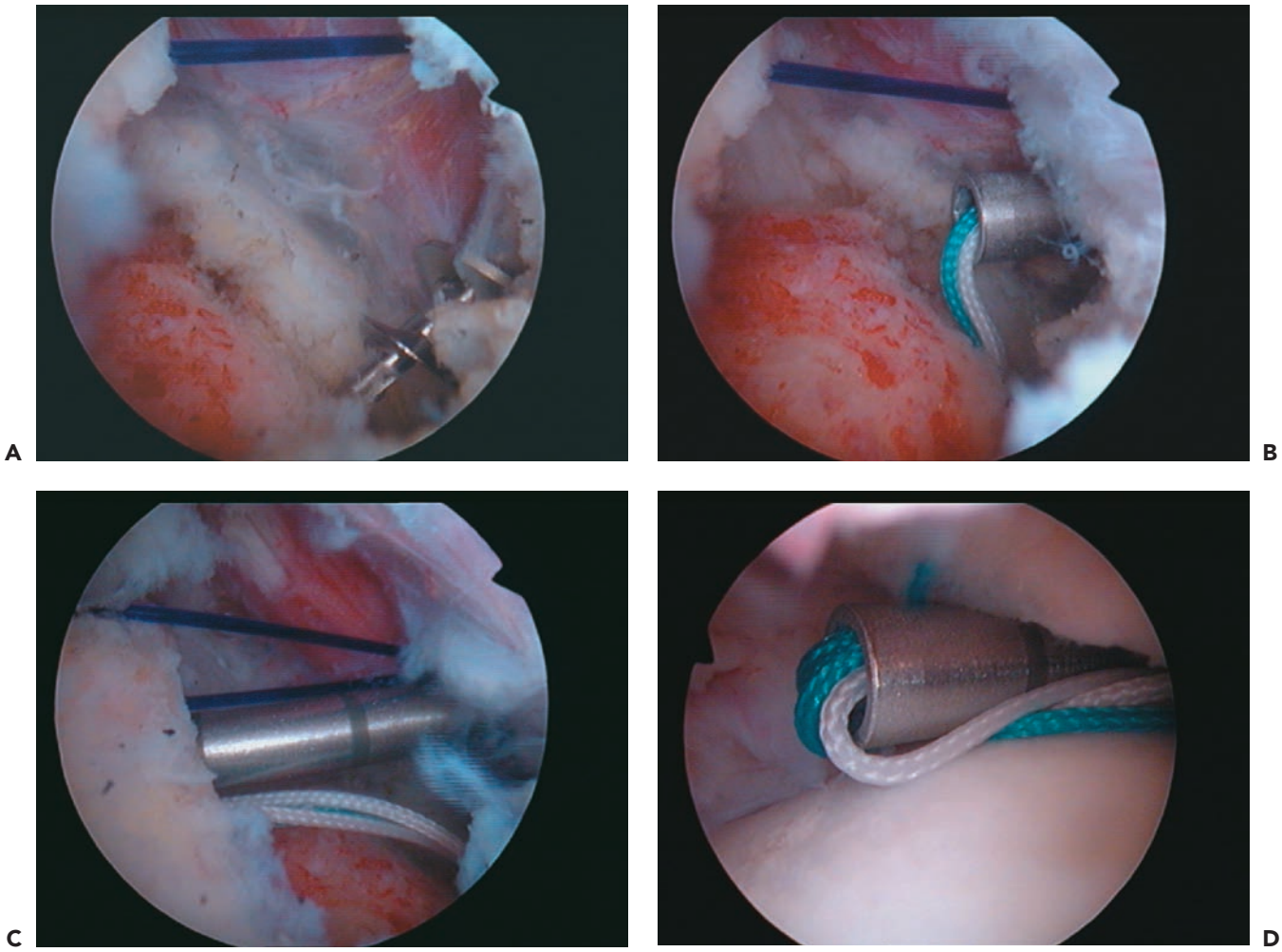
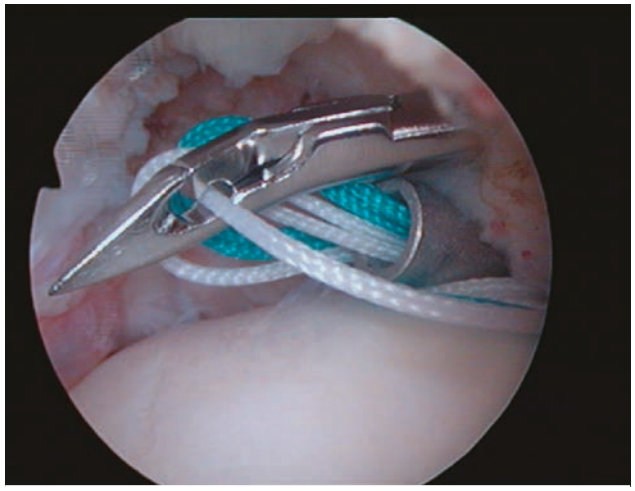


Figure 3-32 Direct suturing by retrieving with Clever hook. **(A)** Lateral anchor from bursal view. **(B)** Two sutures, four post. **(C)**. All punched with the screw handle. **(D)** Intraarticular view. **(E)** Clever hook grasping sutures after penetrating the tendon. **(F)** Both white postgrasped. **(G)** Knot tying.

results obtained in these same patients only 6 months after their original procedures. A good result was judged to be a shoulder with more than 150 degrees of elevation, as well as being devoid of pain. At 6 months postoperatively, 63% of the shoulders were rated as good, and at 14 years postoperatively, 58% were judged to have good results.

Samilson and Binder[195] in 1975 evaluated the results obtained in 33 shoulders with full-thickness tears undergoing surgical repair. The coracoacromial ligament was excised in all cases, and an anterior acromioplasty was performed in 21% of the shoulders in addition to the tendon repair. After average follow-up of 23 months, 84% of these shoulders were rated as having good or excellent results; 76% of the patients returned to work and 70% of the shoulders demonstrated increased strength in external rotation postoperatively.

By 1985, the techniques of rotator cuff repair became more standardized for surgeon acceptance of the importance of physical therapy, anterior acromioplasty, and the preservation of the deltoid muscle origin. Hawkins and coworkers[110] reviewed 100 consecutive rotator cuff repairs at a mean of 4.2 years postoperatively. Repair of the tendon and anterior acromioplasty were employed in all surgical procedures. Postoperatively, 86% of these 100 patients had no or slight pain. The average active abduction postoperatively was 125 degrees (compared to 81 degrees preoperatively). Of the patients in Hawkins' series, 78% could use their affected arm above the shoulder level either normally or with minimal compromise postoperatively, compared with 16% of patients being able to do this preoperatively. Postoperative strength in external rotation of the shoulder was considered to be normal in 42%, compared with 22% of shoulders preoperatively. All but six of the patients (94%) considered themselves to be improved after rotator cuff surgery. Although a statistically significant improvement in strength of the shoulder was demonstrated



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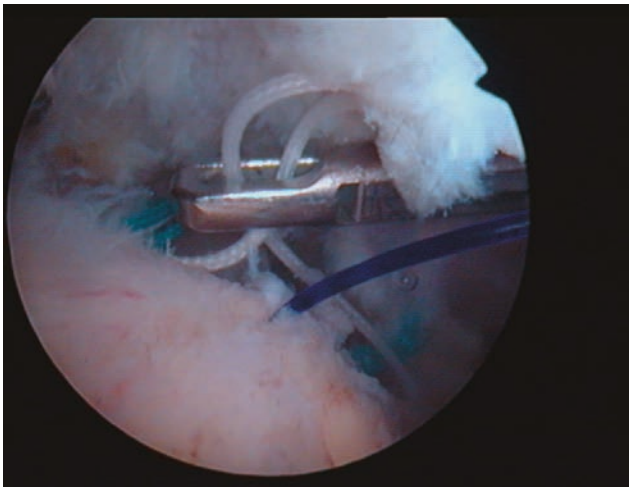

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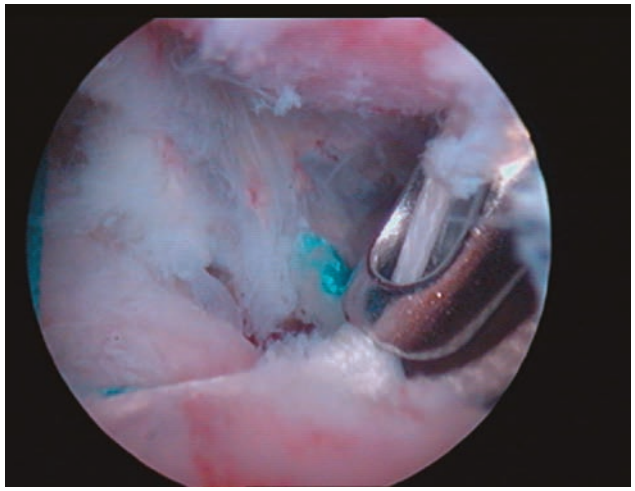

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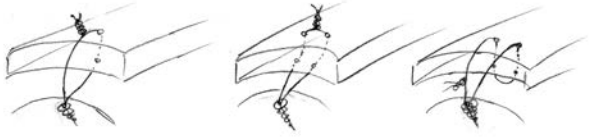

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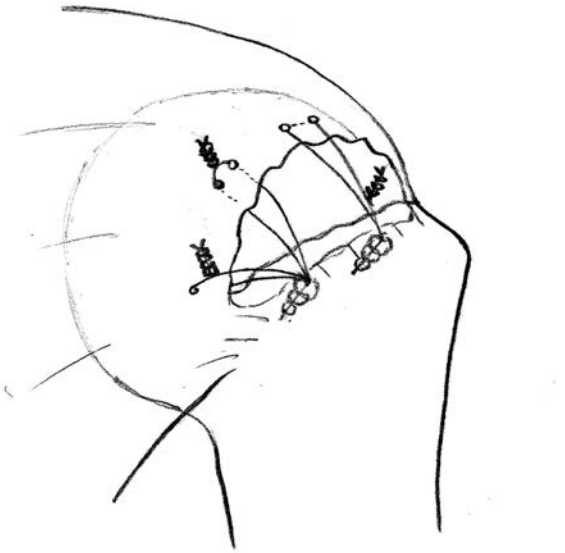


B

Figure 3-33 Different types of common stitch configurations.

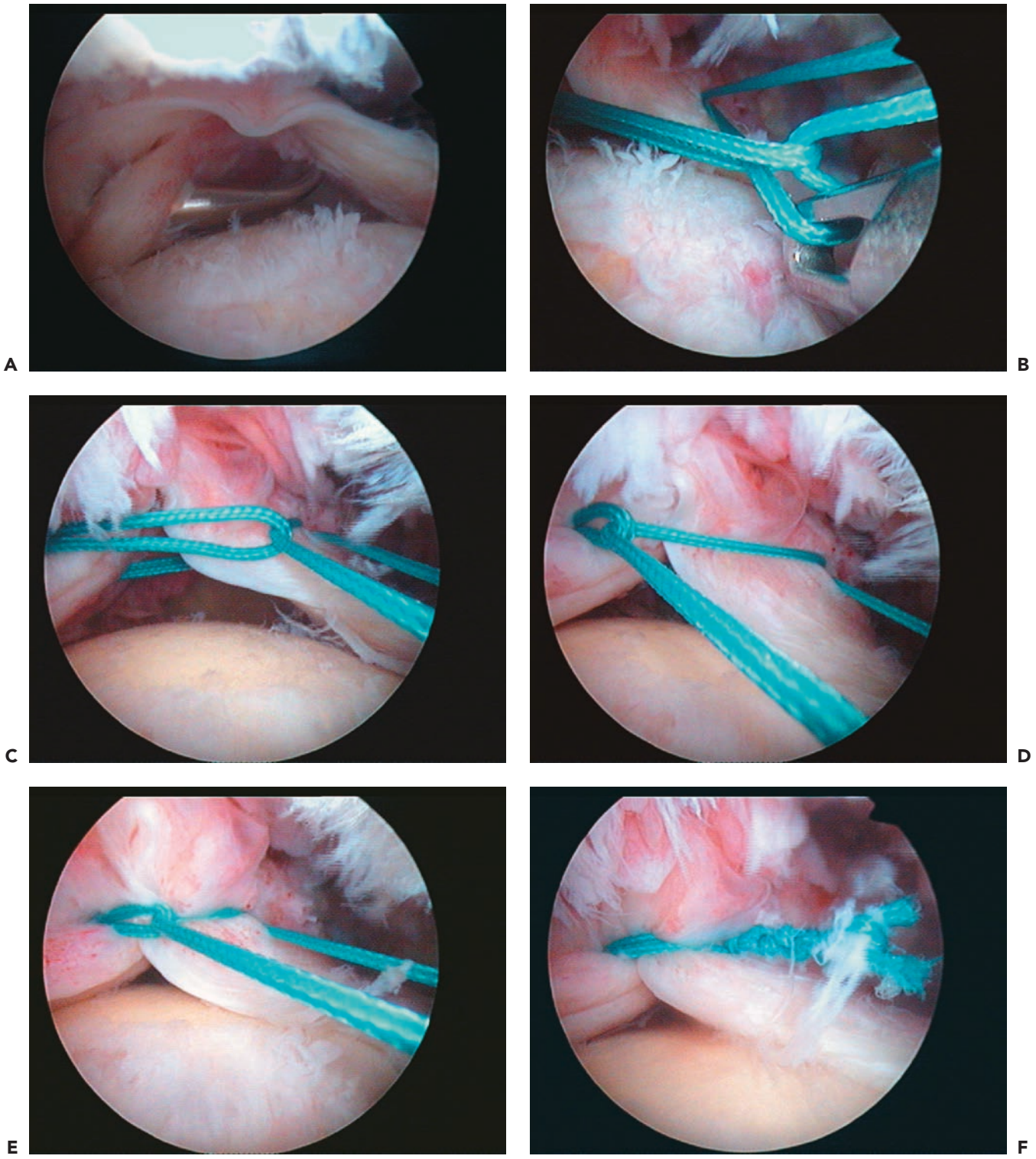


Figure 3-34 **(A)** Lasso suture technique for side-to-side tendon repair. **(B)** The looped end of a single strand of suture is passed through the tendon. **(C)** The same end of the suture is passed through the loop. **(D)** The two ends of the suture are tied to one another. **(E,F)** As the suture is tied, the sides of the tendon tissue are pulled together.

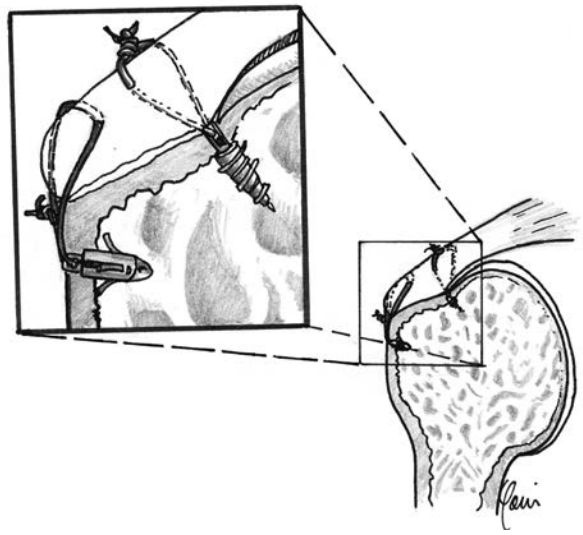


Figure 3-35 Double row suture fixation to create a broad footprint of the tendon from medial to lateral on the greater tuberosity.

postoperatively, the authors of this study were of the opinion that the improvement in function was primarily related to relief of pain.

Ellman and coworkers[69] reviewed 50 rotator cuff repairs after an average follow-up duration of 3.5 years. A full-thickness tear was present in 49 of the shoulders (98%). Anterior acromioplasty was performed in 48 of the shoulders (96%), and in 20 of these same 48 shoulders, distal clavicle excision was also performed. These authors rated their results according to the criteria of Neer.[154] They obtained a satisfactory result in 84% of the shoulders when pain, function, and strength of forward flexion were evaluated. Forty-nine of the patients (98%) were satisfied with their result.

A large series of rotator cuff repairs was reviewed by Neer and coworkers[157] in 1988. These authors analyzed 245 shoulders with tears of the cuff necessitating repair, and 243 of these (99%) also underwent anterior acromioplasty. Follow-up averaged 5.5 years and results were graded as "excellent" (essentially normal shoulder), "satisfactory" (no significant pain, active elevation above horizontal, and patient pleased with result) or "unsatisfactory." Excellent or satisfactory results were obtained in 92% and excellent results were obtained in 78% of these shoulders.

When using these principles for rotator cuff repair, Cofield, in a review of the literature, found an average of 85% of patients had satisfactory results, with reports up to 100%.[51] The Shoulder Service at New York Orthopaedic Hospital for rotator cuff repairs in 486 patients found 96% had satisfactory pain relief and 80% had substantial functional improvement.[179]

Recently, Cofield and coworkers[53] examined 81 shoulders following rotator cuff repair at an average of 7.5 years following surgery. Anterior acromioplasty and repair of the tendon defect were performed on all of the shoulders at surgery. Ninety-three percent obtained satisfactory pain relief and 94% of the patients were of the opinion that they were much better after surgery; 83% returned to work. Overall, 65% had excellent results and 79% had either excellent or satisfactory results.

Cofield[51] reviewed many different series of rotator cuff repairs and averaged the results as described by the various authors. Overall, pain relief occurred in 87% of shoulders, and patient satisfaction averaged 77%.

Factors appearing to influence the outcome of rotator cuff repair appear to be the size of the tear, patient age, and preoperative function. Hattrup evaluated patient age relative to outcome following rotator cuff repair.[106] He was able to show that patients older than the age of 65 years tended to have poorer results as well as larger cuff tears. Cofield and coworkers found that tear size is the single most important factor influencing long-term results.[53] Pollock and coworkers[179] also found that cuff tear size directly correlated with final outcome. Satisfactory results were obtained in 95% of small, 94% of medium, 88% of large, and 84% of massive tears. Others have also found that results for surgical repair of massive tears are inferior to those for smaller tears.[17,53,56,61,97,101,104,131,156,163,173] Harryman and coworkers suggested a reason why larger tears did less well: The repair was less likely to remain intact.[104] Increased preoperative tear size, poorer tissue quality, increased difficulty of tendon mobilization, and the presence of a rupture of the tendon of the long head of the biceps, together, adversely affected outcome.

Numerous studies have found other factors that affect the outcomes of rotator cuff surgery. A study of 30 diabetic patients with rotator cuff repairs revealed a 10% rate of infection and 7% rate of failure compared to a group of matched controls, which had only one failure and no infections.[35] Another study evaluated the effects of smoking in a group of 95 patients compared to a control group of 125 nonsmokers.[137] The mean postoperative University of California, Los Angeles (UCLA) score for the smokers was 25 and for the nonsmokers was 32. Tear size and worker's compensation status were not found to correlate with outcomes in the study. Age is another variable that may affect the outcome of rotator cuff repair, but this has not been consistently found in many case series. The number of excellent and good results for patients older than 62 ranges from 44% to 87% in three cases series designed specifically to evaluate the effects of age on rotator cuff repair outcomes.[100,129,225]

Concomitant acromioplasty has been the standard of care for many years, until recent reports showed data that question the effectiveness of this treatment for patients undergoing rotator cuff repair. Goldberg et al. reported their results of open rotator cuff repair in a series of 27 patients with small and large tears.[95] None of the patients had concomitant acromioplasty. The Simple Shoulder Test (SST)

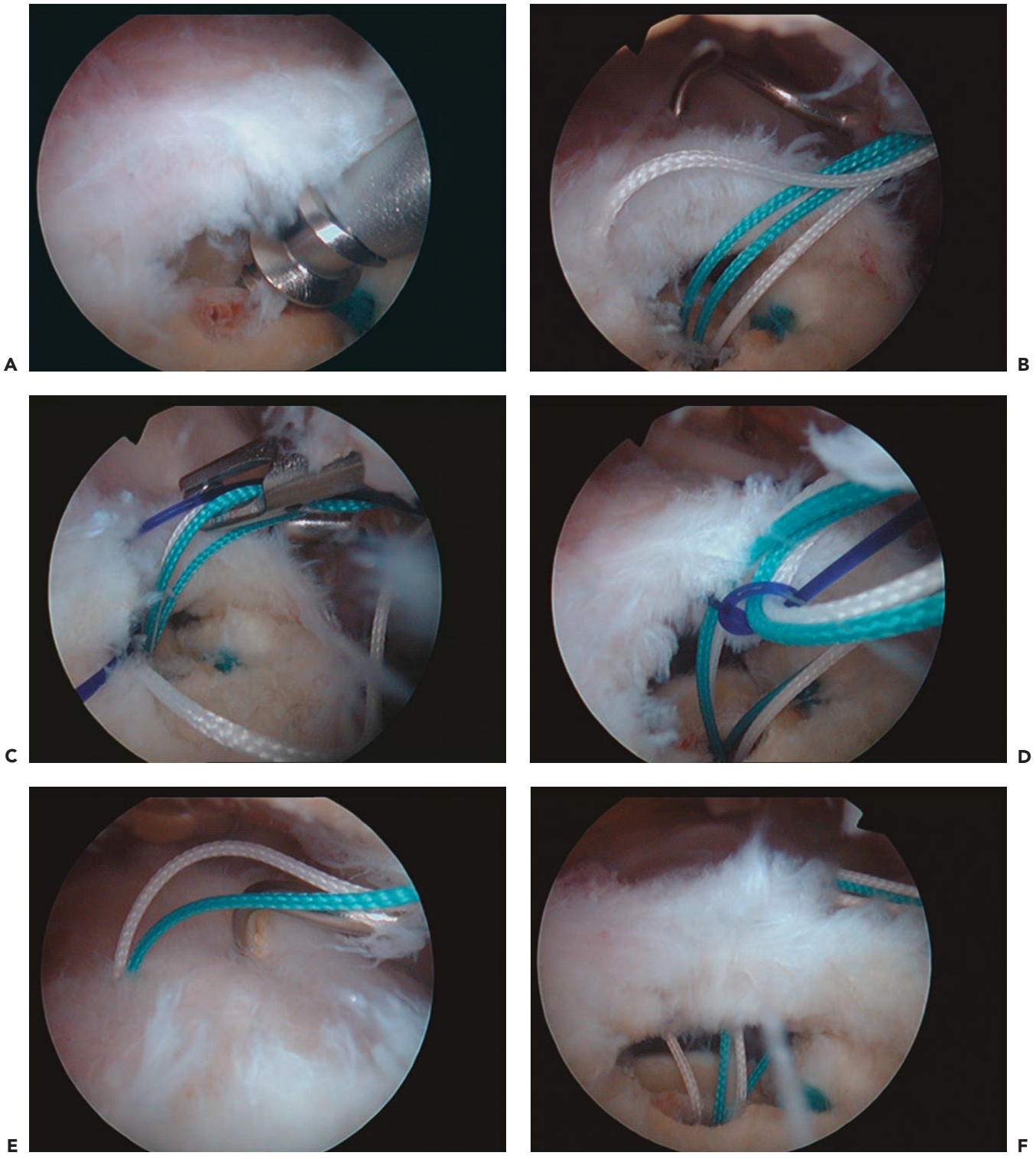


Figure 3-36 Supraspinatus tear. **(A)** First the anchor is placed medially at the articular margin. **(B–F)** The two-step suture-passing technique using the spectrum instrument to pass a PDS 15 mm from the tendon edge **(C).** The PDS is used as a subtle relay to pass two (one of each suture) **(D)** and then the spectrum is again used to pass the other strands of each suture **(E,F). (G,H)** A lateral anchor is placed and the sutures passed as simple sutures and tied. **(I,J)** The medial sutures are tied and the cuff repair is inspected from the bursal and articular surfaces.

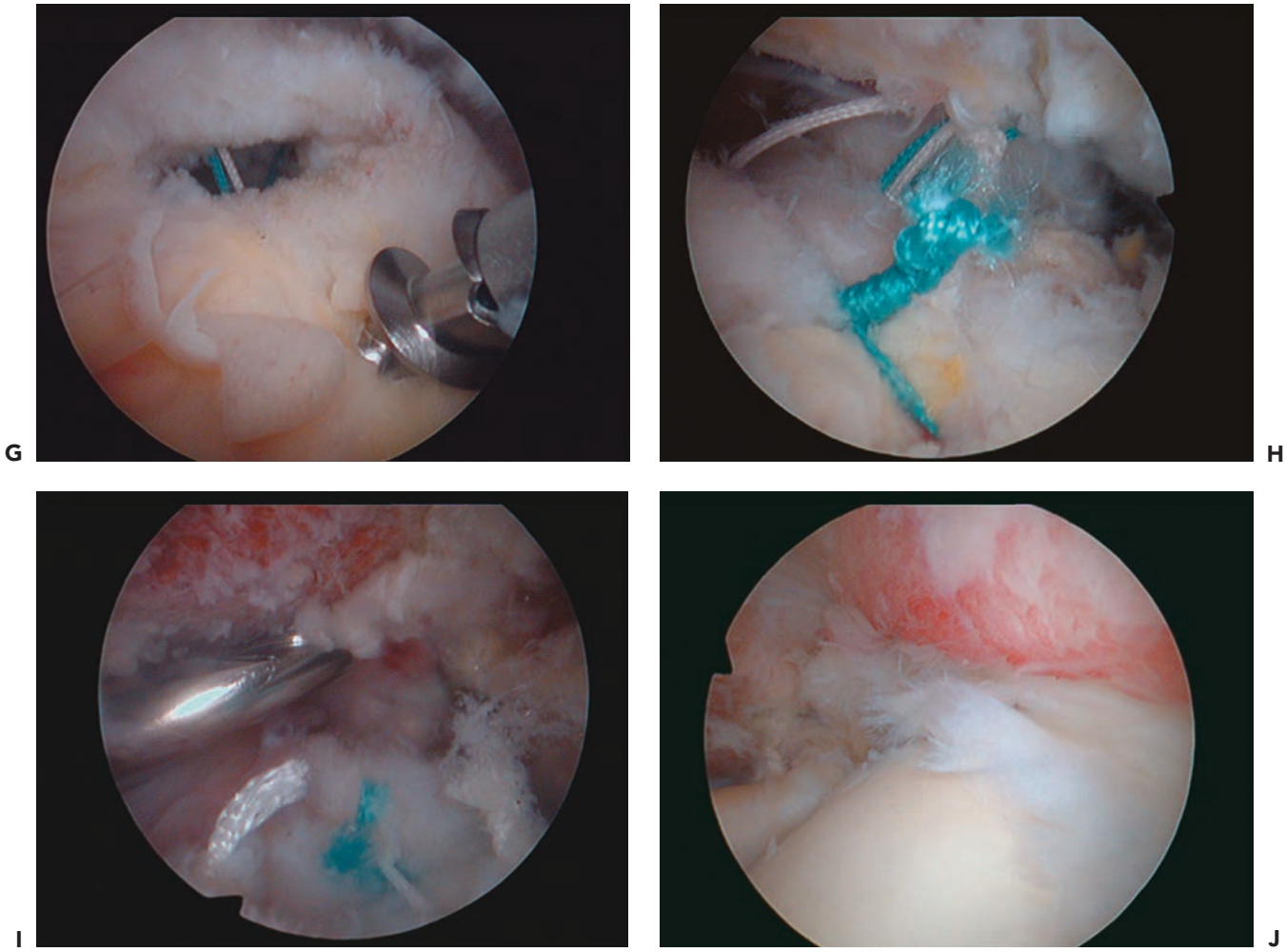


Figure 3-36 (*continued*)

scores improved from 6 to 10 after an average follow-up of 4 years. Gartsman and O'Connor reported their results of a randomized study comparing the treatment of rotator cuff repairs with either a concomitant acromioplasty or no acromioplasty.[89] All patients in the study had a type II acromion, and all the rotator cuff tears involved only the supraspinatus tendon. After a minimum 1-year follow-up, the mean American Shoulder and Elbow Surgeons (ASES) score for the acromioplasty group was 91.5, and for the nonacromioplasty group was 89.2, which was not statistically significant. Tear size, patient age, and preoperative ASES scores were the same in both groups.

Many techniques have been described for the management of massive rotator cuff tears, including débridement,[5,110,145,150,189,227] partial repair,[27] mobilization and repair of cuff tissue,[16,51,56,60,69,110,140,142,154,156,157] tendon transfer,[31,52,90,119,156,161,164] implantation of fascia,[12] allografts,[162] and the placement of synthetic material.[172] Mobilization and transposition of existing rotator cuff tissue has generally yielded better results than implantation of fascia, allografts, synthetic material,[16,51,56,76] or cuff débridement.[5,110,145,150,220,227]

Ellman and coworkers[69] were able to show a correlation between a poor result and preoperative strength and active range of motion. If patients had grade three-fifths strength or less or were unable to abduct their shoulder beyond 100 degrees, there was an increased risk of a poor result. When patients have an unsatisfactory result, it is usually associated with poor function and not pain relief. This is supported by Bigliani et al.[16] and Hawkins et al.,[110] who found that there was good pain relief for repairs of massive tears, but functional improvement was less predictable.

Finally, the final outcome of rotator cuff repair may not necessarily be directly related to complete healing of the tendon. Calvert and coworkers[32] demonstrated good function, pain relief, and satisfaction despite having a documented dye leak at follow-up shoulder arthrography. Packer and coworkers had similar findings[173] and suggest a "water-tight closure" of a cuff defect is not necessary. Likewise, Harryman and coworkers[104] found up to 50% of their

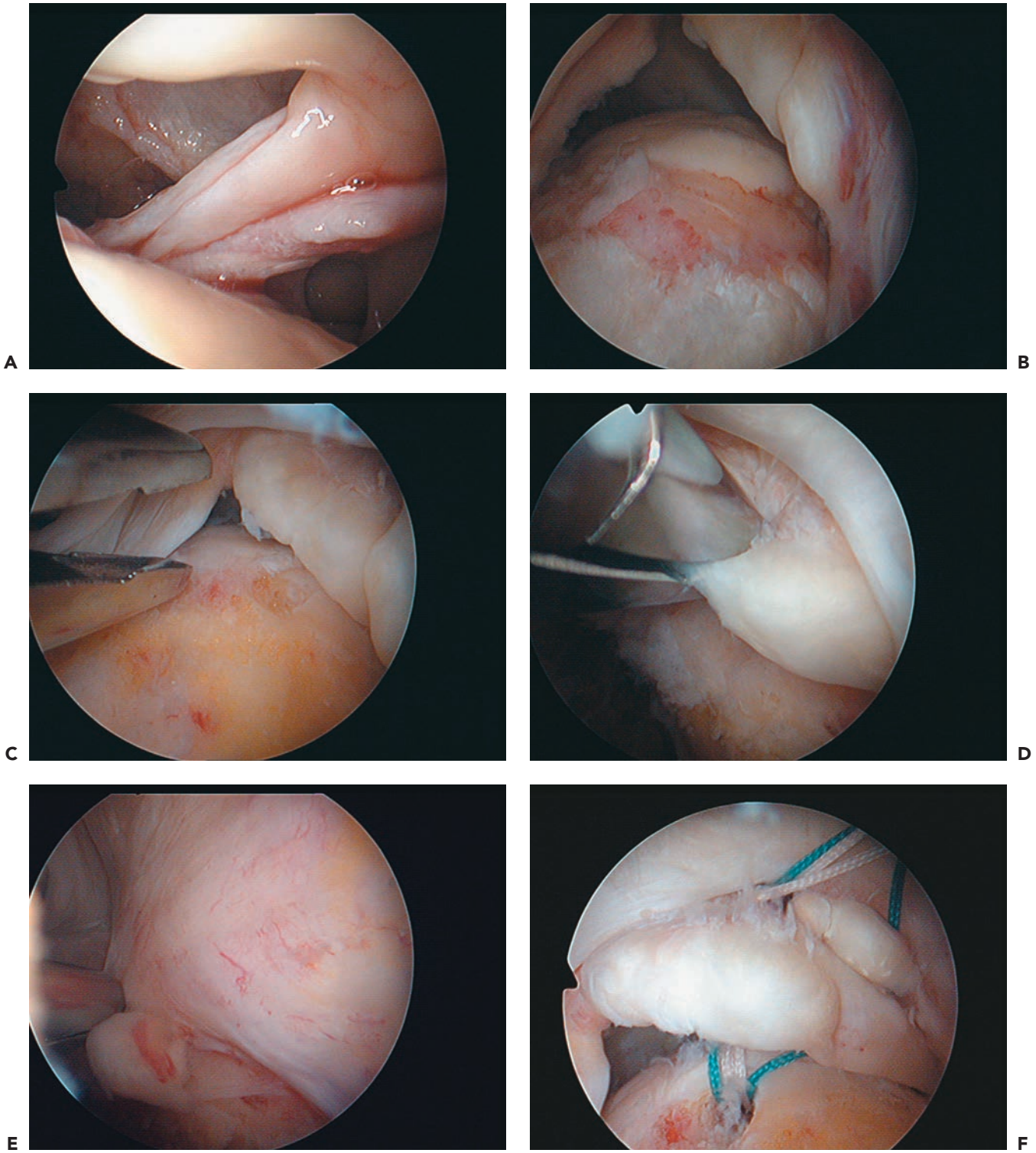


Figure 3-37 Infraspinatus delamination tear. **(A)** Air arthroscopy (articular side) showing the delamination of the cuff tear. **(B)** V-shaped tear from the bursal surface showing the delamination having a deep articular surface tear **(C,D)** that requires repair to the medial footprint of the greater tuberosity **(E–G)** and a superficial surface tear requiring repair to the lateral aspect of the greater tuberosity **(H–L).**

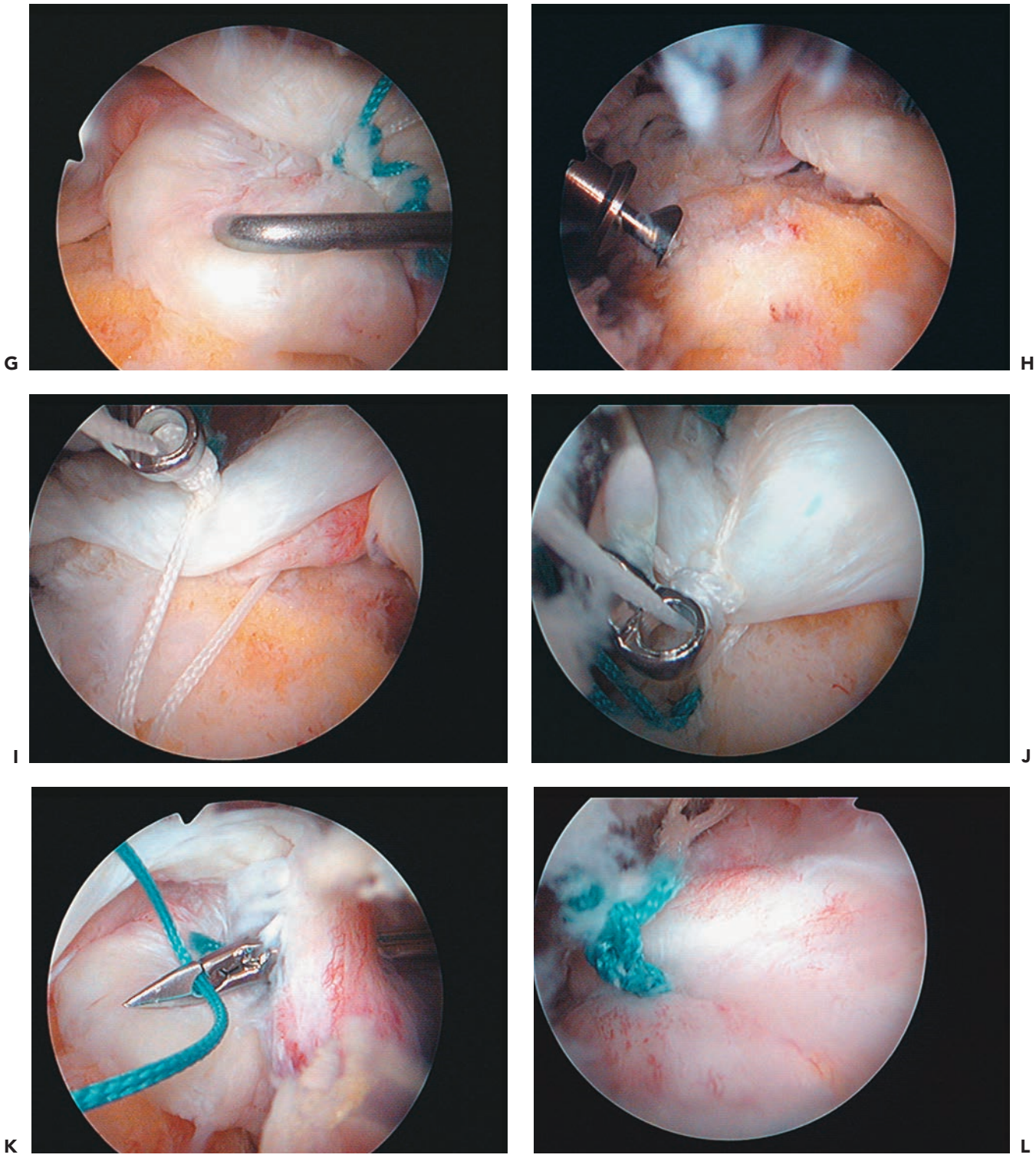


Figure 3-37 (*continued*)

cuff repairs had a postoperative defect. This did not adversely influence patient satisfaction or pain relief, but it did affect shoulder strength. Indeed, in their study the most important factor affecting strength and function at follow-up was a maintained tendon repair. This, combined with the poor results reported for decompression without repair of cuff tears,[145] has led to an increased emphasis on the technical adequacy of tendon repair, including the use of stronger sutures, tendon or bone augmentation, and postoperative bracing.[92,207]

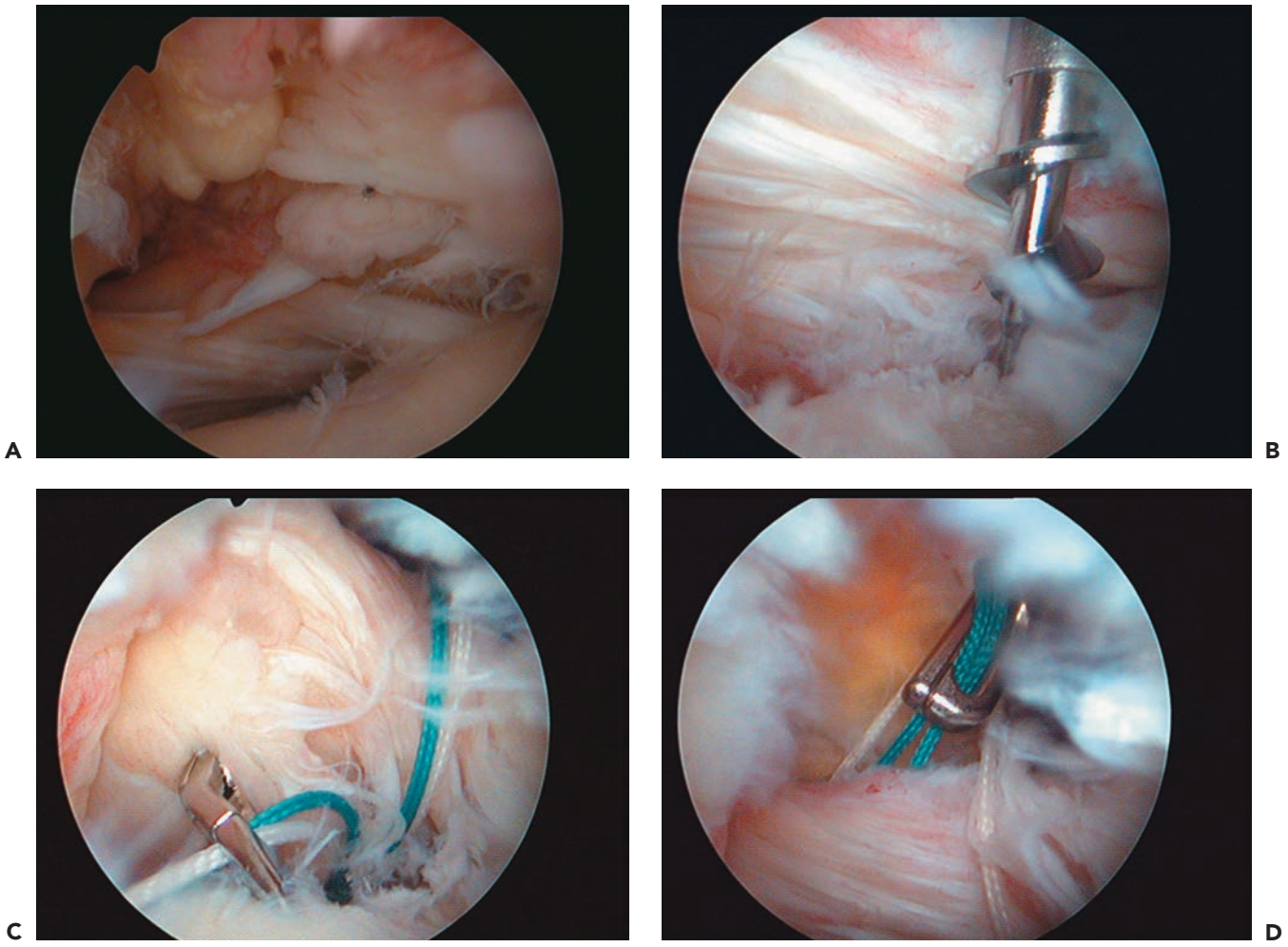


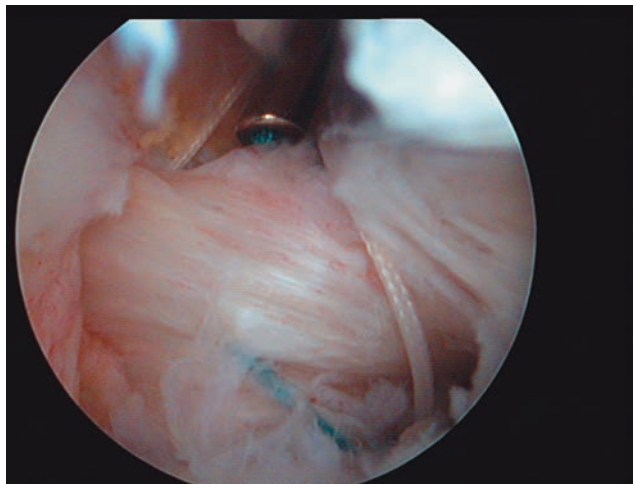
Figure 3-38 **(A)** Upper portion of subscapularis has a 1-cm tear of the rolled edge of the tendon. **(B)** The anchor is placed just medial to the bicipital groove. **(C)** The tendon is pierced and the sutures grasped and pulled through the tendon. **(D–G)** The sutures are retrieved and tied.

A particularly careful and well-documented study[120] found 88% good or excellent results after cuff repair, and good correlation between patient satisfaction and objective measures such as the Constant score.[118,119]

Gerber et al. reported their results of open repair of 29 massive rotator cuff tears after a minimum 2-year follow-up.[93] The tendons were repaired with a modified Mason-Allen suture technique fixed to the humerus through a thin titanium plate for cortical bone augmentation. The Constant score improved from 49% to 85% of the normal shoulder, and forward flexion improved from 92 to 142 degrees. Patients with two-tendon tears had better motion, less pain, and higher Constant scores than patients with three-tendon tears. Postoperative MRI revealed retears in 34% of patients, and the patients with failed repairs had significantly lower Constant scores, more pain, and less active motion. Muscle atrophy could be reversed in the supraspinatus muscle if the repair did not fail, but atrophy could not be reversed in the other muscles of the rotator cuff. Fatty degeneration increased in all muscles regardless of the repair integrity.

For the purpose of finding an association between repair integrity and outcome, Klepps et al. reported their results of open repair of 32 medium and large rotator cuff tears.[126] After a minimum 1-year follow-up, the UCLA shoulder score was 31 for tears less than 3 mm and 29 for tears greater than 3 mm, which was not significantly different. The retear rate by MRI at 1 year was 31%, and patients with failed repairs had lower UCLA scores and worse pain scores.

In 1999, Rokito et al. reported their results of open repair of 30 large and massive rotator cuff tears after a minimum 4-year follow-up.[190] All of the patients were satisfied with the procedure, and the average UCLA score improved from 12 to 31. The mean peak torque in flexion, abduction, and external rotation was 80%, 73%, and 91%,



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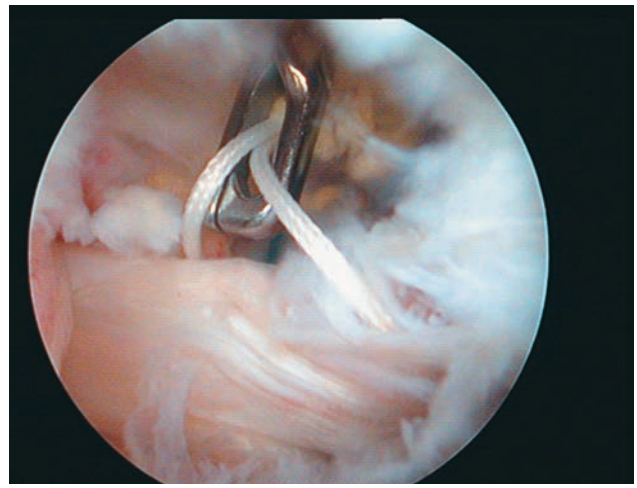

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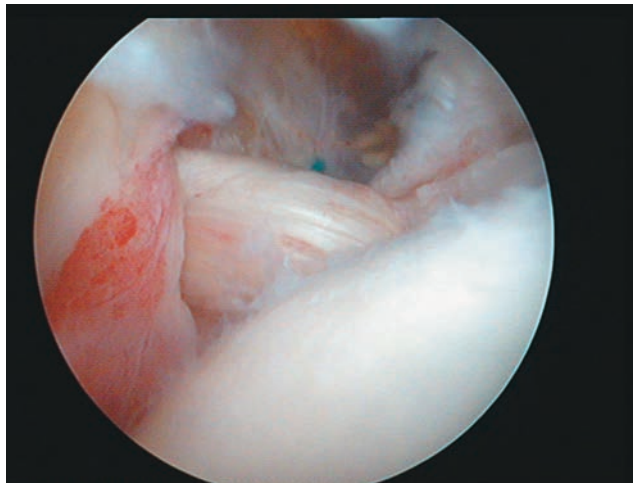

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Figure 3-38 (*continued*)

respectively, of the normal shoulder. This study suggests that the results of open repair are durable after many years, and that patients may require more time to regain their maximum shoulder strength. In the same year, Romeo et al. reported their results of open repair of 72 full-thickness rotator cuff tears after a minimum follow-up of 2 years.[191] Twenty-one tears were massive tears, but they were all repairable. Seventy-six percent of the patients had minimal pain, were able to perform activities of daily living, had 75% of normal strength, and lost less than 20 degrees of forward flexion. Patients with massive cuff tears had an average UCLA score of 81 compared to patients with a smaller tear, who had a score of 91; women with an associated biceps tendon rupture had an average UCLA score of 65 compared to women without a biceps rupture, who had a score of 95.

In a study with the longest follow-up after rotator cuff surgery, Galatz et al. reported their results of 33 open repairs after 10 years of follow-up.[84] The raw Constant scores were the same at 2- and 10-year follow-up, but after the scores were normalized for expected age-related activity level, the Constant scores were even greater after 10-year than after 2-year follow-up. Twelve patients continued to work at their same occupation, and only two patients retired because of problems with their shoulder. The patients' subjective evaluation of the procedure did not change after 10 years (Table 3-2).

The results of arthroscopic acromioplasty with a mini-open rotator cuff repair are good. Levy and coworkers reported 80% satisfactory result.[132] Of their patients 96% were satisfied with the procedure. We have recently reported our experience with mini-open rotator cuff repairs following an arthroscopic acromioplasty.[182] All 30 of our patients had an excellent result at an average of 25 months of follow-up. All the tears in this study group were either small or medium.

The short-term benefits of mini-open repair were reported by Hata et al.[105] The authors compared their results of mini-open repair to a cohort of patients who were treated earlier with a classic open repair. No massive tears were included in the study. At 3- and 6-month follow-up, the mini-open group had 12 degrees more forward flexion,

TABLE 3-2
RECENT CLINICAL STUDIES (LAST 5 YEARS) REPORTING RESULTS OF OPEN ROTATOR CUFF REPAIR

Author	Year	Number of Patients	Outcomes	Minimum Follow-up	Number of Massive Tears	Failed Repairs
Rokito et al.[190]	1999	30	UCLA 31 Excellent + good results 76%	4 years	13 (43%)	
Romeo et al.[191]	1999	72	Satisfactory 76% Constant score Massive 68 Others 82	2 years	21 (29%)	
Knudsen et al.[127]	1999	31	Constant score Intact cuff 75 Cuff defect 62	1 year	0	32%
Gerber et al.[93]	2000	29	Normalized Constant 89%	2 years	29 (100%)	34%
Galatz et al.[84]	2001	33	Excellent + good 91%	10 years	11 (33%)	
Pai and Lawson[174]	2001	58	Excellent + good 76%	1 year	13 (22%)	
Klepps et al.[126]	2004	32	Constant 80	1 year	13 (40%)	31%
Duralde and Bair[65]	2005	24	Excellent + good 67%	2 years	24 (100%)	

but at 1-year follow-up, motion and strength were the same. The average UCLA score was 33 at 1-year follow-up.

Reports by Hersch and Sgaglione showed that excellent results can be achieved in up to 90% of patients when the mini-open approach is used.[114] The authors also cited the added benefit of arthroscopic visualization of the glenohumeral joint to diagnose concomitant biceps, articular, or AC joint pathology. What is lacking from these recent studies are significant numbers of patients with massive rotator cuff tears who have been shown to have worse results after classic open repairs.

Entirely arthroscopic repair is still in evolution, but early results have been variable depending on cuff tear size, location of the tear, and chronicity.[87,135,218] The results seen with arthroscopic repair have been consistent with those seen with open repair when comparing patient and anatomic factors that affect outcome. Weber compared 39 entirely arthroscopic repairs with 101 arthroscopic-assisted mini-open repairs; he found lower early morbidity, but also a higher complication rate, including three loose anchors (8%), in the arthroscopic group.[218] After 6 weeks, the outcomes were identical. Three recent series contained 137 patients followed for at least 1 year after arthroscopic cuff repair;[20,197,210] overall, 88% achieved good or excellent results.

Numerous studies have shown that arthroscopic repair of full-thickness rotator cuff tears provides excellent or good results in 85% to 95% of patients after a minimum 2-year follow-up. The strengths of these studies include the use of validated outcome scores, uniform surgeon experiences, and large patient numbers. One major deficiency in the arthroscopic literature is the small number of massive rotator cuff repairs, which has been shown in previous studies to adversely affect functional outcome scores. Many studies that include all sizes of cuff tears will report that statistical analysis did not find any correlation between outcome scores and tear size, even though the number of massive tears in the study is small. Data analysis in each of these studies may be misleading with regard to tear size and outcome because of the lack of statistical power, not because the association does not exist when using arthroscopic repair techniques. Another confounding variable that is not reported in any of the arthroscopic studies is muscle atrophy or fatty infiltration, which has been shown by Gerber et al. to play a significant role in the outcome of open rotator cuff repairs.[93] So while most reports show that the average rotator cuff tear can be treated successfully with arthroscopic techniques, questions remain regarding the best approach to use for the treatment of massive rotator cuff tears with muscle atrophy.

In 1998, Gartsman et al. reported their results of arthroscopic treatment of 73 rotator cuff tears after a minimum 2-year follow-up.[88] Six patients had massive tears. The

Constant score improved from an average of 41 to 89, and 83% of patients had an excellent or a good result. Burkhart et al. treated 59 patients with arthroscopic rotator cuff repair directly to bone or with margin convergence alone.[28] This series included 13 massive cuff tears. Excellent and good results were found in 95% of patients according to the Constant score. The average score for massive tears was 29.9, while the average score for all others was 31.6.

Only one study to date has reported the results of the integrity of arthroscopically repaired rotator cuffs. Galatz et al. repaired 18 rotator cuff tears that were all larger than 2 cm and evaluated their patients with ultrasound 1 year after the repair.[85] Seventeen of the 18 patients had recurrent tears, yet the average ASES score improved from 48 to 80, and all of the patients were satisfied with the procedure (Table 3-3).

The personal experience of one of the authors (LL) with 116 shoulders in 115 patients operated on for full-thickness rotator cuff tears from 1998 to 2001 was evaluated.[20] In this series, there were 63 (55%) men and 52 (45%) women with a median age of 57 years (range 36 to 80 years). There were 13 anterosuperior lesions (six large subscapularis tears) and 40 superior, 36 posterosuperior, and 27 massive rotator cuff tears.

The patients were evaluated pre- and postoperatively according to the criteria of the Constant and Murley Score (CMS).[4] Preoperatively, all patients had a standard set of radiographs, arthrograms, and arthro-CT scans; postoperatively all had standard radiographs, 89 patients had an arthrograms, and 17 of these had an additional CT scan. Pre- and postoperative images were measured for humeral head–acromion distance, arthrographic signs of retear, tear retraction, and, when CT scan was available, fatty degeneration of the muscles according to the criteria of Goutallier et al.[99]

Average follow-up was 26 months (24 to 60 months). There were no infections for frozen shoulder. Revision surgery was performed for retear, biceps problems, and captured cuff problems.

The CMS increased from 40.3 for that of a normal shoulder preoperatively to 80.1 postoperatively. All parameters of CMS were improved. The postoperative scores correlated with the high patient satisfaction. The greatest improvement was with anterosuperior tears, with an average increase of 43.6%. The average increase for superior lesions was 41.1%, for the posterosuperior lesions 39.5%, and for the massive lesions only 36.4%.

The best improvement in pain was found in the posterosuperior lesions, the greatest improvement in activities of daily living were with superior lesions, and the best outcome in mobility and strength were with anterosuperior lesions. The influence of the intraoperative state of the LHB and its treatment were not studied in the anterosuperior lesion. Eighty-seven of the 116 patients had a postoperative arthrogram or arthro-CT. Forty-two (48.3%) were watertight and 45 (51.7%) had a positive arthrogram. The presence of complete healing based upon the presence of a leak on arthrogram was different among the different lesions.

A negative arthrogram is clearly a complete healing of the tendon but a leak of contrast can be correlated with a recurrent tear or to a nonwatertight repair; however, in some shoulders this may still represent partial healing or almost complete healing in a case when the rotator interval was not closed. Our arthrogram data are difficult to evaluate because of incomplete recording of the intraoperative estimate of the cuff repair or the nature of the rotator interval closure.

Correlation between clinical results and postoperative arthrograms demonstrate that patients with a leak of contrast (51.7%) had a greater gain in all CMS variables than those with a watertight cuff, but both patient groups had similar absolute postoperative Constant scores. This further supports our opinion that a leak on a postoperative arthrogram is not always correlated to a clinically significant recurrent tear. Conversely, a poor clinical result, in

TABLE 3-3
REVIEW OF RECENT LITERATURE ON THE RESULTS OF ARTHROSCOPIC ROTATOR CUFF REPAIR

Author	Year	Patients	Outcome	Massive Tears
Gartsman et al.[88]	1998	73	Excellent + Good 83%	6 (8%)
Tauro[211]	1998	43	Excellent + Good 64%	0
Burkhart et al.[28]	2001	59	Excellent + Good 95%	13 (22%)
Murray et al.[144]	2002	48	Excellent + Good 96%	0
Bennett[13]	2003	24	ASES 83 Constant 73	0
Bennett[14]	2003	37	ASES 77 Constant 74	37 (100%)
Jones and Savoie[124]	2003	50	Excellent + Good 88%	13 (26%)
Wolf et al.[223]	2004	95	Excellent + Good 94%	0

which recurrent surgery was necessary, was always correlated with a massive leak of contrast on arthrogram and a massive retraction at the second surgery. In some cases revision surgery was required for persistent untreated biceps pathology or a cuff capture due to adhesions, and in these cases there was a negative arthrogram.

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4 Management of Irreparable Rotator Cuff Tears: The Role of Tendon Transfer

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INTRODUCTION

Irreparable tendon tears of the rotator cuff are found in a small proportion of patients who undergo operative treatment, as approximately 95% of all tears are reparable by

conventional methods.[131] Open and arthroscopic repair techniques have been shown to consistently improve pain and function in 90% of selected patients, although structural integrity of the repair following surgery may not correlate with positive outcomes.[4,10,30,31,35,37–39,60,62,76,83,108,110,132] The variability in clinical outcome regardless of the integrity of the repair may be more a reflection of the unclear natural history of this disease rather than the response to treatment.[4,35,44,60,72,76,83] It is clear, however, that larger tears carry a much greater risk of failure following surgery and are associated with worse clinical outcomes.[44] Furthermore, recent studies have shown that simple débridement of massive tears are not associated with a high degree of satisfaction in the younger or active patient.[101]

While the prevalence of irreparable tendon tears is quite low, they can profoundly impact the functional capacity of patients and can be associated with significant pain. Numerous tendon transfers have been proposed in the treatment of irreparable rotator cuff tears. These transfers have included the pectoralis major,[36,42,45,70,73,111,133,138] latissimus dorsi,[2,43,45,52,66,94,130,131,134] supraspinatus,[26,59] subscapularis,[23,74] trapezius,[92] teres major,[21] long head of the triceps,[61,87,120] and teres minor.[98] Interpositional materials have also been recommended as a possible treatment and have included allografts,[97] fascial autografts,[7] synthetic materials,[95,102] the lateral deltoid,[3] the long head of the biceps tendon,[58] and xenograft.[115] The variability in the published results of these techniques demonstrates a lack of consensus on the optimal treatment of massive, irreparable rotator cuff tears. Moreover, most reports are based on anecdotal experience or retrospective review. Thus, inherent biases and confounding variables make it difficult to draw any meaningful conclusions about the best method of treatment. For these reasons, the prognosis after an attempted reconstruction using these techniques remains unclear.

The criteria that enable one to distinguish patients with irreparable rotator cuff tears who are candidates for tendon transfer from those who are better served by other methods or skillful neglect will be discussed. The following chapter will also focus on the treatment of irreparable anterosuperior and posterosuperior rotator cuff tears using techniques that have shown consistency in clinical practice and validation in the literature.

In some patients, although there is a massive irreparable cuff tear, there is sufficient rotator cuff and deltoid function to allow the patient to have sufficient strength and elevation to serve their functional needs. In most cases these individuals are over the age of 70 years, are retired, and their primary complaint is pain. In some cases these patients maintain weak over-shoulder-level elevation. When weakness is not a primary complaint and pain is of primary concern, a limited-goals arthroscopic débridement of the bursae, release of a damaged biceps tendon, and débridement of a painful and symptomatic acromioclavicular arthritis can provide sufficient improvement of pain to result in improved patient function and patient satisfaction. In these cases a muscle transfer may help with functioning but may not be required for the patient's needs. This is particularly a consideration in the older patient with low functional demands.

DEFINITION OF AN IRREPARABLE TEAR

The definition of an "irreparable" rotator cuff tendon tear has evolved in recent years based on increasing scientific evidence that substantiates the critical interplay of tendon reparability with muscle and tendon quality.[46,47,55,56] It is becoming accepted that tendon size is a less important determinant of reparability and outcome as is the degree of tendinous and muscular degeneration, fatty infiltration, and cranial migration of the humeral head.[30,55,88,126,137] The poor correlation in published reports between tear size and functional outcome has confirmed this concept.[9,13,22,113]

Irreparable tears that may be indicated for a latissimus muscle transfer are defined as those involving at least two rotator cuff tendons (supraspinatus and infraspinatus) with retraction that is not amenable to mobilization and repair to the anatomic footprint with the arm in less than 60 degrees of abduction.[52] Mobilization may include release of the coracohumeral ligament, interval slide, and circumferential capsulotomy.

We currently believe that the determination that a rotator cuff tendon tear is irreparable can be made by preoperative evaluation without the need to attempt surgical mobilization. This contention is based on an understanding of the biomechanical and structural changes that accompany a chronic, massive tear of the rotator cuff.

Preoperative criteria that enable the surgeon to determine whether a rotator cuff tendon tear is irreparable are both clinical and radiographic. The relevant clinical factors include profound weakness of internal or external rotation in combination with a marked "lag sign."[64] The weakness is usually such that, in the case of a posterosuperior tear, the patient cannot raise his or her arm against gravity (Fig. 4-1). In an irreparable tear of the subscapularis, some patients maintain the ability to raise their arm overhead, although they have profound internal rotation weakness.[133]

In the case of an irreparable posterosuperior tear, the patient will have a lag between passive and active external rotation. In these cases, the patient's arm will fall into internal rotation when the examiner releases it from maximal passive external rotation (Fig. 4-2). When the arm is at the side in adduction, this degree of lag is consistent with an irreparable tear of the infraspinatus (Fig. 4-3).

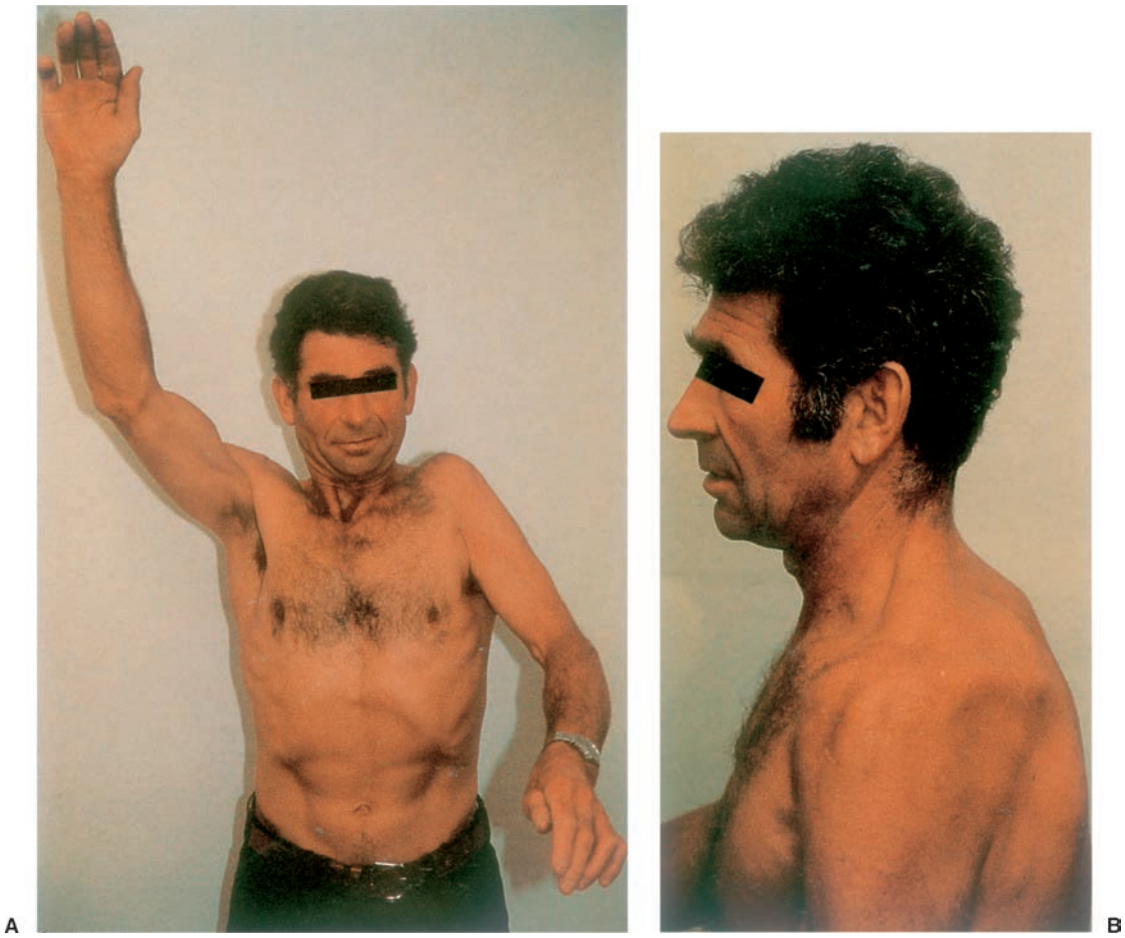


Figure 4-1 A 52-year-old mason with a traumatic posterosuperior rotator cuff injury. At examination, he showed **(A)** the inability to raise the arm against gravity, or pseudoparalysis, with **(B)** clinical evidence of severe atrophy of the supraspinatus and infraspinatus muscles.

Figure 4-2 A 59-year-old man with a chronic, massive tear of the posterosuperior rotator cuff. Demonstration of the external rotation lag sign **(A,B)** with the arm at 90 degrees of abduction, which demonstrates disruption of the infraspinatus and teres minor.

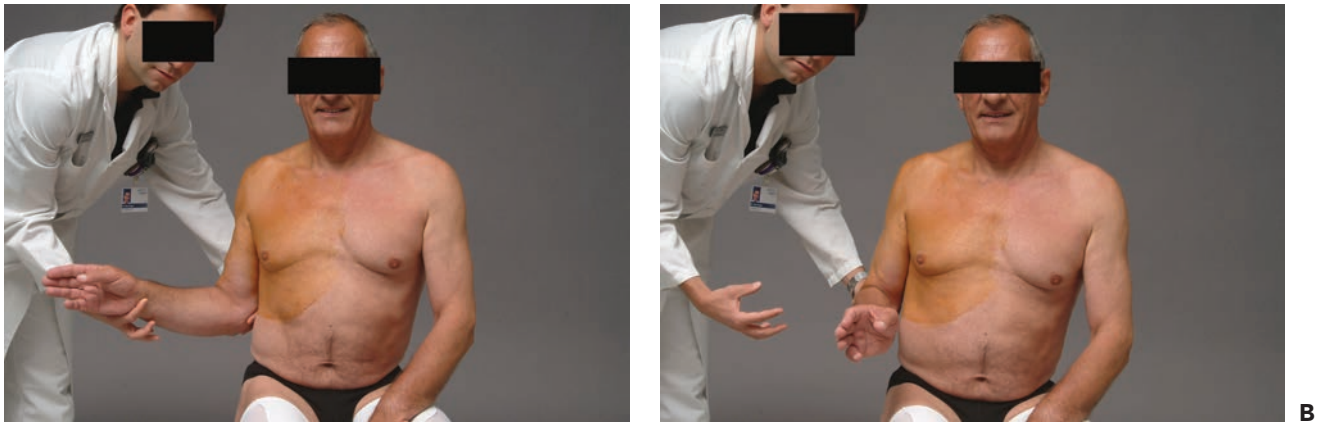


Figure 4-3 With the arm in adduction, a lag between maximal passive and active external rotation **(A,B)** is pathognomonic for a tear of the infraspinatus.

When the patient's arm is abducted and held in maximal passive external rotation position, the patient may be able to actively maintain this position if the teres minor is intact.

Anterosuperior rotator cuff tears in which the subscapularis is completely torn, with or without a concomitant supraspinatus tear, typically display positive lag signs in internal rotation. The "lift-off" sign is the inability to lift the hand off the lower back, while the "belly-press" sign is the inability to maintain the hand against the abdomen without the elbow moving backward when resistance is applied against internal rotation (Fig. 4-4).[46,47,64] Each of these maneuvers tests the function of the subscapularis at terminal internal rotation.[46,47,64]

Other clinical findings that suggest an irreparable massive tear are superior displacement of the humeral head combined with anterior or posterior translation. In cases of massive posterosuperior tears, the humeral head may display proximal migration in combination with excessive scapular motion. In contrast, patients with massive anterosuperior tears will exhibit superior migration of the humeral head with anterior translation when attempting abduction (Fig. 4-5).

The radiographic parameters suggesting an irreparable tear include static superior subluxation with an acromiohumeral interval less than 5 mm on an anteroposterior radiograph[8,15,30,72] and static anterior subluxation in the case of a chronic subscapularis rupture.

Magnetic resonance imaging (MRI) and computed tomography (CT) scans that demonstrate profound muscle atrophy with a Goutallier stage of 3 or greater also suggest a tear with poor-quality tissue that will consistently fail to heal with surgery (Fig. 4-6).[44,55,56,72,131,135]

Although most irreparable tears tend to be "massive tears" of the posterosuperior cuff, irreparable and massive tears are certainly not synonymous. The characterization of a cuff tear as irreparable is based not only on size, but also on tissue quality, degree of retraction, and, in some cases, the skill and experience of the surgeon.[52] Despite the fact that tendinous tissue can be sewn together either with arthroscopic or open methods, the incidence of disruption of large and massive tears is very high.[9,27,72,84,130,142]

The definition of a "massive tear" continues to be controversial. The most commonly accepted definition of a massive tear in North America is defined by Cofield as one with a maximum diameter of 5 cm or greater.[22] Others, including Gerber,[44,64,72,99] have claimed that this, perhaps, is not an ideal definition as absolute size may be variable among patients and inconsistently reported before or after débridement of nonviable edges during surgery (Fig. 4-7). For this reason, we support a definition of "massive tears" as those involving disinsertion of two or more rotator cuff tendons.[107,111]

In Neer's series of 340 rotator cuff tears operated on over a 13-year period, 145 (43%) were classified as massive.[96] Ellman and associates reported that 9 of 54 (17%) rotator cuff repairs involved massive tears.[30] Harryman et al. reported that 28 of 105 (27%) surgically treated tears were massive.[60] Warner reported that 146 of 407 (36%) rotator cuff repairs involved massive tears, and that 19 of 407 (5%) involved combined supraspinatus and subscapularis tears.[130] Despite the fact that the definition of massive tears was variable in these studies, they do provide a rough estimate of their frequency in a referral shoulder specialist practice. It is very likely that the percentage of all tears that undergo surgery that are massive tears is substantially less in the general surgical population.

Posterosuperior tears are the most common configuration of massive tear and, by definition, involve the supraspinatus and infraspinatus tendons.[131] Less commonly but not infrequently, the subscapularis and supraspinatus are involved with a unique epidemiology, etiology, and prognosis that has been highlighted relatively recently and is gaining appreciation in the literature.[44,47,138]

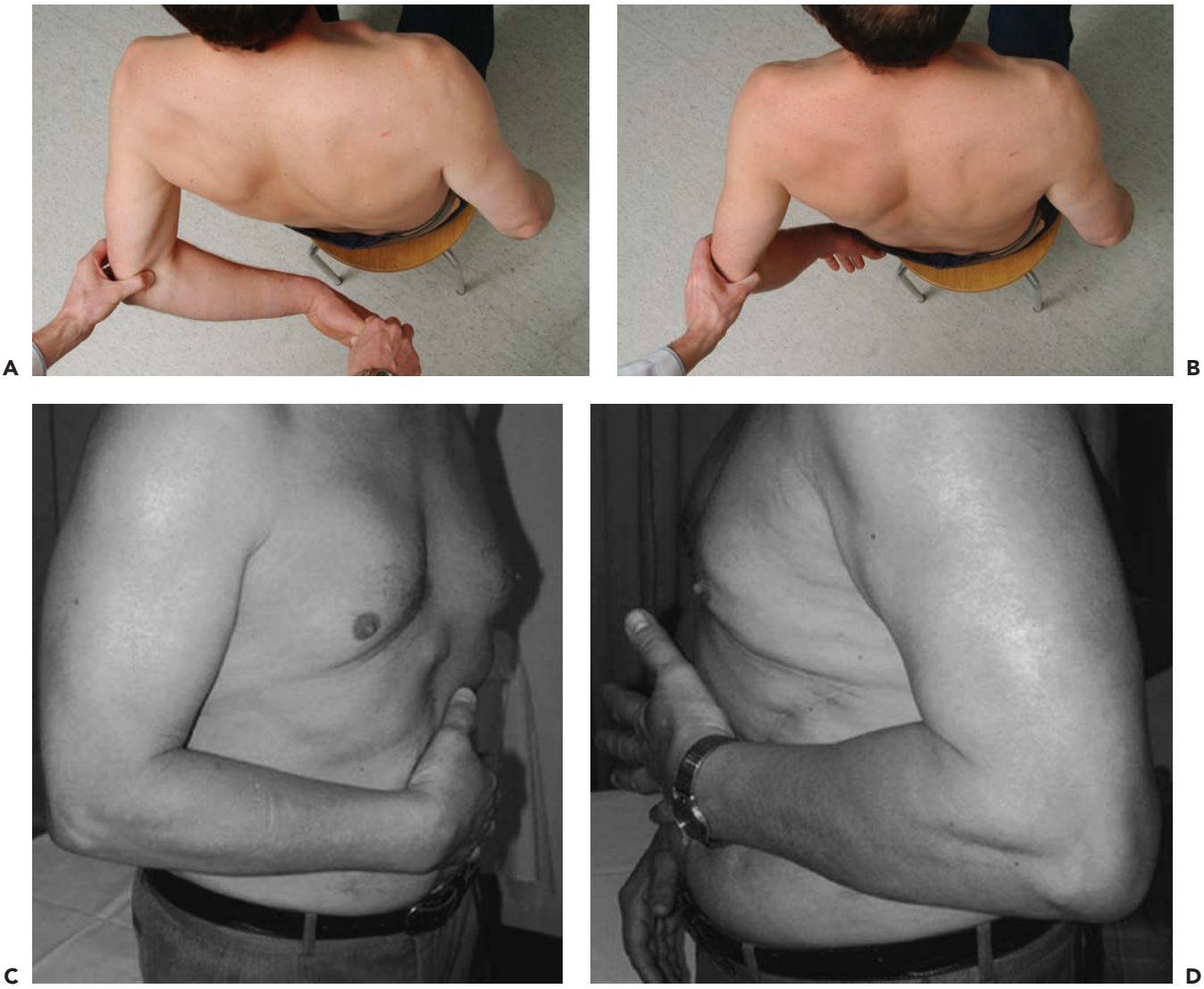


Figure 4-4 The lift-off and belly-press tests are used to test the integrity of the subscapularis tendon. In the lift-off test the patient is asked to lift his or her hand off the lower back. It has been found to be more sensitive and specific if there is the presence of an internal rotation lag sign after the clinician releases the hand from maximal internal rotation with the arm off the back **(A,B)**. In the belly press sign **(C,D)**, the patient exerts an internal rotation force on the belly, with the elbow forward and anterior to the midline of the trunk. If the subscapularis is ruptured, the patient is unable to keep his or her hand on the stomach with resisted internal rotation, and the elbow will fall back posteriorly. (Reprinted with permission from Gerber A, Clavert P, Millett PJ, Holovacs TF, Warner JJP. Split pectoralis major transfer and teres major tendon transfers for reconstruction of irreparable tears of the subscapularis. *Tech Shoulder Elbow Surg* 2004;5:5–12.)

PATHOANATOMY AND PATHOMECHANICS

The role of the rotator cuff muscles and of the deltoid have been studied clinically and experimentally in great detail.[15,25,53,65,68,75,89] The force generated by an individual rotator cuff muscle is largely determined by its physiologic cross-sectional area (PCSA) (Table 4-1).[65,75,82,122,124] The moment is the product of the force and the distance from the center of rotation to a perpendicular line drawn along a muscle's line of action (Table 4-2).[5,6,11] As Tables 4-1 and 4-2 indicate, the supraspinatus is relatively weak given its small PCSA, smaller size, and closer insertion to the axis of rotation. Furthermore, its relative contribution is decreased with increasing abduction. Keating et al. have shown[75] that the supraspinatus, for example, makes a small (15%) contribution to the overall abduction moment arm of the shoulder compared to the subscapularis (52%) and to the combined effort of the infraspinatus and teres minor (33%).

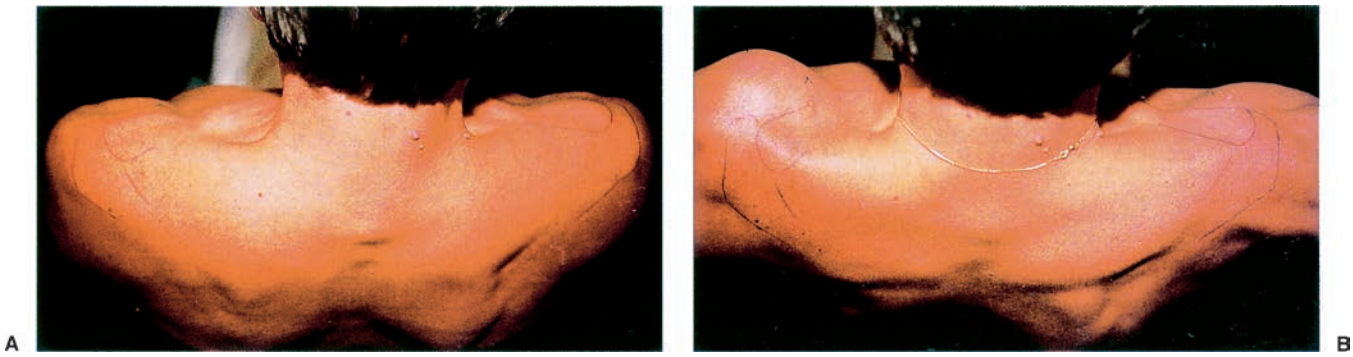


Figure 4-5 Dynamic anterosuperior subluxation indicates major injury to the supraspinatus and subscapularis. The clinical diagnosis is made if **(A)** the patient has normal contour of both shoulders at rest and **(B)** subluxates his or her shoulder anterosuperiorly while resisting abduction. The condition of anterosuperior subluxation can be precipitated by open or arthroscopic subacromial decompression, with release of the coracoacromial ligament. If anterosuperior subluxation becomes static, clinically or radiographically, successful restoration of overhead elevation by direct surgical repair is exceedingly rare, and subacromial decompression is detrimental.

The excursion of the rotator cuff tendons is relatively small (range 0.5 to 4.0 cm) during scapular abduction. In contrast, the deltoid muscle has an excursion of 6.5 cm (Table 4-3).[90] The rotator cuff muscles are therefore important stabilizers of the humeral head that provide a fixed fulcrum of rotation that is primarily generated by the deltoid during abduction.[90] Disruption of the supraspinatus with either the infraspinatus or subscapularis disrupts this balance and can result in loss of efficiency of the deltoid during abduction.[8,81,122,137]

Clinical[13,18–20] and experimental[122] data show that the shoulder may remain well compensated even though there is a structural lesion of the rotator cuff. Extension posteriorly may result in cranial migration[125,126] and extension anteriorly may lead to loss of elevation and allow anterosuperior subluxation.[13,43,100] Even in some cases of massive rotator cuff tear, however, biomechanical decompensation does not occur, and the exact reasons for this remain unclear. It is likely that the remaining muscles have sufficient force to maintain an anterior–posterior force couple, which keeps the humeral head centered and allows for a fixed fulcrum for deltoid contraction to raise the arm.[14,15,18,19] Equally confounding is the observation that some patients with relatively small tears may have poor function (Fig. 4-8).[13,15,18,20]

Muscle Atrophy and Fatty Degeneration

Goutallier et al. (59) were the first to emphasize the correlation of overall shoulder function with the degree of fatty degeneration and muscle atrophy that was evident radiographically in selected muscles of the rotator cuff. He recently validated a global fatty infiltration index (GFDI) consisting of the degree of fatty infiltration of the supraspinatus, infraspinatus, and subscapularis divided by three. He has shown that a value of 2 or greater is predictive of tear recurrence following surgery.[57] Of 220 patients who underwent rotator cuff repair, the highest percentage of recurrent tears was noted in the posterosuperior tears (56%). While Goutallier used CT to assess and grade muscle atrophy and fatty infiltration, the use of MRI has been investigated by others who have shown good to excellent inter- and intraobserver reliability, but poor correlation between MRI and CT.[32] More recently, Pfirrmann and

TABLE 4-1
PHYSIOLOGICAL CROSS-SECTIONAL AREAS FOR ROTATOR CUFF MUSCLES (IN % OF TOTAL CUFF)

Muscle	Bassett[6]	Keating[75]	Herzberg[65]
Subscapularis	46	52	51
Supraspinatus	16	15	16
Infraspinatus	38	33	33

TABLE 4-2
ROTATIONAL POTENTIAL OF ROTATOR CUFF (PERCENTAGE)

Muscle	Bassett[6] arm 90 abd + 90 er[a]	Keating[75] arm at side[a]
Subscapularis	42 (2.8 cm)	52 (2.3 cm)
Supraspinatus	13 (2.1 cm)	14 (2.0 cm)
Infraspinatus + teres minor	45 (3.1 cm)	32 (2.2 cm)

[a] Number in parentheses are average movement of the arms.

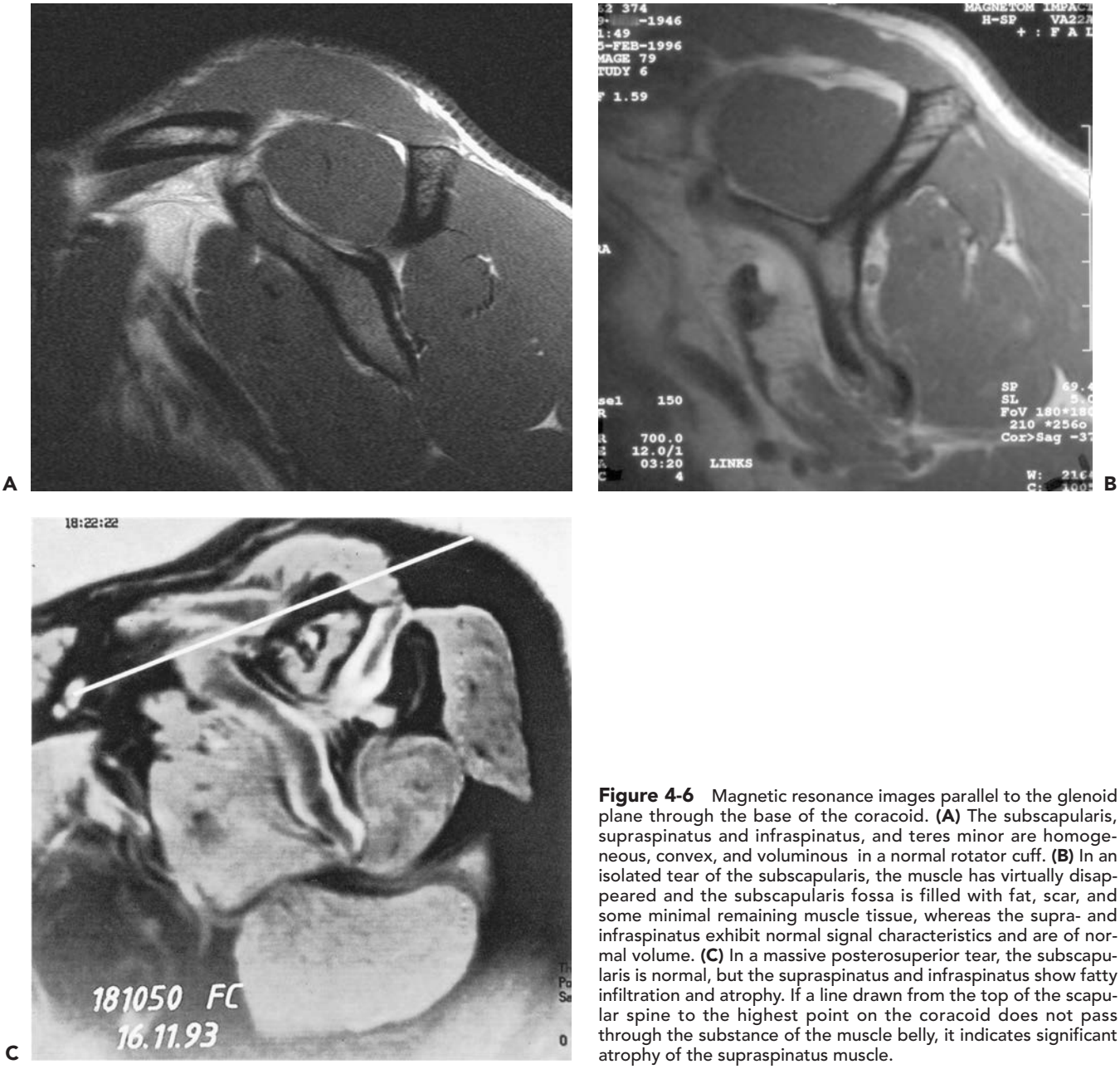


Figure 4-6 Magnetic resonance images parallel to the glenoid plane through the base of the coracoid. **(A)** The subscapularis, supraspinatus and infraspinatus, and teres minor are homogeneous, convex, and voluminous in a normal rotator cuff. **(B)** In an isolated tear of the subscapularis, the muscle has virtually disappeared and the subscapularis fossa is filled with fat, scar, and some minimal remaining muscle tissue, whereas the supra- and infraspinatus exhibit normal signal characteristics and are of normal volume. **(C)** In a massive posterosuperior tear, the subscapularis is normal, but the supraspinatus and infraspinatus show fatty infiltration and atrophy. If a line drawn from the top of the scapular spine to the highest point on the coracoid does not pass through the substance of the muscle belly, it indicates significant atrophy of the supraspinatus muscle.

coworkers[109] used MRI techniques to measure fat content in the rotator cuff muscles, and this method promises to be the most accurate means of characterizing muscle health in patients with massive rotator cuff tears.

Recent experiments have attempted to elucidate the pathogenesis of fatty infiltration of muscle following a rotator cuff tear. Gerber and coworkers showed that fatty infiltration in a sheep model of rotator cuff tears is a necessary consequence following macroarchitectural change rather than a degenerative process.[91] As the tendon tears and the muscle retracts, the pennation angle of the muscle decreases, enabling the space in between individual muscle fibers to become replaced with fat (Fig. 4-9). The structural changes that occur in this manner within the muscle unit are associated with significant changes in their structural behavior. Specifically, the muscle present in a chronically retracted tear with fatty infiltration is characterized by increased passive tension during tensile loading that creates a pathologic stress–strain behavior that compromises stability and healing of tendon-to-bone repairs.[63] In other words, as the muscle retracts and becomes filled with fat, it becomes stiffer and less compliant. Further work by Gerber following repair of chronically retracted tears indicates that there is no improvement in vascularization, intramuscular pressure, and individual muscle fiber composition.[49] Thus, in a chronic, massive rotator cuff tear, once muscle retraction

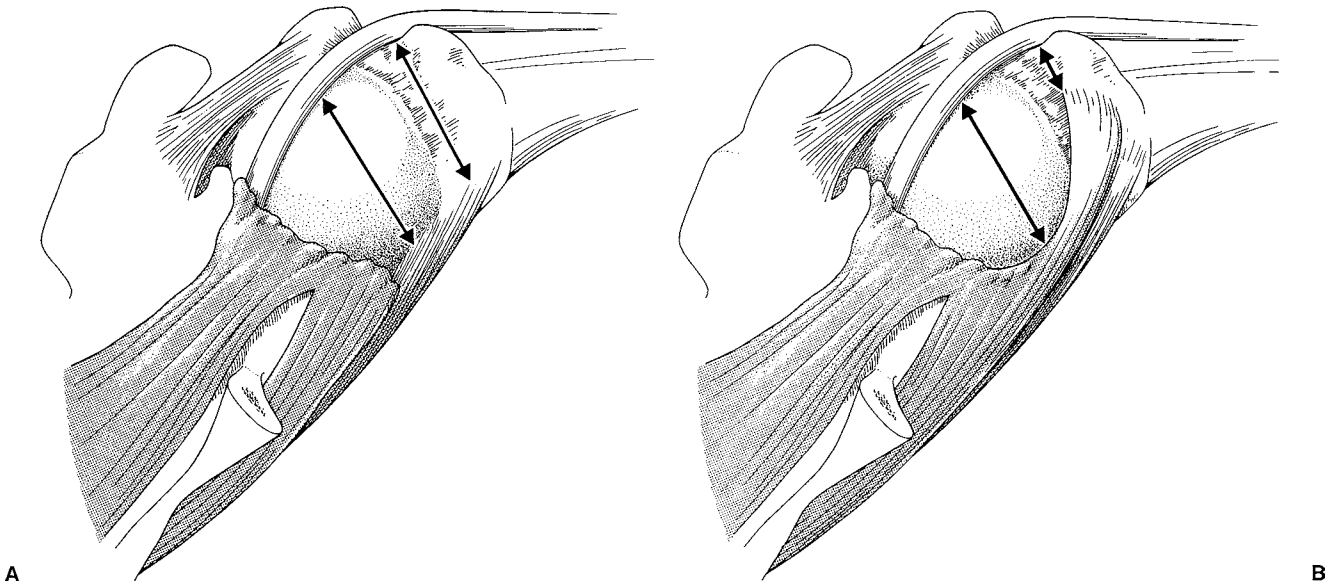


Figure 4-7 If the rotator cuff is measured in centimeters, this should be done by measuring the distance of retraction of the cuff tendons from their insertion to the greater or lesser tuberosity. This will also document **(A)** that two tendons are torn. If the size of the tear is measured from the top of the humeral head, a relatively small tear **(B)** can be mistaken for a massive tear due to buttonholing of the proximal humerus through the defect.

TABLE 4-3
RESULTS IN 21 SHOULDER GIRDLE MUSCLE UNITS

Muscle unit (n=21)	Group	Muscle Fiber arrangement	Potential excursion (cm)		Mass fraction (%)		Relative tension (%)	
			Mean	SD	Mean	SD	Mean	SD
Upper trapezius (clavicular)	A	Longitudinal	13.8	2.7	2.9	1.1	2.6	1.0
Upper trapezius (acromial)		Longitudinal	10.1	1.8	2.8	0.6	3.5	0.7
Middle trapezius		Longitudinal	10.4	1.8	2.4	0.7	2.9	0.7
Lower trapezius		Longitudinal	14.8	2.1	3.2	0.8	2.7	0.7
Levator scapulae		Longitudinal	15.3	1.8	2.1	0.7	1.7	0.6
Rhomboidei		Longitudinal	12.5	2.0	4.0	0.7	4.0	0.7
Serratus anterior (upper part)		Longitudinal	11.1	1.7	3.5	0.9	3.9	0.7
Serratus anterior (lower part)		Longitudinal	17.6	2.0	7.9	1.3	5.6	1.1
Pectoralis minor		Longitudinal	13.2	1.1	2.3	0.5	2.1	0.5
Supraspinatus	B	Pennate	6.7	0.6	2.8	0.6	5.2	0.8
Subscapularis		Multipennate	7.3	0.5	8.6	1.5	14.5	2.6
Infraspinatus		Pannate	8.6	0.9	6.7	0.8	9.7	1.4
Teres minor		Longitudinal	8.8	1.5	1.8	0.5	2.6	0.8
Teres major		Longitudinal	14.9	1.9	5.1	1.0	4.3	0.8
Anterior deltoid		Longitudinal	11.5	1.6	3.2	0.7	3.4	0.7
Middle deltoid (anterior part)		Multipennate	9.2	1.0	2.2	0.5	3.0	0.9
Middle deltoid (posterior part)		Multipennate	9.0	0.9	7.8	1.1	10.8	1.3
Posterior deltoid		Longitudinal	13.9	1.6	4.1	1.4	3.7	1.4
Latissimus dorsi	C	Longitudinal	33.9	4.0	15.9	2.7	5.9	1.1
Pectoralis major (clavicular)		Longitudinal	14.5	2.1	2.7	0.9	2.3	0.5
Pectoralis major (sternal)		Longitudinal	18.8	2.2	8.0	2.9	5.4	2.4

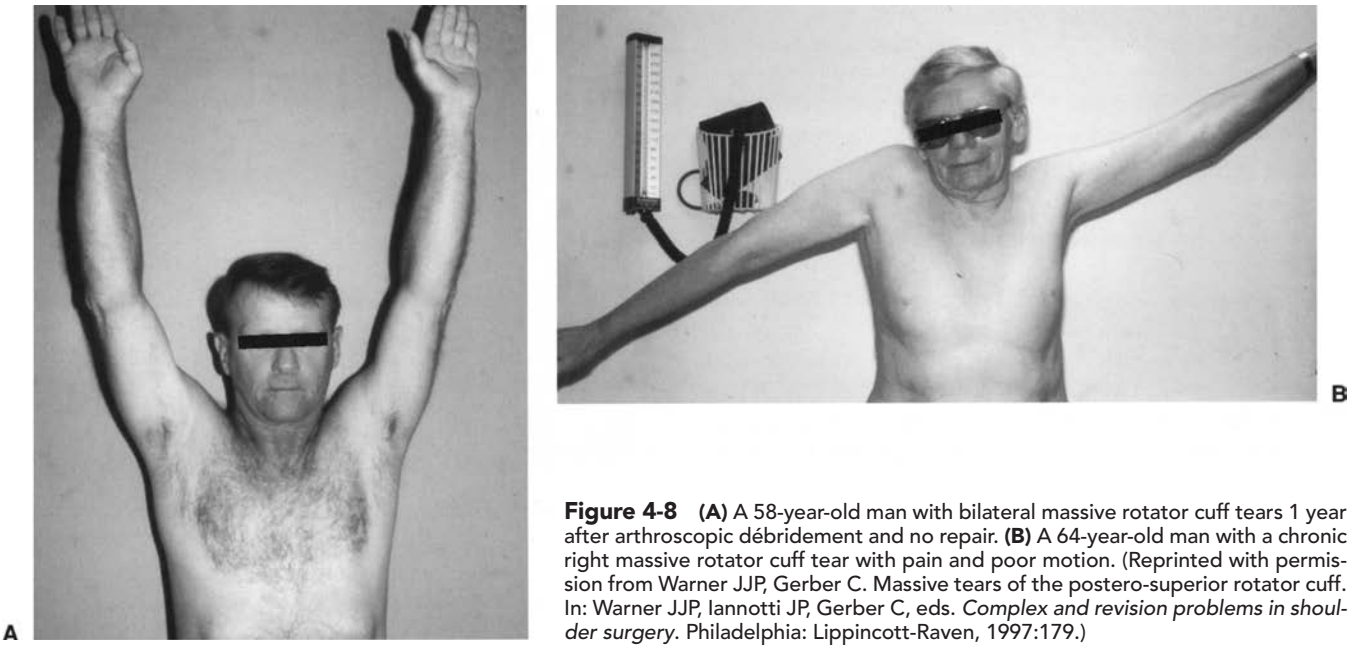


Figure 4-8 **(A)** A 58-year-old man with bilateral massive rotator cuff tears 1 year after arthroscopic débridement and no repair. **(B)** A 64-year-old man with a chronic right massive rotator cuff tear with pain and poor motion. (Reprinted with permission from Warner JJP, Gerber C. Massive tears of the postero-superior rotator cuff. In: Warner JJP, Iannotti JP, Gerber C, eds. *Complex and revision problems in shoulder surgery*. Philadelphia: Lippincott-Raven, 1997:179.)

occurs and atrophy and fatty infiltration ensue, the associated increase in interstitial connective tissue alters the structural properties of the muscle itself. In these cases, direct repair of the tendon to bone becomes impossible.[49]

In support of this conclusion is the observations of Warner and colleagues that there is a correlation between the MRI appearance of atrophy and fatty degeneration and overall shoulder function and biomechanics (Fig. 4-10).[135] Others have correlated the degree of fatty infiltration with quality of muscle and tendon tissue.[49,51,91]

The role of the long head of the biceps in superior stability of the humerus has been a subject for debate.[67,79,125,127,128,139,141,150] We agree with the observations of Walch et al.[127] that the degenerated biceps in the setting of a massive rotator cuff tear may be a significant pain generator and should be routinely tenotomized. Thus, we always perform a biceps tenotomy or tenodesis if the patient has any symptoms of biceps irritation or if there is significant degeneration observed in the tendon at the time of surgery.

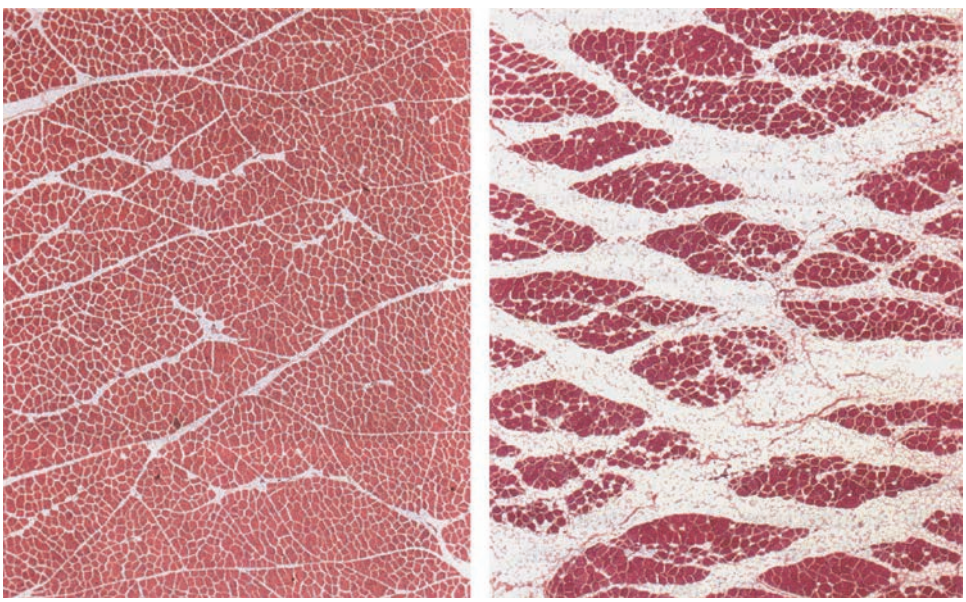


Figure 4-9 Cross-sectional (H&E staining, 16× magnification) through intact (left side) and fatty infiltrated (right side) sheep infraspinatus muscle. On the right, fat and fibrous tissue (white) has infiltrated the free space in between individual muscle fibers following tendon retraction and muscle atrophy in an experimental model of chronic rotator cuff tear. (Reproduced with permission from Meyer DC, Hoppeler H, von Rechenberg B, Gerber C. A pathomechanical concept explains muscle loss and fatty muscular changes following surgical tendon release. *J Orthop Res* 2004;22:1004–1007.)

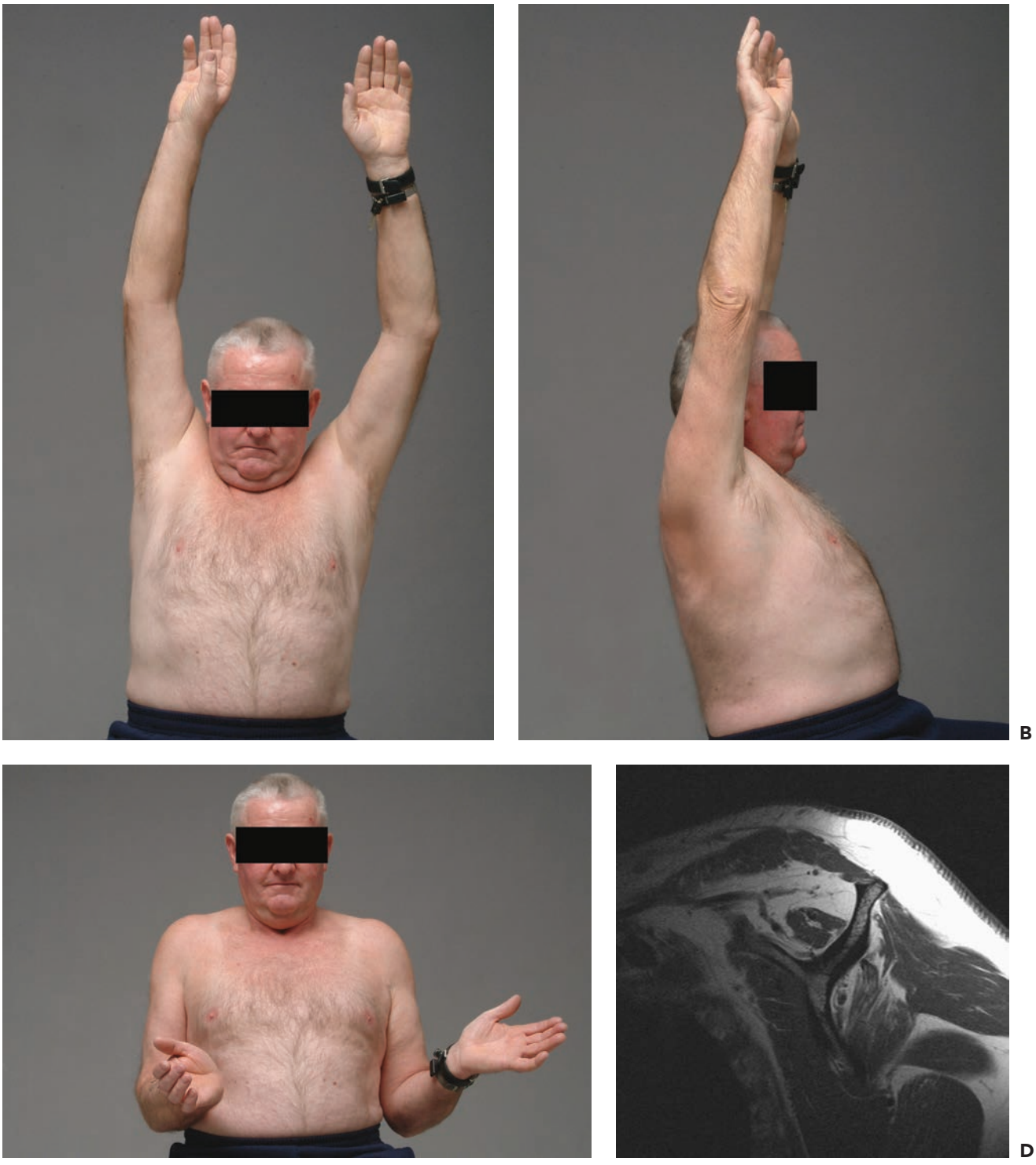


Figure 4-10 **(A,B)** A 72-year-old man with a massive, irreparable posterosuperior rotator cuff tear is able to maintain good flexion due to maintenance of a good anterior–posterior force couple and a well-functioning deltoid. **(C)** However, he displays a significant loss of active external rotation. **(D)** Oblique sagittal plane magnetic resonance imaging demonstrates severe fatty degeneration of the supraspinatus and infraspinatus muscles.

THE MECHANICAL BASIS OF TENDON TRANSFER FOR RECONSTRUCTION OF THE ROTATOR CUFF

The requirements for safe and effective tendon transfer for rotator cuff deficiency have been described by several investigators.[11,65,117] First, two types of muscle have to be distinguished if tendon transfer surgery is to be considered. One type consists of short, strong muscles with short amplitudes that are less able to generate tension; the other consists of relatively long muscles with large amplitudes that are more able to generate tension. Shiino,[117] Herzberg et al.,[65] and Wang et al.[129] emphasized the anatomic and physiologic requirements for muscle transfer including a constant vascular pedicle, adequate muscle length, sufficient cross-sectional area, and an acceptable deficit following harvest. These data are extremely useful to define optimal transfer for compensation of a functional deficit of the rotator cuff (Fig. 4-11).

The subscapularis functions as an intrinsic internal rotator and head depressor. It provides a posteriorly directed force on the humeral head while internally rotating and abducting the humerus. Herzberg described in elegant studies that the subscapularis and infraspinatus act in synergy to facilitate abduction and active elevation, whereas the subscapularis in isolation dramatically inhibits these motions.[17,65]

The supraspinatus is a relatively weak muscle whose contribution to shoulder motion decreases with increasing abduction (14% overall).[5,75] Again, the tendon excursion of the deltoid is much greater and responsible for a larger proportion of simple plane abduction.[45]

A series of recent biomechanical studies have attempted to determine why certain tendon transfers are mechanically more effective than others for the treatment of massive rotator cuff tears. In a simulated biomechanical model, Magermans and coworkers compared the effectiveness of latissimus dorsi, teres major, or a combination of these two units to the insertions of either teres minor, infraspinatus, supraspinatus, or subscapularis.[86] They concluded that a transfer of the teres major to the supraspinatus insertion produced the best functional outcome with respect to moment arm, muscle length, and muscle force. They suggested that this difference is largely attributed to the greater physiologic cross-sectional area of the teres major compared to the latissimus dorsi muscle (6.08 cm^2 vs. 5.62 cm^2, respectively), relative muscle length (86.4% vs. 80.5%), and moment arm (2.0 cm vs. 1.8 cm).[94] Our practical experience, however, has favored the latissimus transfer because the teres major is often bulky and has an extremely short tendon that is difficult to grasp, prone to pullout, and often does not reach farther than the infraspinatus insertion once it is mobilized. It needs to be further recognized that both the latissimus dorsi and teres major are internal rotators of the humeral head and are normally antagonistic to the external rotation function of the posterior rotator cuff.

INDICATIONS FOR TENDON TRANSFER

Posterosuperior Tears

The indications for tendon transfer for posterosuperior defects are summarized in Table 4-4. The indications for tendon transfer have been established based on a number of clinical studies and clinical observations.[2,43,52,94] In general, these procedures are indicated in individuals who have refractory pain and weakness, but an otherwise normal joint, in the setting of an irreparable rotator cuff tear.

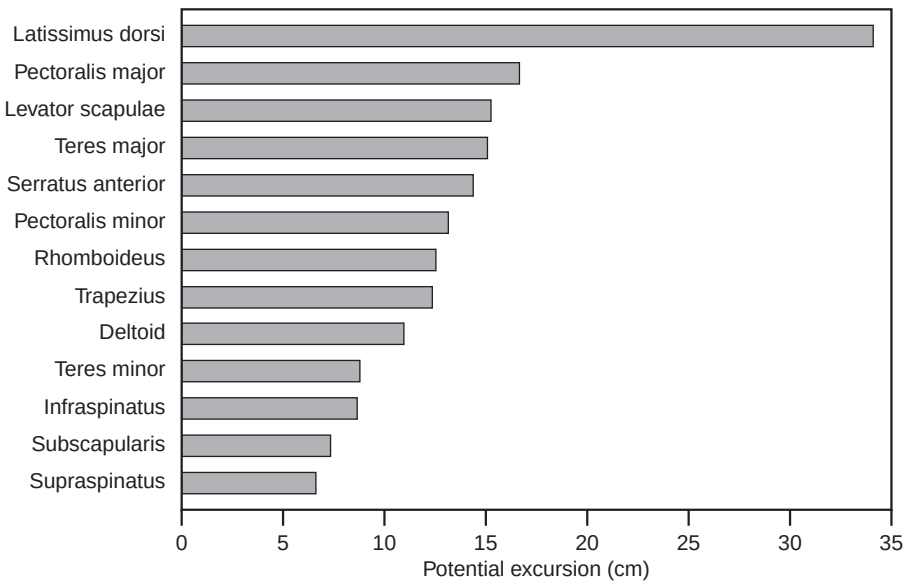


Figure 4-11 Distribution of potential excursion in 13 shoulder girdle muscles studied. Each muscle is presented as a whole without division into bellies. (Reproduced with permission from Herzberg G, Urien JP, Dimnet J. Potential excursion and relative tension of muscles in the shoulder girdle: relevance to tendon transfers. *J Shoulder Elbow Surg* 1999;8 :430–437.)

TABLE 4-4
SURGICAL INDICATIONS FOR LATISSIMUS DORSI TRANSFER IN POSTEROSUPERIOR DEFECTS

- Chronic supraspinatus/infraspinatus and/or teres minor tear or failed prior repair with retraction that is not amenable to surgical mobilization
- Isolated supraspinatus/infraspinatus and/or teres minor tear that is mobile but has a high likelihood of failure (fatty degeneration III or IV, acromiohumeral distance <5 mm on true anteroposterior radiograph)
- No evidence of static posterior subluxation on the axillary lateral radiograph
- Fatty degeneration of the supraspinatus or infraspinatus muscles of grade III or IV on magnetic resonance imaging or computed tomography scan

In the case of a posterosuperior tear that is irreparable, the degree of weakness is crucial in the decision of whether a tendon transfer is an appropriate treatment. If the patient has mild to moderate weakness, a latissimus transfer can be expected to give sufficient force to elevate the arm against gravity above shoulder level. We determine this by assisting the patient in forward flexion. If the degree of assistance required to elevate the arm overhead is minimal, then a latissimus transfer is indicated. If, however, the patient requires a great deal of assistance to raise the arm, and when the examiner releases the arm the patient cannot maintain it overhead, then a latissimus transfer is not likely to effectively restore the patient's ability to raise the arm overhead. We describe this patient as having a pseudoparalysis (see Fig. 4-1).

Contraindications to latissimus dorsi tendon transfer include associated anterosuperior tears that involve the subscapularis, cases of static anterior subluxation, advanced arthritis, or in the setting of axillary nerve injury or infection.

Anterosuperior Tears

The indications for pectoralis major transfer in anterosuperior defects are summarized in Table 4-5. In the case of an anterosuperior tear that is irreparable, many patients are able to raise their arm overhead, and certainly in this circumstance a pectoralis major transfer is indicated. If, however, there is static anterosuperior subluxation and inability of the patient to raise his or her arm overhead, a pectoralis major transfer will not restore effective elevation of the arm and the patient will probably remain symptomatic. In these cases, a reverse shoulder prosthesis may be the only option for restoration of function and alleviation of pain.

Of course, all considerations for treatment must be catered to an individual patient's disability and expectations for pain relief and functional recovery. Comorbid conditions including diabetes, cardiovascular disease, pulmonary disease, uncontrolled seizure disorder, or immunosuppression may have a negative impact on the patient's potential for recovery and ability to adhere to a rigorous postoperative rehabilitation regimen.

Contraindications to pectoralis major transfer in our experience exist in the setting of associated irreparable posterosuperior tears, cases of static superior subluxation, advanced osteoarthritis, or ongoing infection or axillary nerve injury. These contraindications are not yet clearly substantiated in the literature.

TABLE 4-5
SURGICAL INDICATIONS FOR PECTORALIS MAJOR TRANSFER IN ANTEROSUPERIOR DEFECTS

- Chronic subscapularis tear or failed subscapularis repair
- Isolated subscapularis tear or combined subscapularis/supraspinatus tear if the supraspinatus is reparable (mobile, fatty degeneration II or less, acromiohumeral distance >5 mm on true anteroposterior radiograph)
- No evidence of static anterior subluxation on the axillary lateral radiograph
- Subscapularis tear with retraction to the glenoid on preoperative imaging that is not mobile to the anatomic footprint at the time of surgery despite surgical releases
- Fatty degeneration of the subscapularis muscle of grade III or IV on magnetic resonance imaging or computed tomography scan

The important factors in the history and clinical examination that enable the surgeon to make the diagnosis of an anterosuperior, posterosuperior, or combined anteroposterosuperior rotator cuff tear are discussed in Chapter 2 and will not be described in this chapter.

Radiographic evaluation in all patients consists of a true anteroposterior plain radiograph with the arm in neutral rotation. This enables the assessment of the acromiohumeral distance (Fig. 4-12). Even more accurate is the LeClerq view, which is performed with resisted abduction.[8] The axillary lateral radiograph allows one to determine the presence of static anterior subluxation and the stage of glenohumeral arthritis (Fig. 4-12).[114] Further imaging includes either a CT or an MRI with intraarticular contrast. MRI is the currently preferred method for assessment of tear quality, size, and degree of retraction, fatty degeneration, and muscle atrophy.[33,55]

SURGICAL TREATMENT OF ANTEROSUPERIOR DEFECTS

Historical treatment options for management of irreparable tears of the subscapularis have consisted of transfer of the acromial portion of the trapezius, the pectoralis minor, and the pectoralis major. Relevant to the success of these transfers is an understanding of their amplitudes and relative strengths as described earlier.

Despite the poor amplitude and strength characteristics of the trapezius, Patte et al.,[106] Patte and Debeyre,[105] Goutallier et al.,[54] and Yamanaka and Mikasa[140] reported good results utilizing this transfer for isolated insufficiency of the subscapularis. The tendon of the acromial portion of the trapezius is released with a small bony fragment, the musculotendinous unit is mobilized, the distal clavicle is resected, and the transfer is performed through the acromioclavicular joint to the lesser tuberosity (Fig. 4-13). Goutallier et al. reported their results in 25 cases as good to excellent pain relief and "acceptable" functional improvement. Unfortunately, absolute recovery of function, strength, and range of motion was not reported.

Recent experience by Resch[111,112] and others[36,69,70,73,77] has demonstrated that the pectoralis major appears to be the most reliable alternative for managing an irreparable tear of the subscapularis. Our own experience[42,44–47,70,73,93,111,112,131,133] supports this conclusion. The biomechanics of this transfer, however, are not ideal as the pectoralis major, once transferred, is unable to recreate the vector of the subscapularis, which produces a posteriorly directed force on the humeral head. Presently, there is no transfer that restores the strength and amplitude of the subscapularis optimally, although surgical techniques that route the tendon underneath the conjoined tendon have attempted to mimic the true course and force vector of the native musculotendinous unit.[36,77,78,111,112]

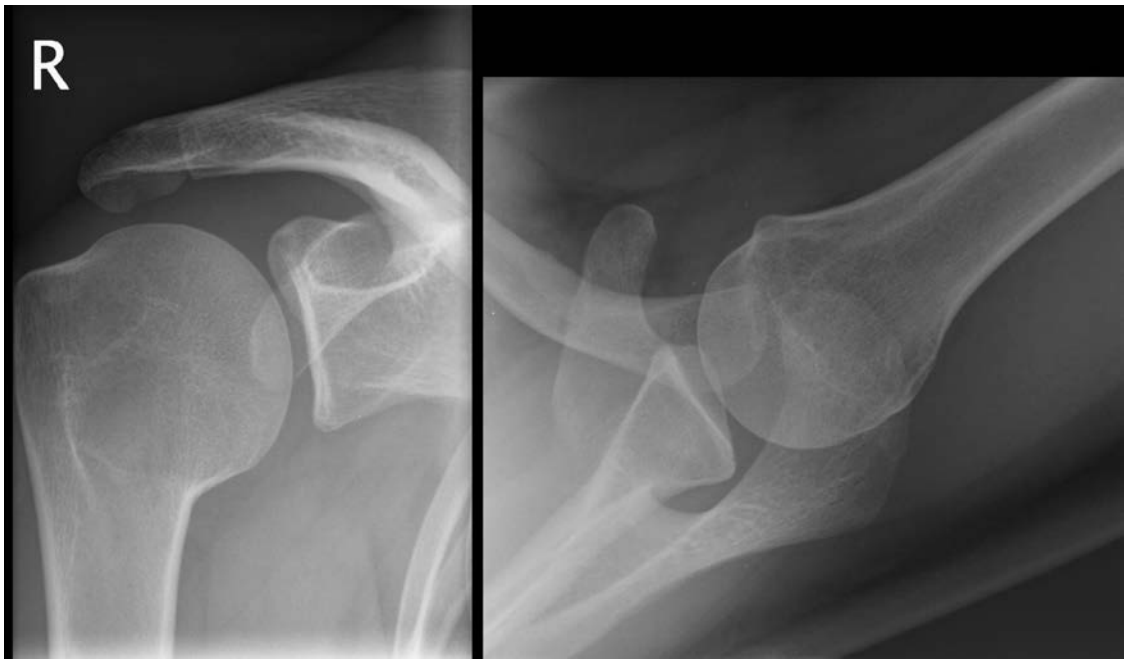


Figure 4-12 True anteroposterior (AP) and axillary lateral radiograph of the glenohumeral joint. The true AP view of the glenohumeral joint allows for assessment of the acromiohumeral distance and static superior subluxation, while the axillary lateral view is used to assess for static anterior or posterior subluxation.

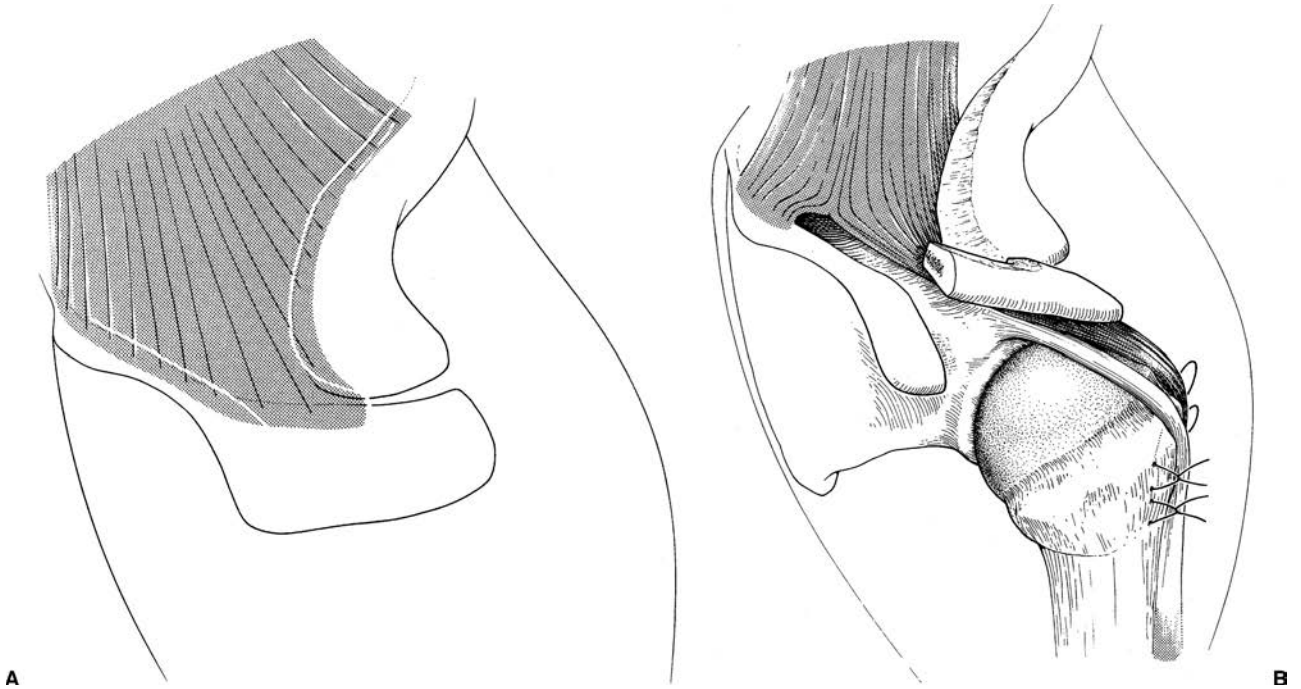


Figure 4-13 Technique of trapezius transfer for subscapularis deficiency. **(A)** The trapezius is elevated with a piece of the underlying acromion. **(B)** After elevation of the musculotendinous unit, it is either passed through the enlarged acromioclavicular joint or through the split acromion and reinserted at the lesser tuberosity. If a frontal split of the acromion has been used, this is closed with simple transosseous sutures.

Results of Pectoralis Major Transfer

Wirth and Rockwood were the first to describe the technique of pectoralis major transfer for irreparable tears of the subscapularis.[138] They reported on 13 shoulders found to have irreparable injury to the subscapularis at the time of surgery for recurrent anterior instability. Seven patients were treated with pectoralis major transfer, five with transfer of the pectoralis minor, and one with transfer of both muscles. They noted satisfactory results in 10 patients, all of whom displayed active contraction of the muscle transfer, and unsatisfactory results in three patients at a mean follow-up of 5 years after surgery.

The pectoralis major transfer has also been reported by others[24,45,93,111,112,133] in the setting of an irreparable anterosuperior rotator cuff tear with associated advanced tissue degeneration and fatty infiltration.[28,34,46,55] Gerber reported on a series of 28 patients with chronic irreparable subscapularis tears treated with pectoralis major transfer.[73] At an average 32-month follow-up, the mean relative Constant scores increased from 47% to 70%, with statistically significant improvements in pain, active elevation, and abduction strength. The outcomes were clearly less favorable if the subscapularis tear was associated with a concomitant irreparable supraspinatus tear (Constant score 49% vs. 79%). In addition, patients having undergone pectoralis major transfer were clearly inferior to those following direct repair of reparable subscapularis tears.

Resch reported on his experience using a modification of the procedure described above in which he transfers the superior two-thirds of the pectoralis muscle underneath the conjoined tendon (Fig. 4-14).[112] This modification was made in an effort to reproduce the normal course of the subscapularis tendon beneath the short head of the biceps and coracobrachialis muscle. Four of the 12 patients had concomitant tearing of the supraspinatus muscle. At an average follow-up of 28 months, 9 of 12 patients were graded as excellent/good, 3 as fair, and none as poor. All patients reported pain improvement, and the average Constant scores increased from 20 to 63.[112]

Warner and colleagues reported on another modification of the procedure, which involves transfer of the inferior, sternal portion of the pectoralis major tendon beneath the clavicular head of the muscle and superficial to the conjoined tendon (Fig. 4-15).[42,93] They felt that this would help avoid the risk of injury to the musculocutaneous nerve that can occur with transfer underneath the conjoined tendon. Furthermore, they also observed that in some cases of severe scarring after prior surgery, safe dissection underneath the conjoined tendon was problematic. They reported on 20 cases, 9 of which were combined transfer of the teres major and pectoralis major. The rationale for use of the teres major was that it would provide a greater stabilizing force than the sternal head of the pectoralis major alone. The combined transfer was used for

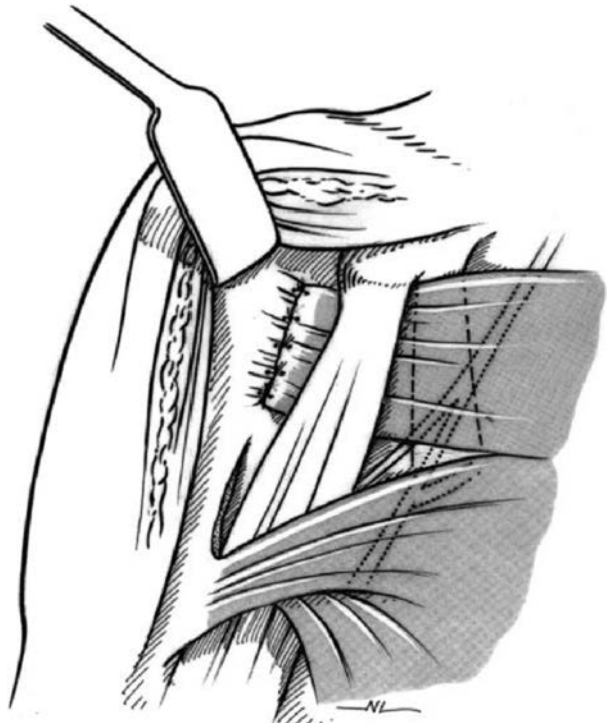


Figure 4-14 Technique of subcoracoid pectoralis major transfer of the superior half of the tendon to the lesser tuberosity for an isolated rupture of the subscapularis. (Reproduced with permission from Resch H, Povacz P, Ritter E, Aschauer E. Pectoralis major tendon transfer for irreparable rupture of the subscapularis and supraspinatus tendon. *Tech Shoulder Elbow Surg* 2002 ;3:167–173.)

patients with complete absence of the subscapularis tendon, whereas the isolated sternal head of the pectoralis was used for patients where the lower muscle fibers of the subscapularis remained intact. For the 11 patients that underwent isolated transfer of the sternal head of the pectoralis major, the mean American Shoulder and Elbow Surgeons (ASES) score improved from 42 to 61 points at an average follow-up of 38 months postoperatively. For patients who underwent the combined pectoralis/teres major transfer for a complete irreparable tear, the mean ASES score increased from 34 to 55 points. The majority of patients in both groups noted substantial pain relief.[42]

Other anatomic and cadaveric studies have attempted to determine the optimal route of transfer for the pectoralis major tendon to optimize the force vector.[36,77,78] Yamaguchi and colleagues recommended dissection of the musculocutaneous nerve from the overlying conjoined tendon and passage of the tendon of the pectoralis major beneath the conjoined tendon but superficial to the nerve. The decision to transfer the superior one-half or the entire tendon is based on the degree of subscapularis atrophy and the age of the patient, with elderly patients and those with significant atrophy generally undergoing transfer of the entire tendon (Fig. 4-16).[77,78] The transfer is attached to the greater tuberosity of the humerus. Two of their 14 patients who underwent this procedure suffered transient musculocutaneous neurapraxia. Nine of their 14 patients were satisfied with the procedure, with significant improvements in pain scores (visual analog scale [VAS] score from 7 to 3) and active flexion (28 to 61 degrees).[77,78] Clinical studies have failed to demonstrate a clear advantage among these different modifications.

SURGICAL TREATMENT OF POSTEROSUPERIOR DEFECTS

An isolated supraspinatus tear is rarely irreparable. Herzberg et al.[65] showed that isolated contraction of the supraspinatus leads to anterolateral elevation of approximately 60 degrees and external rotation of 20 degrees. Furthermore, its efficiency is substantially greater when it is activated simultaneously with the lateral deltoid.

The supraspinatus advancement, first described by Debeyre et al.,[26] is a technique that is more of a muscle "slide" rather than a transfer. Nonetheless, it has been deemed effective in the treatment of isolated irreparable tears of the supraspinatus. It is performed by releasing the muscle belly of the supraspinatus to allow lateral advancement of the tendon, being cognizant to avoid injury to the suprascapular neurovascular pedicle.[136] Although their success led them to extend this methodology to the treatment of the rare irreparable infraspinatus tear,[104,107] this technique remains largely historical, as others have not been able to validate its usefulness.

Cofield described a partial subscapularis transfer that utilizes the superior two-thirds of the tendon to replace an irreparable supraspinatus tear.[23] This rotational flap has been effective in his hands for medium-sized tears,[23] but unfortunately, much less effective in the treatment of massive tears.[74] Karas and Giachello showed that patients who undergo transfer of the subscapularis for an irreparable supraspinatus tear actually had worse function after the procedure.[74] It is our experience that the function of the subscapularis is of critical importance to overall shoulder function such that transfer of the superior two-thirds, which is usually inherently partially injured, is usually not advised. This concept, to protect and preserve the subscapularis, is supported by clinical and basic science observations that patients with massive tendon tears but a preserved anterior–posterior force couple can still elevate their arms and maintain the humeral head centered on the glenoid.[15–19,122,123]

Takagishi[121] described the trapezius transfer for isolated irreparable tears of the supraspinatus, which appears structurally well suited based on amplitude and strength. The technique involves transfer of the acromial portion of the tendon to the greater tuberosity. In a slight modification, Yamanaka and Mikasa[140] used an acromial-split technique rather than a distal clavicle resection and reported on seven cases with no excellent results.

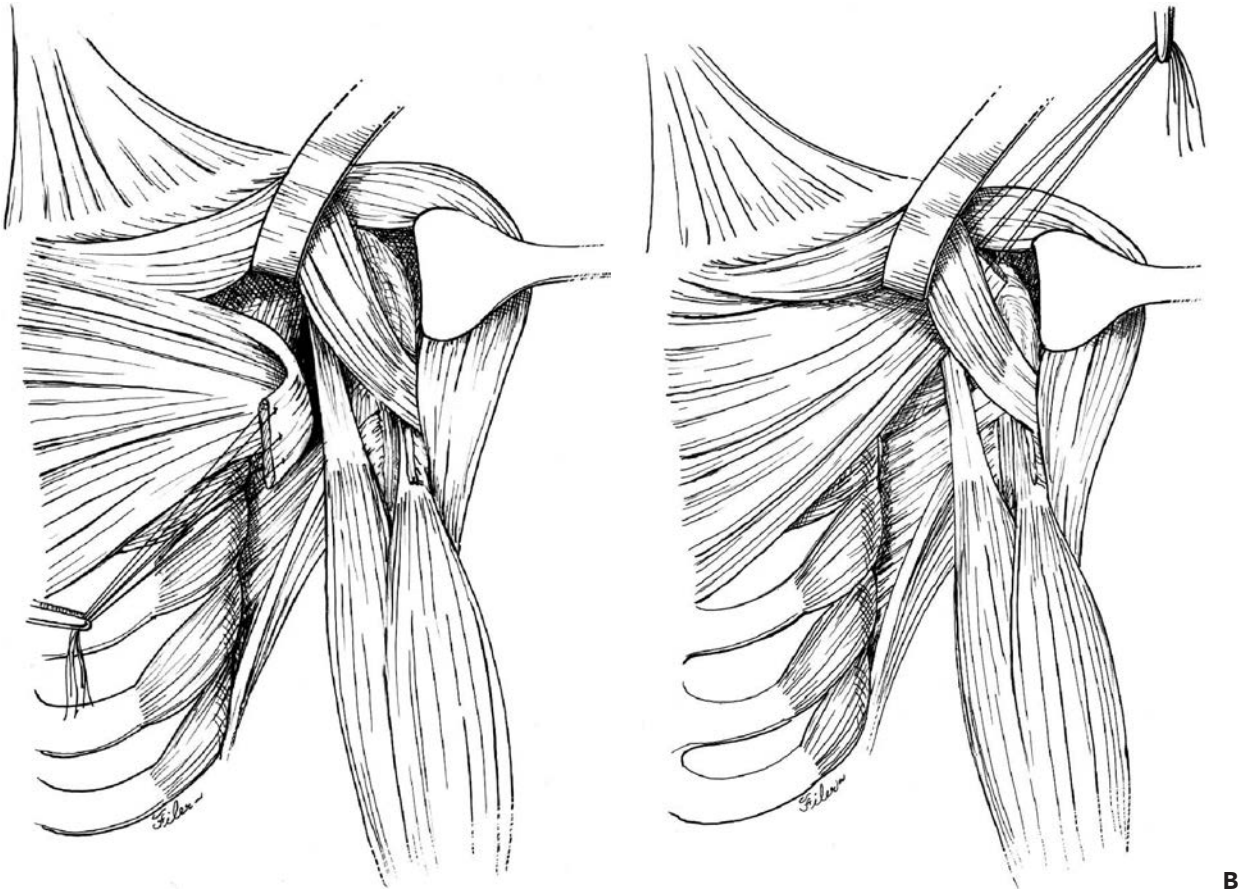


Figure 4-15 The sternal head of the pectoralis major tendon is **(A)** detached and **(B)** passed beneath the clavicular head prior to transfer to the lesser tuberosity. (Reprinted with permission from Gerber A, Clavert P, Millett PJ, Holovacs TF, Warner JJP. Split pectoralis major transfer and teres major tendon transfers for reconstruction of irreparable tears of the subscapularis. *Tech Shoulder Elbow Surg* 2004;5:5–12.)

Apoil and Augereau[3] recommended the lateral deltoid transfer based on the original description by Takagishi[121] (Fig. 4-17). This technique involves the release of an approximately 2.5-cm-wide strip of the anterolateral deltoid from the acromion, which is transferred to the supraspinatus stump or the superior aspect of the glenoid. They noted excellent results, which have been duplicated by Gazielly in 25% of his series of 20 patients who underwent this procedure.[40] A prospective study by Le Huec et al. yielded satisfactory pain relief and high patient satisfaction, but no gain in strength.[80] More recently, Gedouin et al. reported on 41 patients with a minimum 5-year follow-up who underwent the deltoid transfer for irreparable rotator cuff tears.[41] In his series, 92% of patients were satisfied with the procedure, and average Constant scores improved by 25 points (from 37 to 62 points).[41] Active flexion improved from 113 to 148 degrees with associated improvement in strength (flexion force from 1.3 to 2.9 kg).[41] Despite the experience of these surgeons, our concern is that the critical importance of a functional deltoid, especially if further treatment of rotator cuff arthropathy is undertaken using the reverse shoulder prosthesis, obviates the perceived advantage of a deltoid transfer (Fig. 4-18).

The infraspinatus is rarely irreparable in isolated cases as it usually exists in combination with an irreparable supraspinatus. The infraspinatus functions in active elevation as well as external rotation. The infraspinatus advancement has been described earlier and has provided favorable results for some surgeons (Fig. 4-19). However, this has not been reproduced in the hands of others.

Based on the work of Herzberg et al.,[65] the latissimus dorsi possesses the proper structural and physiologic characteristics for treatment of infraspinatus tears in isolation or with coexistent supraspinatus tears, and thus it has been our preferred method of treatment in such cases for the past 15 years. In his studies of the mechanical effects of latissimus transfer to multiple areas, Herzberg also confirmed that the ideal insertion site is at the tip of the greater tuberosity or at the supraspinatus footprint.

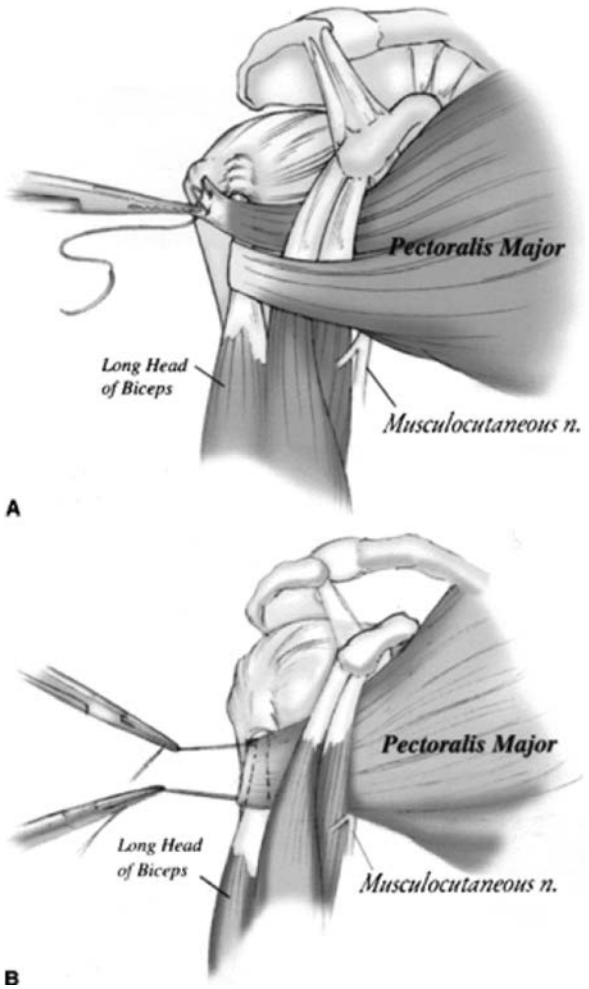


Figure 4-16 Subcoracoid pectoralis major transfer superficial to the musculocutaneous nerve. **(A)** Split transfer. **(B)** Complete transfer. (Reprinted with permission from Klepps S, Galatz L, Yamaguchi K. Subcoracoid pectoralis major transfer: a salvage procedure for irreparable subscapularis deficiency. *Tech Shoulder Elbow Surg* 2001;2:85–91.)

Results of Muscle Transfer for Posterosuperior Defects

Gerber et al. were the first to report on successful management of massive irreparable posterosuperior tears using a latissimus dorsi tendon transfer in 1988.[52] He noted that these patients gained an average of 50 degrees of active elevation and 13 degrees of active external rotation following this procedure. He demonstrated that preexisting injury of the subscapularis and inability to repair that tendon was associated with poor recovery of function.[43,44]

Recently, Gerber et al. reviewed their mid- to long-term clinical and radiographic outcome of patients who underwent latissimus dorsi transfer at an average follow-up of 53 months (range 24 to 126 months).[48] Thirteen of these patients had a deficient subscapularis. The age- and gender-matched Constant score improved from 55% to 73%, with significant improvements in pain, flexion, external rotation, and strength in abduction. It was again noted that patients with a dysfunctional subscapularis did significantly more poorly in all parameters tested.

One explanation for inferior results in combined anterosuperior defects has been proposed by Burkhart, who emphasized the importance of a balanced force couple and fulcrum in the shoulder.[15,17] Others have confirmed this concept in biomechanical studies.[85,122]

The effectiveness of latissimus dorsi transfer has also been compared in primary versus revision surgery. Warner and Parsons[134] found significantly higher functional gains postoperatively in primary surgery compared to revision surgery. At a mean follow-up of 19 months, patients who underwent latissimus dorsi transfer as a salvage procedure had a higher rate of rupture of the transfer (44% vs. 17%) as well as significantly worse Constant scores (55% vs. 70%).[134]

Aoki and coworkers[2] reported on 12 cases of latissimus dorsi transfer for irreparable rotator cuff tears at an average follow-up of 3 years. They noted excellent results in 4 of 12, good results in 4 of 12, and poor results in 3 of 12, and were able to correlate electromyelography (EMG) activity of the muscle during active motion in 9 of 12 cases.[2] Active flexion improved from 99 to 135 degrees on average, with associated improvements in strength in external rotation. They concluded this technique to be an effective salvage procedure in restoring function and decreasing pain in irreparable tears.

Irlenbusch and colleagues performed a latissimus dorsi transfer in 22 patients, 7 of whom were for failed prior open rotator cuff repair surgeries at an average 9 months follow-up.[66] Average Constant scores improved from 43 to 67 versus 33 to 62 in revision cases. Poor results were associated with concomitant rupture of the subscapularis or deltoid insufficiency. Pain improved significantly in all patients.

Celli et al. reported on the results of isolated teres major transfer for irreparable infraspinatus tears.[21] Although isolated tears of the infraspinatus are exceedingly rare, they reported on six patients, all of whom were satisfied with the procedure. Average Constant scores improved from 40 to 62, and active external rotation improved by 35 degrees in abduction and 25 degrees in adduction.

Malkani and colleagues recently reported on transfer of the long head of the triceps in the treatment of irreparable tears of the supraspinatus and infraspinatus.[87] In a review of 19 shoulders in 18 patients who underwent this surgical procedure at a minimum of 2 years follow-up, 100% of patients were satisfied with their outcome, with average overall improvement in their University of California Los Angeles (UCLA) score from 9.7 to 28.8.

SURGICAL TREATMENT OF COMBINED ANTERO- AND POSTEROSUPERIOR DEFECTS

The only report in the literature of combined latissimus and pectoralis major transfer for anteroposterosuperior

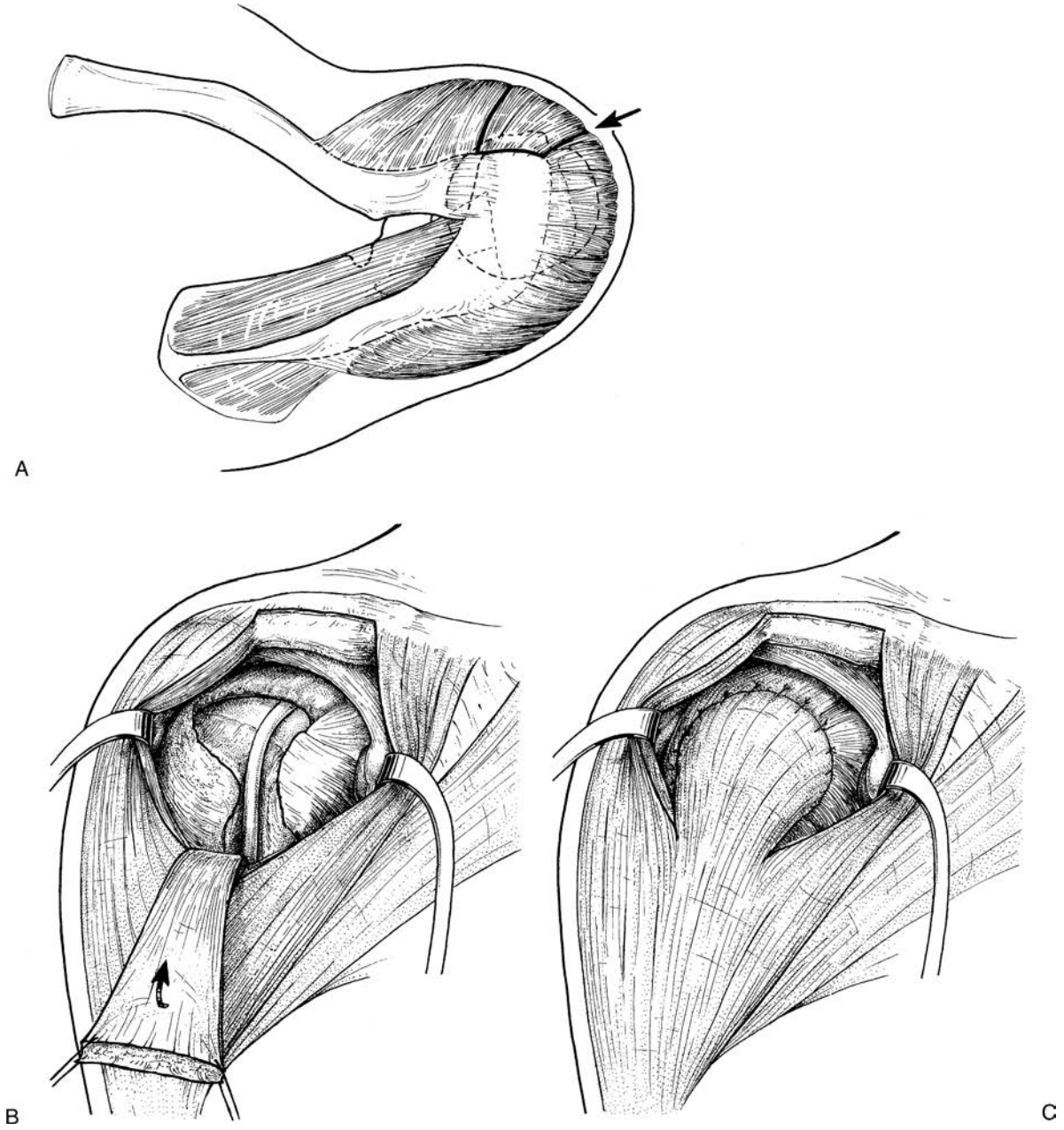


Figure 4-17 Deltoid flap technique. **(A)** An anterolateral strip of approximately 2.5 cm is tailored through a superolateral approach. **(B)** The tear is identified and débrided. **(C)** The deltoid flap is sutured to the stump of the supraspinatus, infraspinatus, subscapularis, or superior labrum depending on the pathology that is present.

rotator cuff defects was presented by Aldridge and colleagues.[1] They retrospectively reported on 11 consecutive patients treated in this fashion with a minimum 2-year follow-up. They noted an improvement in active elevation of 42 to 86 degrees and of active external rotation from 0 to 13 degrees. Five out of 11 patients showed a significant improvement in pain, strength, and function, while four showed no improvement. Standardized outcome instruments showed statistically significant improvements, with the average Constant score improving from 21 to 36, and the average UCLA score from 13 to 19.

Our experience has been that combined anterior and posterior transfers are generally not successful owing to the requirements for different postoperative therapy programs and the major weakness that cannot be restored with the mechanical advantage of these combined transfers. Thus, we do not advocate this approach.

Figure 4-18 Deltoid injury. **(A)** 52-year-old patient with right deltoid injury following open rotator cuff repair. **(B)** A 64-year-old woman who was treated with a lateral deltoid transfer for an irreparable supraspinatus tear. Her right shoulder shows a residual deltoid injury postoperatively.

TREATMENT ALGORITHM: ANTEROSUPERIOR TEARS (FIG. 4-20)

In patients with an isolated irreparable tear of the subscapularis tendon, pectoralis major transfer is likely to provide a reliable method of pain relief and functional recovery. The combination of a supraspinatus tear may also be treated with a pectoralis major transfer, with or without an associated teres major transfer, as long as there is no fixed anterosuperior displacement of the humerus from the glenoid. In patients with chronic anterosuperior tears who have static superior and anterior subluxation of the humeral head, a pectoralis major transfer will not recenter the humeral head and it will not give reliable recovery of function. In these cases a reverse shoulder prosthesis may be the only solution for restoration of function. This method of treatment is described elsewhere in this text (Chapter 23).

TREATMENT ALGORITHM: POSTEROSUPERIOR TEARS (FIG. 4-21)

In patients with an irreparable tear of the supraspinatus and infraspinatus who also have marked weakness of external rotation, a latissimus dorsi tendon transfer is a reliable method of treatment. However, the degree of weakness must not be greater than mild to moderate. Again, our method for determining this is based on the degree of assistance required to enable the patient to elevate his or her arm overhead. If the patient is completely unable to initiate any muscle force to assist the examiner in raising his or her arm, and if the arm then falls back down when the examiner removes his or her hand, the patient is considered to have a pseudoparalysis of the shoulder. In such cases, the magnitude of force provided by a latissimus dorsi that is transferred to the greater tuberosity will be insufficient to elevate the arm. In most of these patients the humeral head shows static superior displacement with an acromiohumeral distance less than 5 mm. These individuals are best managed using a reverse shoulder prosthesis.

TECHNIQUES OF MUSCLE TRANSFER

Latissimus Dorsi Transfer for Irreparable Posterosuperior Rotator Cuff Tear

Dr. Warner's Technique in Lateral Decubitus

The patient is placed into a lateral decubitus position on a long bean bag that is contoured around the patient. An articulated hydraulic arm holder (Spider Arm Positioner, Tenet Medical Engineering, Calgary, Alberta, Canada) is used on the contralateral side of the operative table, and this permits placement of the arm in the proper position for tendon harvesting as well as setting the tension of the tendon transfer during fixation. This positioning allows the surgeon to perform the procedure with one assistant provided the arm holder is available (Fig. 4-22).

An anterosuperior approach is utilized and the deltoid is split directly off the lateral acromion. The deltoid is then elevated using electrocautery in the subperiosteal plane off the anterior and posterior acromion so that this sleeve is in continuity with the deltoid and trapezius fascia. By making this vertical split directly laterally, we are able to expose the humerus from the teres minor to the subscapularis (Fig. 4-23). Furthermore, the dissection is performed parallel to the muscle fibers of the deltoid, which we believe protects the deltoid from disruption when it is repaired.

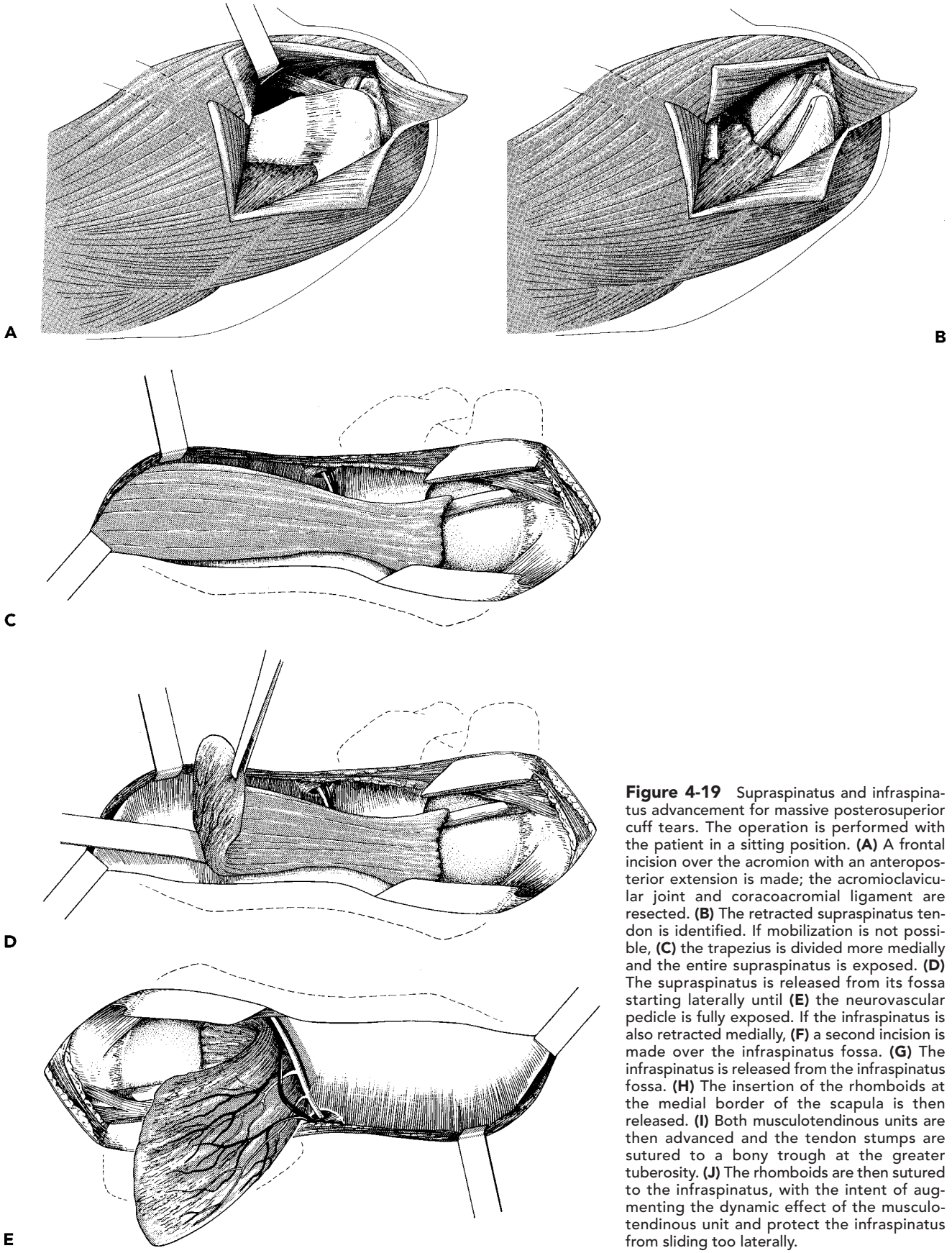


Figure 4-19 Supraspinatus and infraspinatus advancement for massive posterosuperior cuff tears. The operation is performed with the patient in a sitting position. **(A)** A frontal incision over the acromion with an anteroposterior extension is made; the acromioclavicular joint and coracoacromial ligament are resected. **(B)** The retracted supraspinatus tendon is identified. If mobilization is not possible, **(C)** the trapezius is divided more medially and the entire supraspinatus is exposed. **(D)** The supraspinatus is released from its fossa starting laterally until **(E)** the neurovascular pedicle is fully exposed. If the infraspinatus is also retracted medially, **(F)** a second incision is made over the infraspinatus fossa. **(G)** The infraspinatus is released from the infraspinatus fossa. **(H)** The insertion of the rhomboids at the medial border of the scapula is then released. **(I)** Both musculotendinous units are then advanced and the tendon stumps are sutured to a bony trough at the greater tuberosity. **(J)** The rhomboids are then sutured to the infraspinatus, with the intent of augmenting the dynamic effect of the musculotendinous unit and protect the infraspinatus from sliding too laterally.

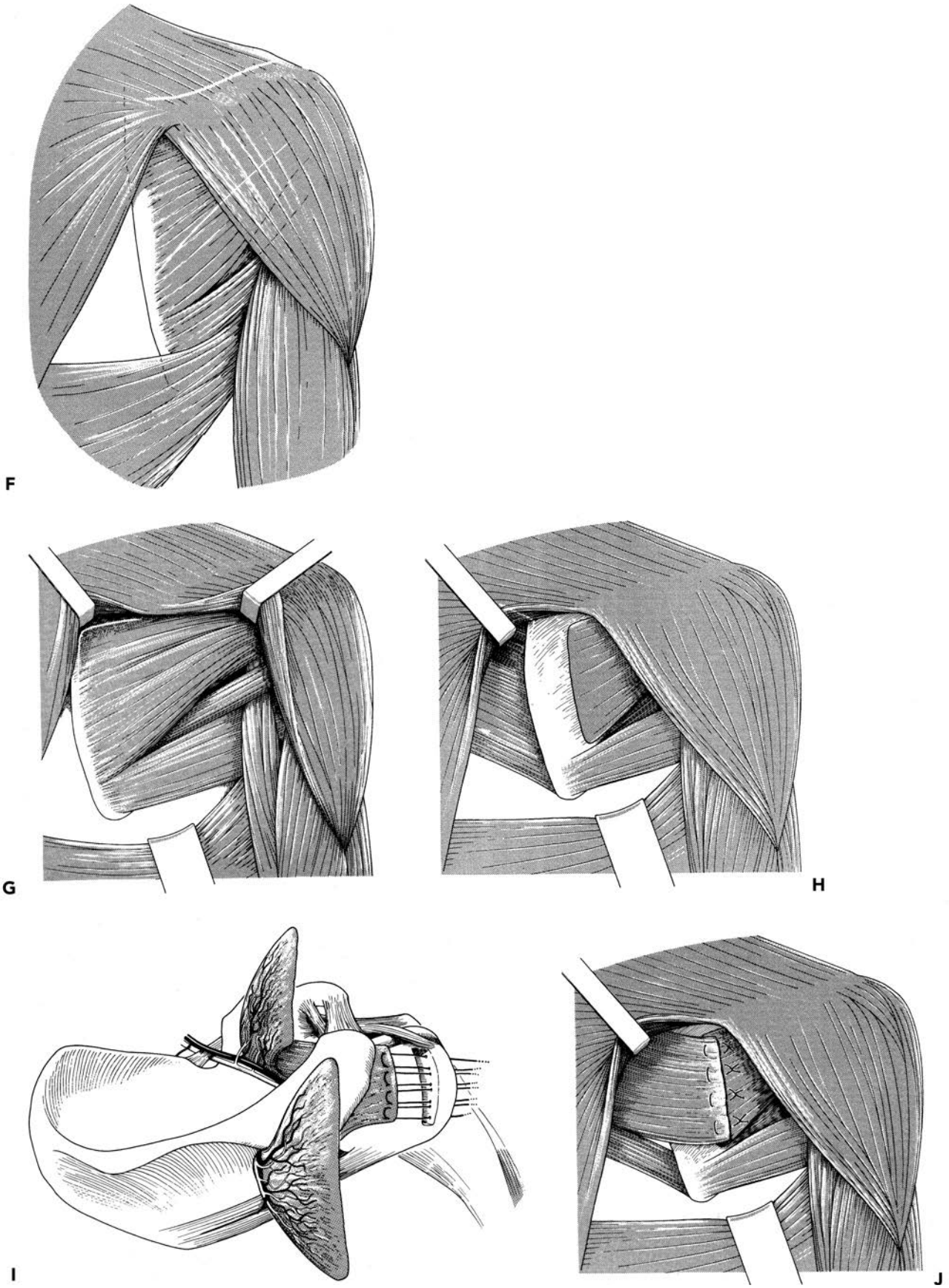


Figure 4-19 (*continued*)

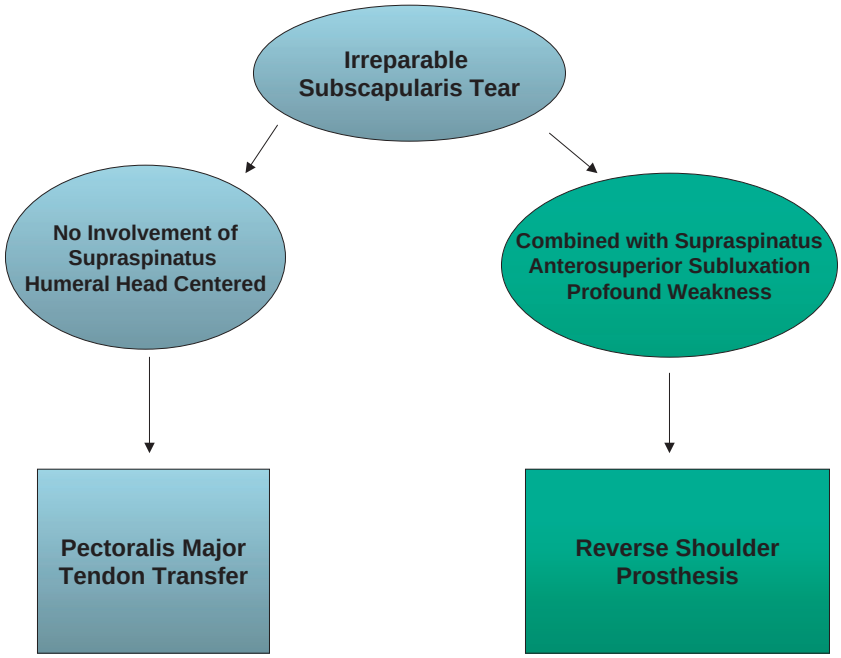


Figure 4-20 Algorithm for the treatment of anterosuperior rotator cuff tears.

If the coracoacromial ligament has not been disrupted by prior surgery, we are careful to define it and avoid its detachment as it is an important restraint to superior displacement of the humeral head in patients with a massive rotator cuff tear. More often than not, however, an acromioplasty and resection of the coracoacromial ligament has already been performed. In cases where there has been no acromioplasty, we perform a modified acromioplasty using a small saw and remove only the undersurface of the acromion from lateral to anterior, being careful not to detach the origin of the coracoacromial ligament.

Adhesions often exist between the acromion and the rotator cuff and between the rotator cuff and the deltoid. Internal rotation of the arm will expose the posterior rotator cuff, while external rotation will expose the anterior rotator cuff. Adhesions are sharply released in this interval.

If the supraspinatus and infraspinatus tendon remnants are discernible, they are mobilized as much as possible, and sutures are placed through them to be used to secure the latissimus dorsi along its medial edge following the transfer.

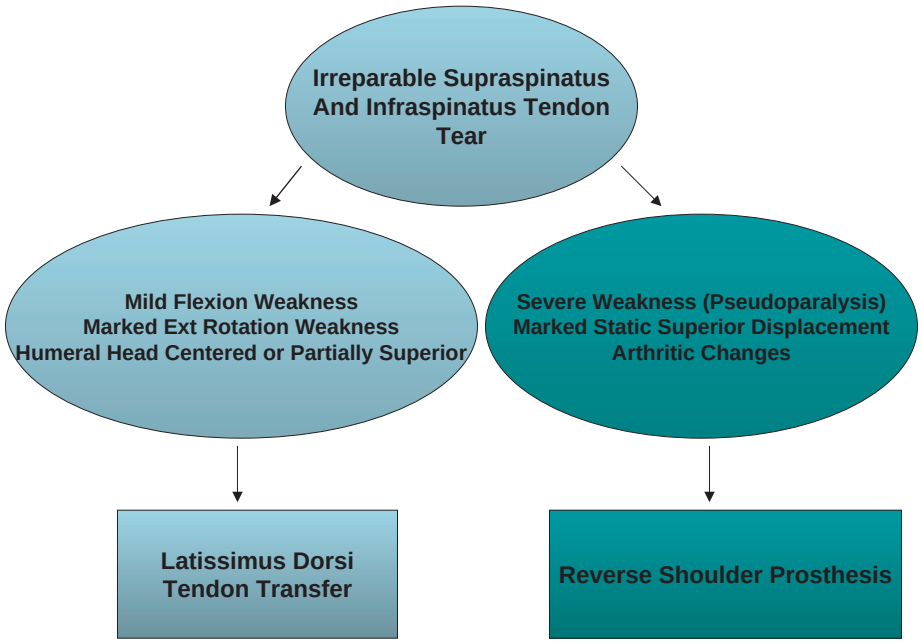


Figure 4-21 Algorithm for the treatment of posterosuperior rotator cuff tears

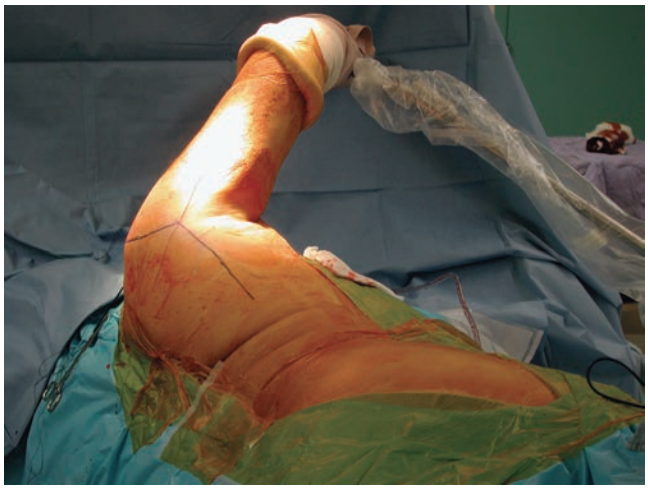


Figure 4-22 Patient positioning in the lateral decubitus position for latissimus dorsi tendon transfer using an articulated hydraulic arm holder (Spider Arm Positioner, Tenet Medical Engineering, Calgary, Alberta, Canada).

Once it is defined, the remnant of the infraspinatus or teres minor is secured with several sutures of nonabsorbable, braided material (#2 Ethibond, Ethicon, Johnson & Johnson, Westwood, MA). The upper insertion of the subscapularis is also defined and sutures are placed through it as well.

The biceps, if present, is usually degenerated in these individuals, and in such cases it is tenodesed within the bicipital groove using a bone anchor (TwinFix, Smith & Nephew Endoscopy, Andover, MA).

Next, the greater tuberosity is débrided clear of soft tissue and remnant of the stump of the torn tendon. A rongeur is used to abrade the bone, but a bone trough is not created. We prefer to use suture anchors to fix the tendon transfer, as these can be placed at points along the greater tuberosity to secure the latissimus tendon directly into the "footprint" of the supraspinatus and infraspinatus. Usually three or four screw-in anchors appropriate for tuberosity fixation are placed into the greater tuberosity.

The latissimus dorsi is harvested only after all of the above preparatory steps have been performed. Exposure of the latissimus dorsi is facilitated by placing the patient's arm into flexion, adduction, and internal rotation. The articulated hydraulic arm holder can maintain the arm in this position during tendon dissection. It is helpful at this stage of the procedure for the assistant to stand on the side opposite the surgeon, as this facilitates retraction and exposure of the tendon insertion during the dissection.

An L-shaped incision is made along the anterior belly of the latissimus muscle and then along the posterior axillary line. Curving the incision laterally at its proximal portion will make exposure of the tendon insertion easier (Fig. 4-24A). During the dissection, the muscles of the latissimus (most anterior muscle on the chest wall), teres major, long head of the triceps, and posterior deltoid are defined. The latissimus is dissected first from its attachments to the anterior chest wall, followed by identification of the interval between it and the teres major (Fig. 4-24B). This interval can be variable and difficult to define because in some patients, the two muscles may seem to be conjoined. However, careful dissection and retraction, which places each under tension, will allow clear dissection of the latissimus muscle from the teres major.

As the dissection proceeds toward the insertion of the latissimus dorsi tendon, several fascial bands may be encountered that course anteriorly. If the surgeon palpates the insertion of the tendon on the humerus, the correct plane of dissection can be clarified. Maintenance of the arm in internal rotation will make this step easier.

The tendon insertion is then isolated and the latissimus is detached from the humerus sharply. A traction suture in a whipstitch configuration facilitates dissection of the muscle toward its origin until the thoracodorsal neurovascular pedicle is identified (Fig. 4-24C). This structure courses on

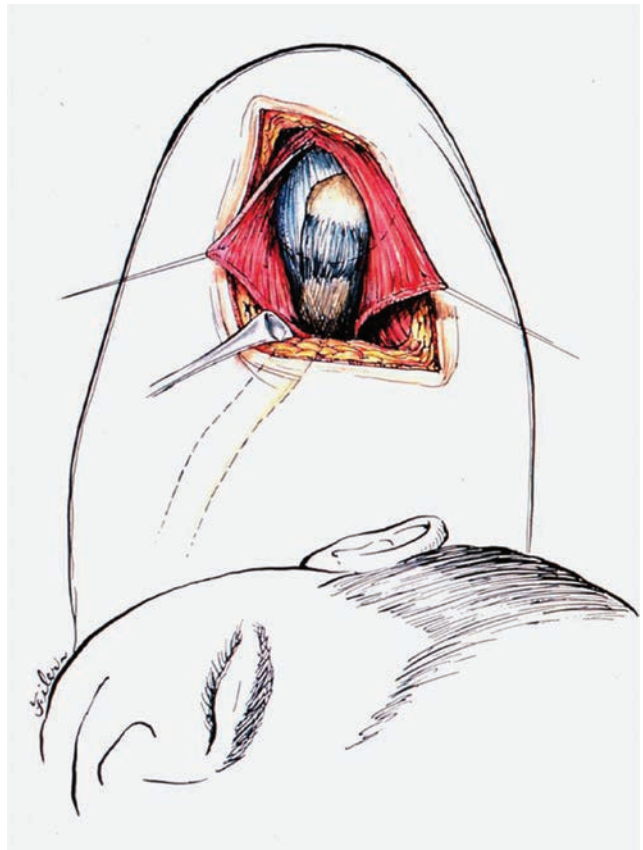


Figure 4-23 Anterosuperior approach to the rotator cuff utilizing a deltoid split directly off the lateral acromion. The deltoid is elevated using electrocautery in the subperiosteal plane off the anterior and posterior acromion so that this sleeve is in continuity with the deltoid and trapezius fascia.

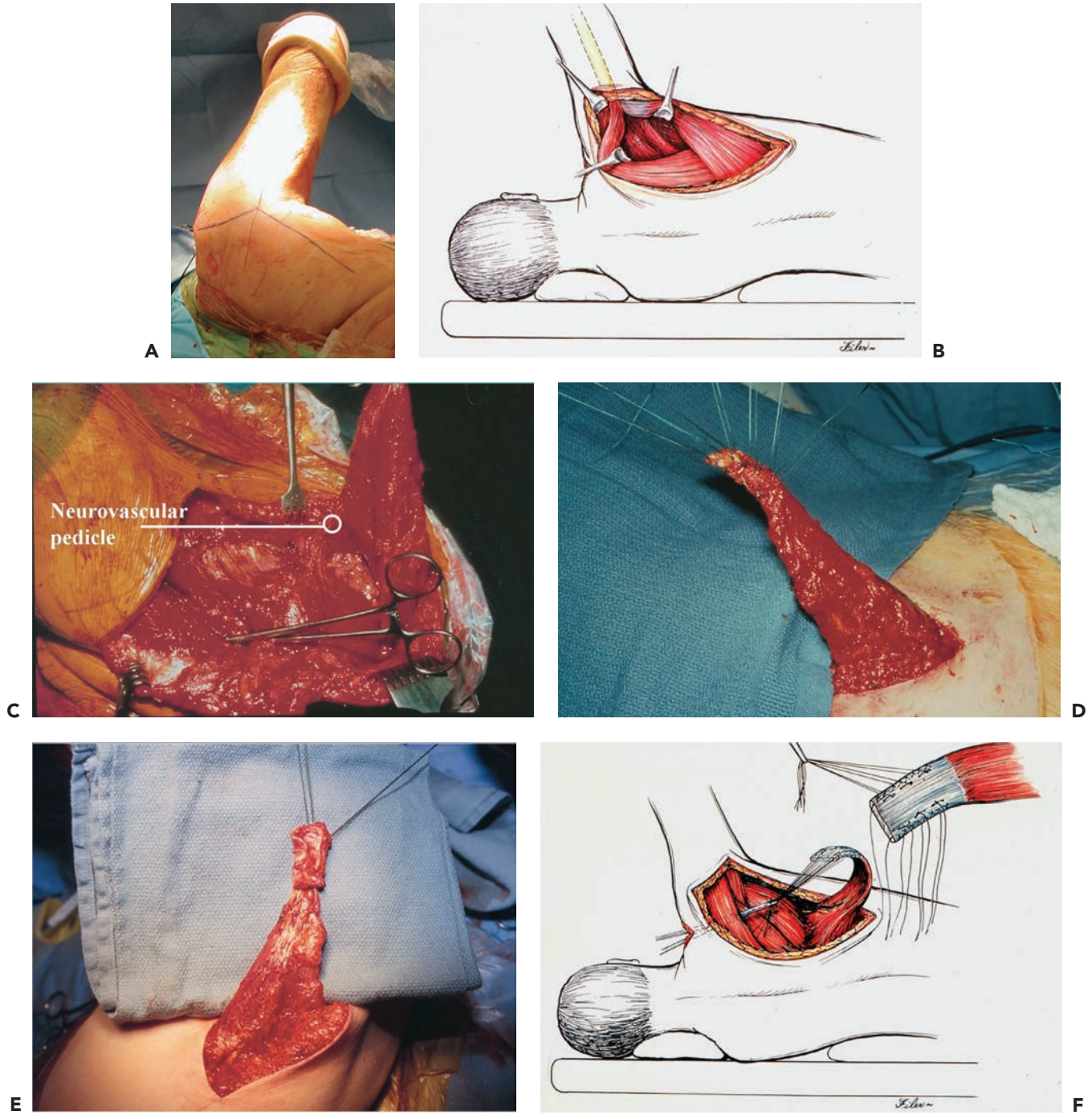
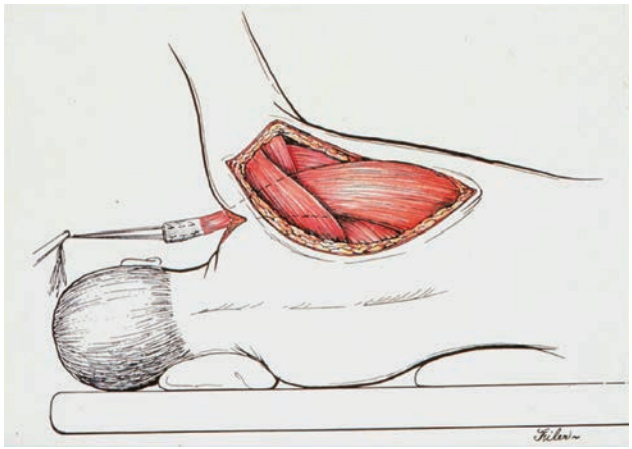


Figure 4-24 Technique of latissimus dorsi tendon transfer (JPW). **(A)** Skin incision follows anterior border of latissimus and curves along posterior axillary line. **(B)** Identification of teres major (superior)–latissimus dorsi (inferior) interval. **(C)** Thoracodorsal neurovascular pedicle. **(D)** During mobilization of musculotendinous unit, the tendon is tagged with a nonabsorbable suture in a Krakow configuration to facilitate traction and later transfer. **(E)** Fascia lata augmentation of latissimus tendon. **(F,G)** Transfer of tendon beneath through interval, defined by inferior border of posterior head of deltoid and teres minor into subacromial space. **(H)** Definitive fixation of tendon transfer using bone anchors or transosseous tunnels.

the chest wall and can be clearly defined if tension is maintained on the tendon by an assistant. We place sutures on either edge of the tendon to facilitate this step (Fig. 4-24D).

The extent of tendon release and mobilization of the musculotendinous unit is determined by the surgeon intraoperatively such that it can be pulled to the level of the posterior acromion. This will ensure sufficient length for the transfer over the greater tuberosity. We usually release the tendon from attachments to the chest wall all the way to the inferior edge of the scapula. While the neurovascular



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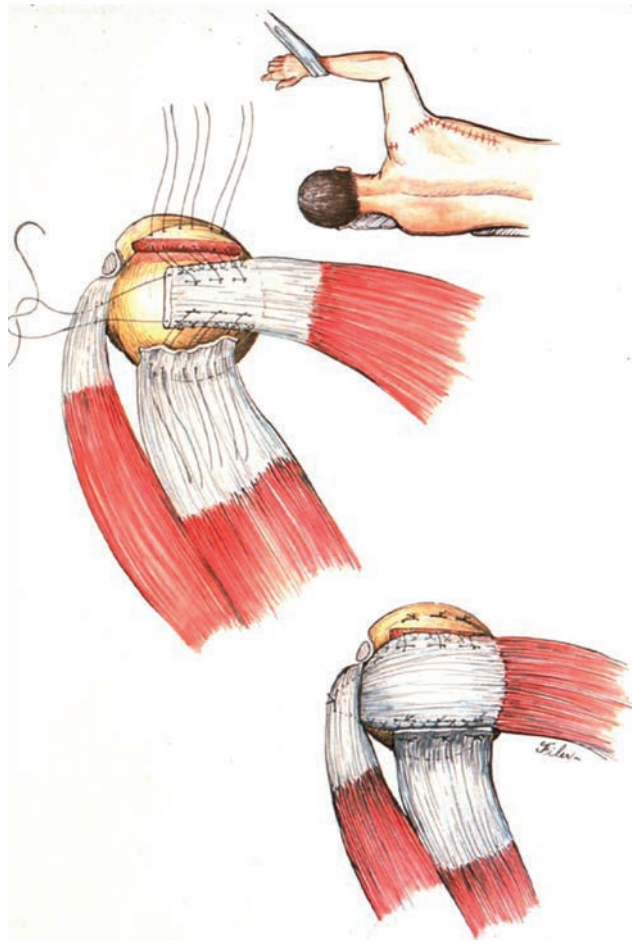


H

Figure 4-24 (*continued*)

pedicle is identified, its dissection and release is never necessary to gain mobility for the transfer.

Before transferring the tendon, we prefer to augment it with fascia lata from the ipsilateral thigh. Based on our initial experience, the tendon in many patients is quite diminutive and some patients had late rupture of the transfer. For this reason, we started to use the fascia lata to augment the tendon (Fig. 4-24E). A strip of fascia lata is typically 2 to 3 cm wide by 6 to 8 cm long. The defect in the fascia is then closed.

The fascia lata is secured to the latissimus tendon from the musculotendinous junction to the end of the tendon. While length of the tendon is almost never a problem, it is possible to lengthen the tendon with the graft. We use braided nonabsorbable suture (#2 Ethibond, Ethicon, Westwood, MA) along the length of the tendon and the graft.

The interval underneath the deltoid and superficial to the teres minor is then dissected from posterior, and a curved clamp is placed underneath the acromion so that it comes into this interval (Fig. 4-24F,G). This interval is then sharply and bluntly dissected to ensure that free and unrestricted excursion of the tendon transfer is possible.

A clamp is then passed from the anterosuperior incision beneath the acromion and deltoid to grasp the sutures in the latissimus tendon for the transfer. The arm is then placed in a position to tension the tendon transfer. This is approximately 45 degrees of abduction and 45 degrees of external rotation. Then tendon is fixed to the posterior rotator cuff and over the greater tuberosity using the previously placed bone anchors. Finally, it is fixed to the subscapularis, and the sutures in the remnants of the supraspinatus and infraspinatus are sewn into the medial edge of the tendon (Fig. 4-24H). The deltoid is then repaired to the acromion with transosseous sutures and the deltoid split is closed in a side-to-side fashion. After placing a sterile dressing, the shoulder is placed into a prefabricated abduction orthosis to maintain arm position and to protect the graft from excessive tension (SOBER Abductor, Pharmap, Crolles, France) (Fig. 4-25).

Postoperative Care

Phase I (first 6 weeks) consists of continuous wearing of the brace and passive motion by the therapist bringing the arm into abduction and external rotation. Adduction and internal rotation are not permitted. This ensures that the

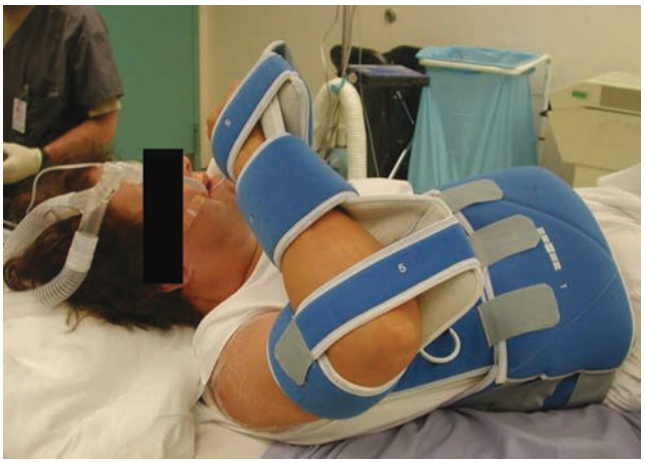


Figure 4-25 Abduction brace for postoperative protection of tendon transfer (SOBER Abductor, Pharmap, Crolles, France).

tendon moves in its new soft tissue tunnel and will not become tethered while it heals.

Phase II (second 6 weeks) consists of removal of the brace and then active assisted range-of-motion as well as continued passive range-of-motion exercises. The patient is permitted to use the arm for daily living activities, and water therapy may be commenced to facilitate return of passive motion arcs.

Phase III (12 weeks to 16 weeks) consists of ongoing active assisted motion and initiation of biofeedback program. A cutaneous biofeedback device is used by the patient (Myotrac, Thought Technology, Ltd., Montreal, Québec, Canada). The patient is then instructed by the therapist in the methods of training the latissimus transfer to become an active elevator and external rotator of the shoulder. The biofeedback unit is applied over the latissimus muscle. The first maneuver that the patient is instructed to perform is the "J-maneuver." The patient's shoulder is placed in approximately 90 degrees of flexion and the patient is instructed to pull downward and across the body. This will cause the latissimus to contract while the biofeedback unit gives the patient audiovisual feedback. During sustained contraction of the latissimus muscle, the patient then attempts to raise the arm into flexion, so as to trace the letter "J" with the hand. This maneuver sustains contraction of the latissimus muscle during elevation of the arm (Fig. 4-26).

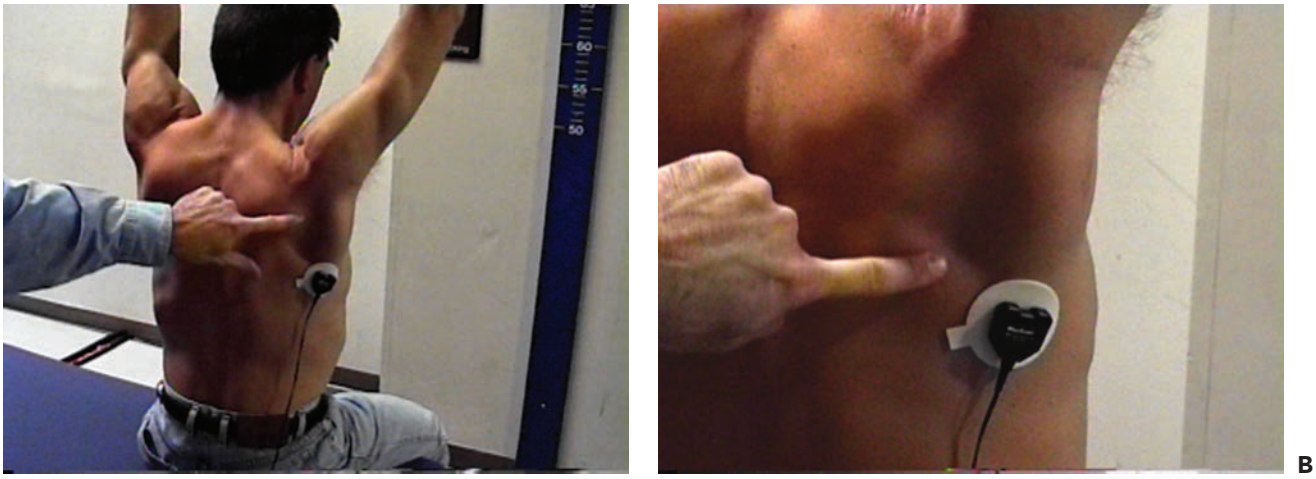
Once the patient is able to initiate and maintain contraction of the latissimus with the "J-maneuver," he or she is instructed to attempt contraction of the latissimus with external rotation. This can be facilitated by having the patient lie on his or her opposite side while pulling downward, followed by active external rotation.

In phase IV (16 weeks), biofeedback continues as long as necessary, and it may take up to 1 year for patients to successfully train the tendon transfer to actively assist in arm elevation and external rotation. Strengthening is begun when the patient achieves latissimus contraction during elevation, and we try to limit this to elastic bands rather than free weights.

Dr. Gerber's Technique in Beach Chair

Surgery is performed under general anesthesia combined with patient-controlled interscalene analgesia (PCIA), which facilitates early passive mobilization postoperatively. The patient is placed in a beach-chair position in such a manner that sufficient access to the entire scapula and latissimus dorsi muscle belly is possible using a full-length beanbag. A mechanical arm holder is not used during the operation.

The bony landmarks of the shoulder are palpated and outlined, including the acromion, acromioclavicular joint, coracoid process, and clavicle.



A B

Figure 4-26 Biofeedback training of latissimus muscle following tendon transfer involves use of a biofeedback unit (Myotrac, Thought Technology, Ltd., Montreal, Québec, Canada) and the J-maneuver.

An anterosuperior approach to the rotator cuff is performed through a 12-cm incision parallel to Langer's lines over the lateral one-third of the acromion (Fig. 4-27A). In general, this incision begins at the posterolateral edge of the acromion and ends anteriorly 2 to 3 cm lateral to the coracoid process. The anterolateral deltoid is detached from its origin with a thin wafer of bone using a flexible 1/2-in. osteotome to allow for preferential bone-to-bone healing following repair of the deltoid at the completion of the procedure (Fig. 4-27B–D). The deltoid is split no more than 5 cm from its origin at the junction of the anterior and middle raphe to avoid possible injury to the axillary nerve.

The subacromial bursa is resected sharply, and minimal anterior acromioplasty is performed, as well as distal clavicle resection if needed. A subacromial retractor (Sulzer Medica, Winterthur, Switzerland) (Fig. 4-28) is placed to allow visualization of the entire rotator cuff, from the teres minor to the subscapularis. The long head of the biceps is routinely tenodesed into the bicipital groove using a nonabsorbable anchor, as it is frequently degenerated in the setting of a massive tear and may persist as a significant pain generator following surgery.

Mobilization of the supraspinatus and infraspinatus tendons is then performed, beginning on the bursal surface and proceeding to the articular surface, followed by placement of traction sutures using nonabsorbable, braided suture (#2 Ethibond, Ethicon, Johnson & Johnson, Westwood, MA) in a modified Mason-Allen configuration (Fig. 4-29). Mobilization may include release of the coracohumeral ligament, interval slide, and circumferential capsulotomy. Release of the articular side of the posterosuperior cuff does not extend beyond 1.5 cm medial to the glenoid rim to avoid inadvertent injury to the suprascapular nerve.[136]

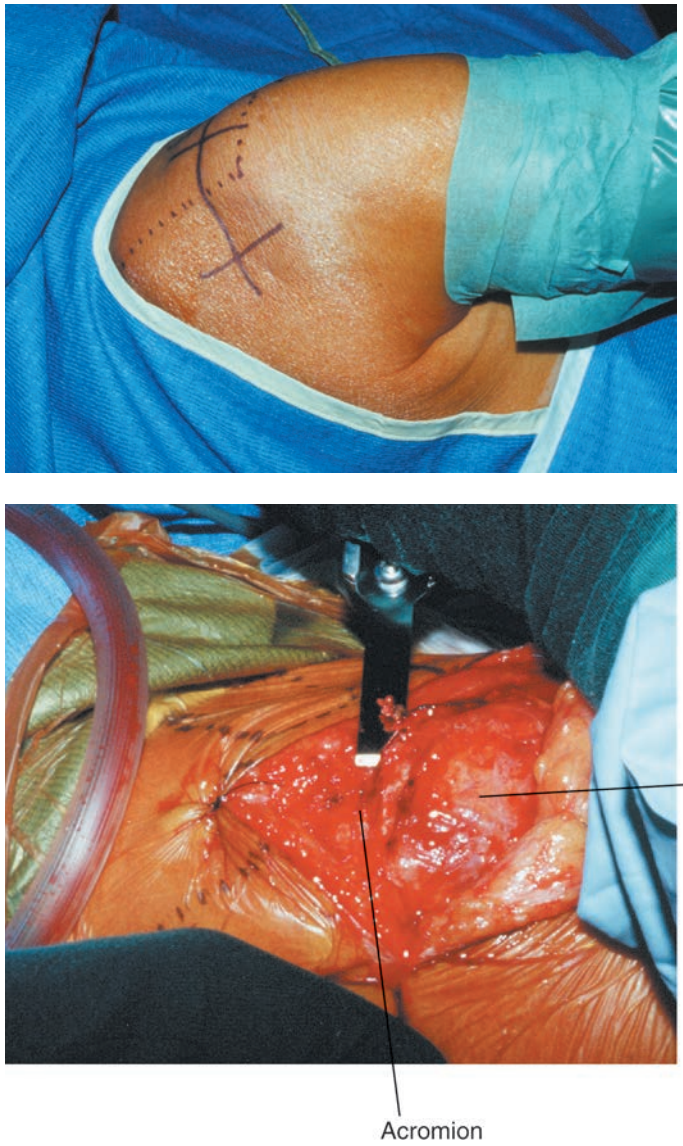
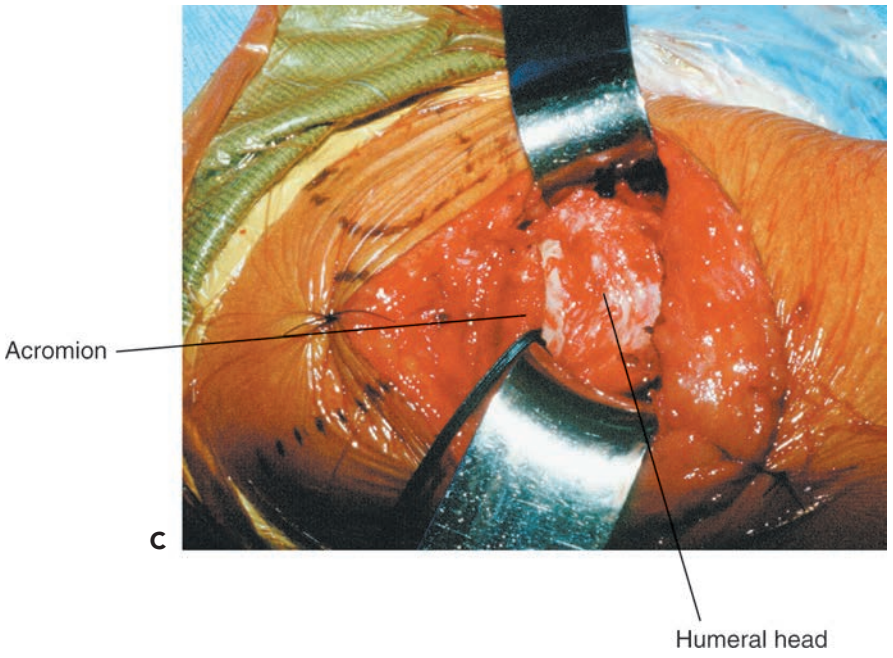


Figure 4-27 Anterosuperior exposure. **(A)** The incision is parallel to Langer's lines over the lateral one-third of the acromion beginning at the posterolateral edge of the acromion and extending anteriorly to 2 to 3 cm lateral to the coracoid process. **(B)** Deltoid exposure with middle deltoid detachment using ½-in. straight osteotome from anterolateral acromion. **(C,D)** Middle deltoid detachment enables exposure underlying humeral head and rotator cuff. (Reprinted with permission from Holovacs TF, Espinosa N, Gerber C. Latissimus dorsi transfers in rotator cuff reconstruction. In: Craig EV, Thompson RC, eds. *Master techniques in orthopaedic surgery: the shoulder*. Philadelphia: Lippincott Williams & Wilkins, 2004.)



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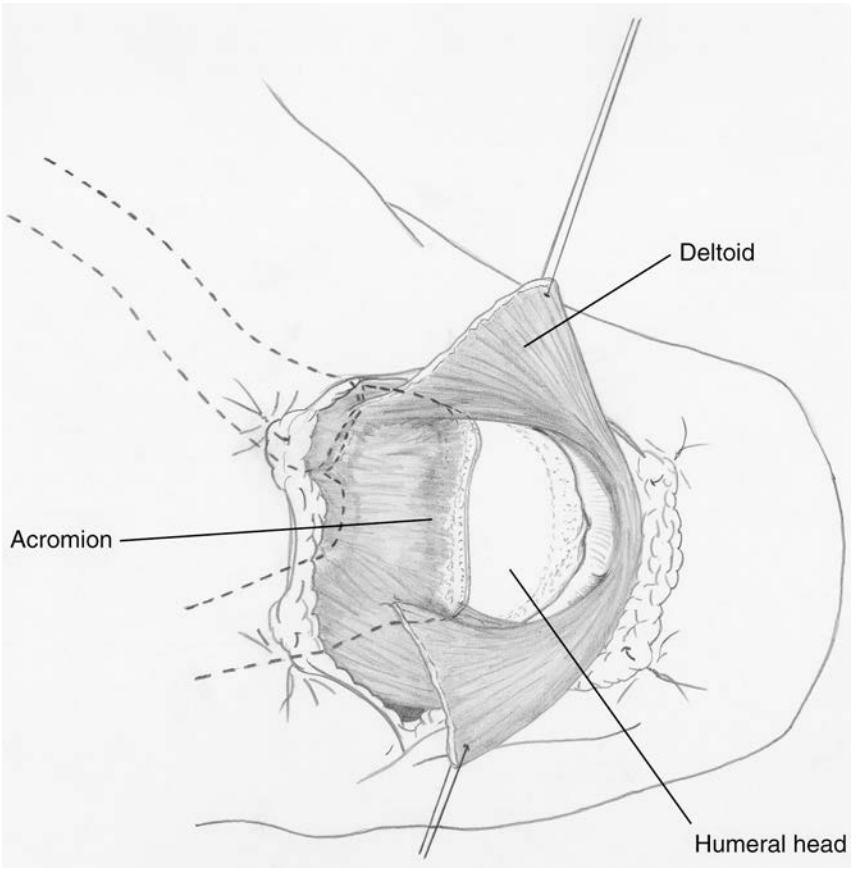


D

Figure 4-27 (*continued*)

Attention is then directed to harvest of the latissimus dorsi tendon. With the arm fully elevated, adducted, and internally rotated using a second assistant, the anterior border of the latissimus dorsi muscle is palpated. An approximately 25-cm incision is made from the anterior border of the latissimus dorsi muscle, which curves to the posterolateral upper humerus about 4 cm distally and does not traverse the axilla (Fig. 4-30A). Sharp dissection is used to raise subcutaneous flaps just superficial to the underlying fascia over the posterior deltoid, long head of the tri-

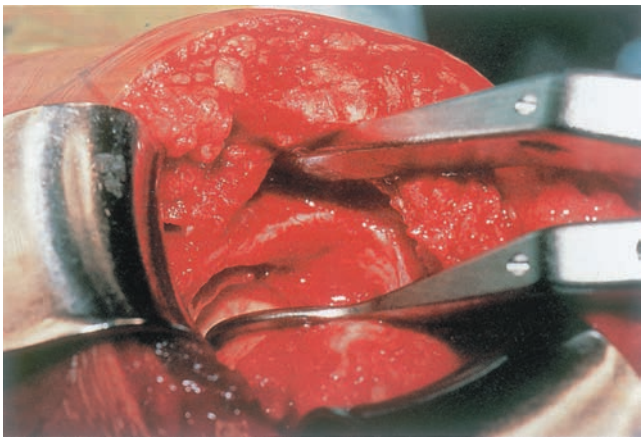


Figure 4-28 Subacromial retractor used during anterosuperior exposure (Sulzer Medica, Winterthur, Switzerland). This device has a ring that is used to retract the humeral head inferiorly and a straight limb analogous to a lamina spreader that distracts the acromion superiorly to enlarge the subacromial space. This greatly facilitates identification and mobilization of a retracted rotator cuff tendon.

ceps, teres major, and latissimus dorsi. The anterior latissimus is dissected off the chest wall fascia, beginning at the level of the muscle belly and proceeding proximally toward the insertion of the tendon on the proximal humerus (Fig. 4-30B). The radial nerve crosses over the humerus immediately distal to the insertion of the latissimus tendon on the humerus, while the circumflex vessels and axillary nerve are located just proximal to the tendon.

The interval between the superior border of the latissimus and the inferior border of the teres major is often difficult to distinguish, but can be more easily identified at the more proximal portion of either muscle. The latissimus tendon is released from its insertion using a long-handled no. 15 scalpel, and tagged with two no. 3 Ethibond sutures in a Krakow configuration (Fig. 4-30C–E). As traction is applied on the muscle, dissection can continue distally to achieve sufficient excursion of the musculotendinous unit to reach the superolateral aspect of the greater tuberosity (Fig. 4-30F). The thoracodorsal neurovascular pedicle does not necessarily need to be identified, but can be found as it enters the muscle approximately 10 cm distal to the musculotendinous junction.

The interval between the inferior border of the posterior deltoid and underlying teres minor is identified and developed using a combination of sharp and blunt dissection. A curved Mayo clamp is passed from the previous anterosuperior exposure into this interval (Fig. 4-30F). It is extremely important to adequately develop this potential space to allow for free and unrestricted excursion of the tendon transfer with the proximal humerus. The latissimus dorsi tendon is then transferred to the superolateral humerus (Fig. 4-30G).

The tagged end of the latissimus is sutured to the superior border of the subscapularis through bone tunnels passing through the lesser tuberosity (Fig. 4-30H). The previously mobilized supraspinatus and infraspinatus tendons are either repaired to a bony trough at their anatomic insertion through bone tunnels (if sufficient mobilization is possible) prior to transfer of the latissimus, or more often, repaired to the medial border of the tendon transfer. The lateral border of the tendon transfer is repaired through bone tunnels passing through the greater tuberosity and exiting laterally over a titanium cortical bone augmentation device, which prevents cutting of the sutures through the bone (Stratec/Synthes, Paoli, PA) (Fig. 4-31).

Suction drains are routinely placed in both the inferior and anterosuperior wounds deep to fascia. The deltoid split is reapproximated with a running 0-PDS monofilament suture (Ethicon, Johnson and Johnson, Westwood, MA). The osteotomy of the anterolateral middle deltoid is repaired with #2 fiberwire (Arthrex, Naples, FL) passed through drill holes in the lateral acromion. The superficial fascia of the deltoid split is reinforced with a running 2-0 PDS monofilament suture (Ethicon, Somerville, NJ).

Sterile dressings are applied, which are watertight to enable early aquatherapy postoperatively. Prior to transfer off the table, the patient is placed into an abduction brace (SOBER Abductor, Pharmap, Crolles, France) at 45 degrees of abduction and 45 degrees of external rotation, and the

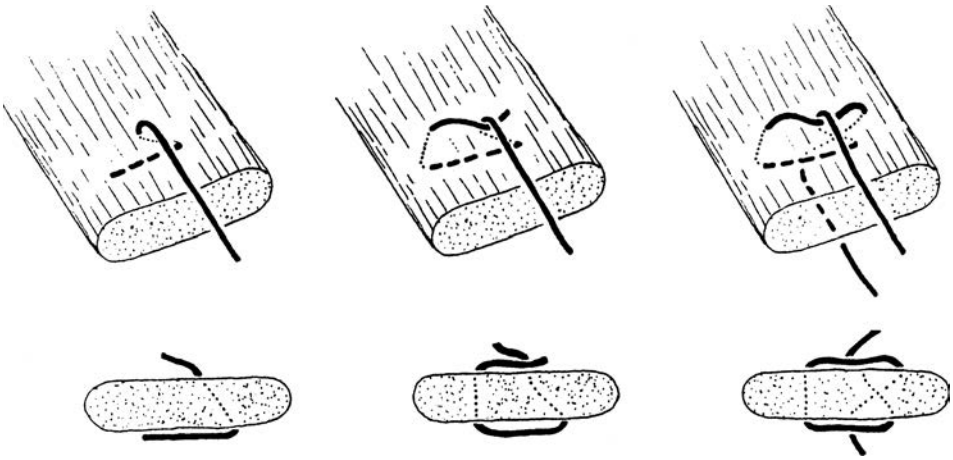


Figure 4-29 A modified Mason-Allen tendon-grasping technique increases the pullout strength of the suture by a factor greater than 2 when compared with a simple stitch or with a mattress-type suture.

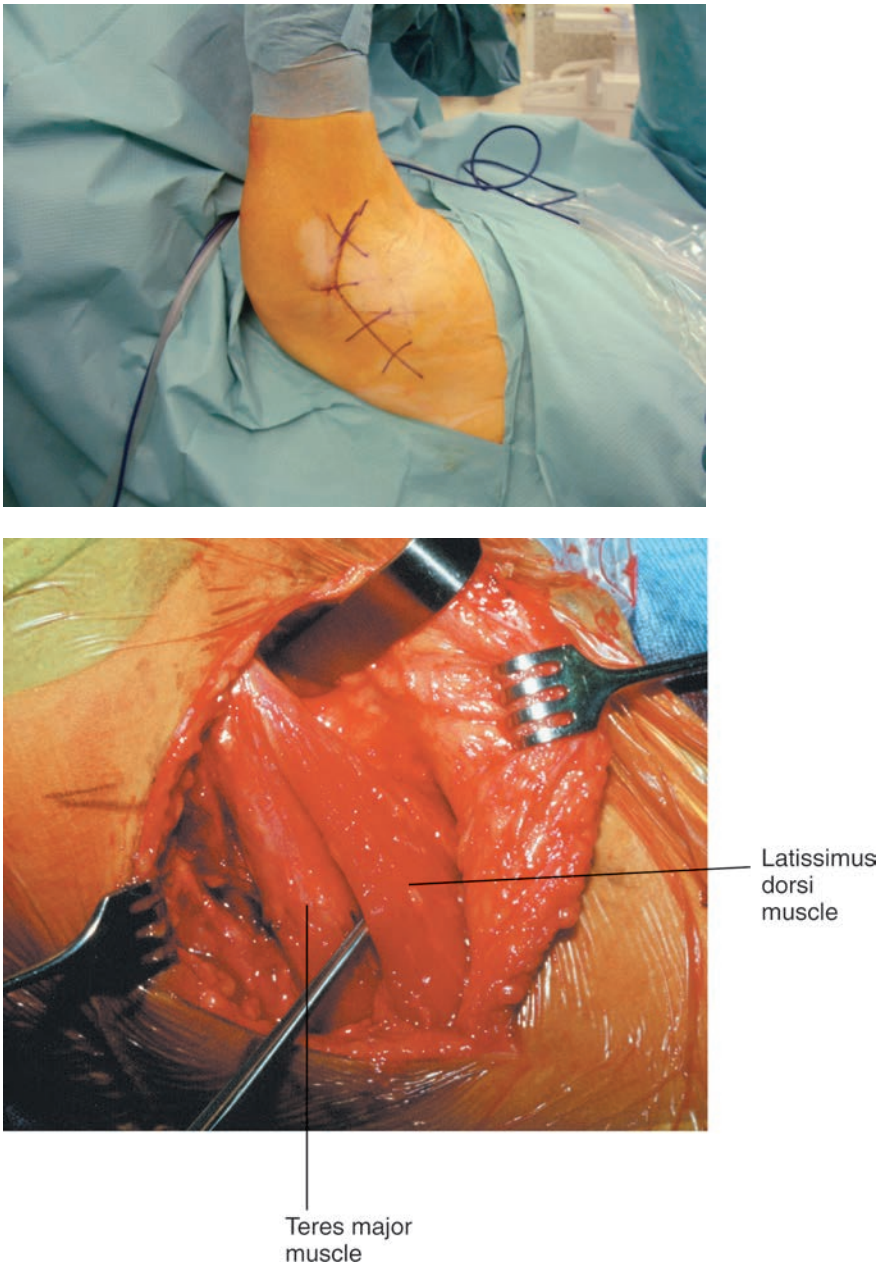


Figure 4-30 Technique of latissimus dorsi tendon transfer (CG). **(A)** Posteroinferior exposure. Line of incision follows the anterior border of the latissimus dorsi muscle belly and curves 4 cm proximal to the axillary fold ending at the inner one-third of the proximal humerus. **(B)** Latissimus dorsi exposure with identification of the posterior deltoid and teres major superiorly. **(C)** Identification of insertion of latissimus muscle on proximal humerus and release with Metzenbaum scissors or no. 15 scalpel blade. **(D)** Fully released tendon. **(E)** Traction sutures are placed in a Krakow configuration. **(F)** The muscle is dissected until the pedicle is identified, a clamp is passed between the deltoid and external rotators, and the tendon is **(G)** pulled superiorly into the subacromial space. The tendon is sutured to the tip of the greater tuberosity and the remaining stump of the supraspinatus and infraspinatus are sutured end to side to the latissimus tendon **(H)**. (Reprinted with permission from Holovacs TF, Espinosa N, Gerber C. Latissimus dorsi transfers in rotator cuff reconstruction. In: Craig EV, Thompson RC, eds. *Master techniques in orthopaedic surgery: the shoulder.* Philadelphia: Lippincott Williams & Wilkins, 2004.)

brace is worn continuously for 6 weeks following surgery (Fig. 4-25).

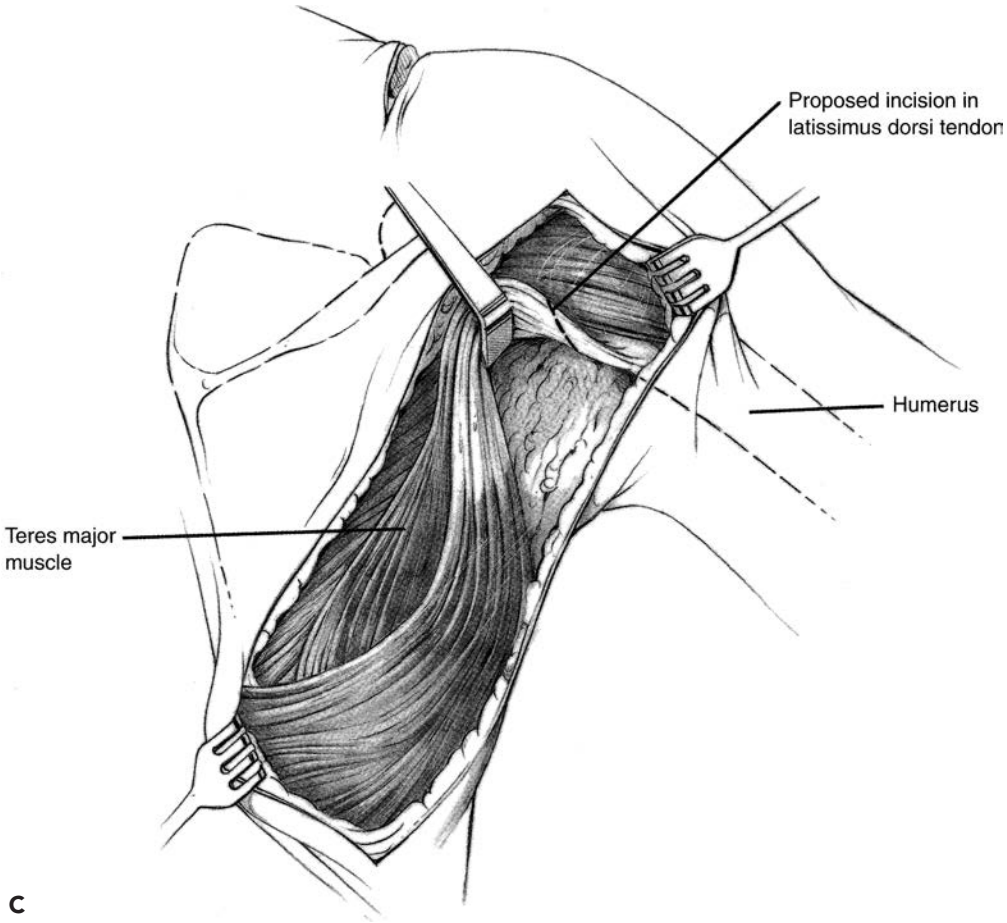
Postoperative Care

Passive range-of-motion exercises with the arm maintained in 45 degrees of external rotation and isometric contraction of the deltoid are initiated on postoperative day 1. Passive motion is important not only with respect to tendon healing,[29,32,118,119] but also to maintain glenohumeral motion and prevent adhesion of the tendon transfer to surrounding soft tissues. Aquatherapy is also initiated at this time on a routine basis. The splint is discontinued after 6 weeks, at which time active external rotation and abduction are initiated, as well as passive internal rotation. The patient is allowed to begin performing gentle activities of daily living at this time. Strengthening exercises are not allowed until 3 months after surgery and are continued for 6 to 9 months. It may take up to 12 months for complete retraining of the transferred latissimus dorsi to occur (Fig. 4-32).

Pectoralis Major Transfer for Anterosuperior Rotator Cuff Defect

Dr. Warner's Technique for Pectoralis Major Transfer

The patient is placed in the beach-chair position such that free access to the shoulder is possible (Tmax Beach Chair,



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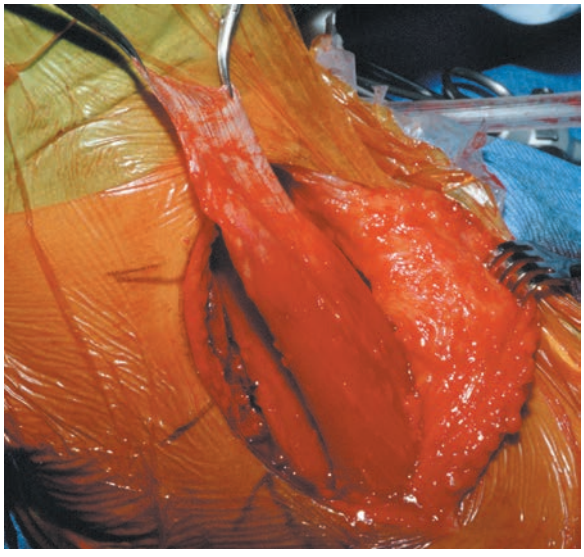

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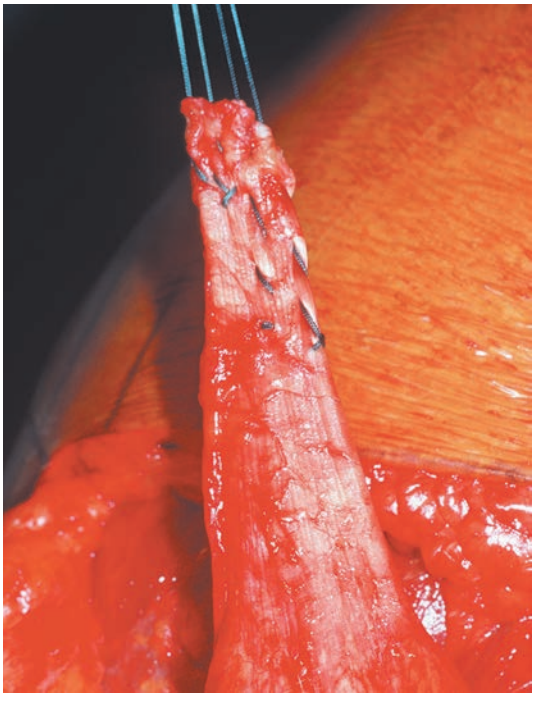

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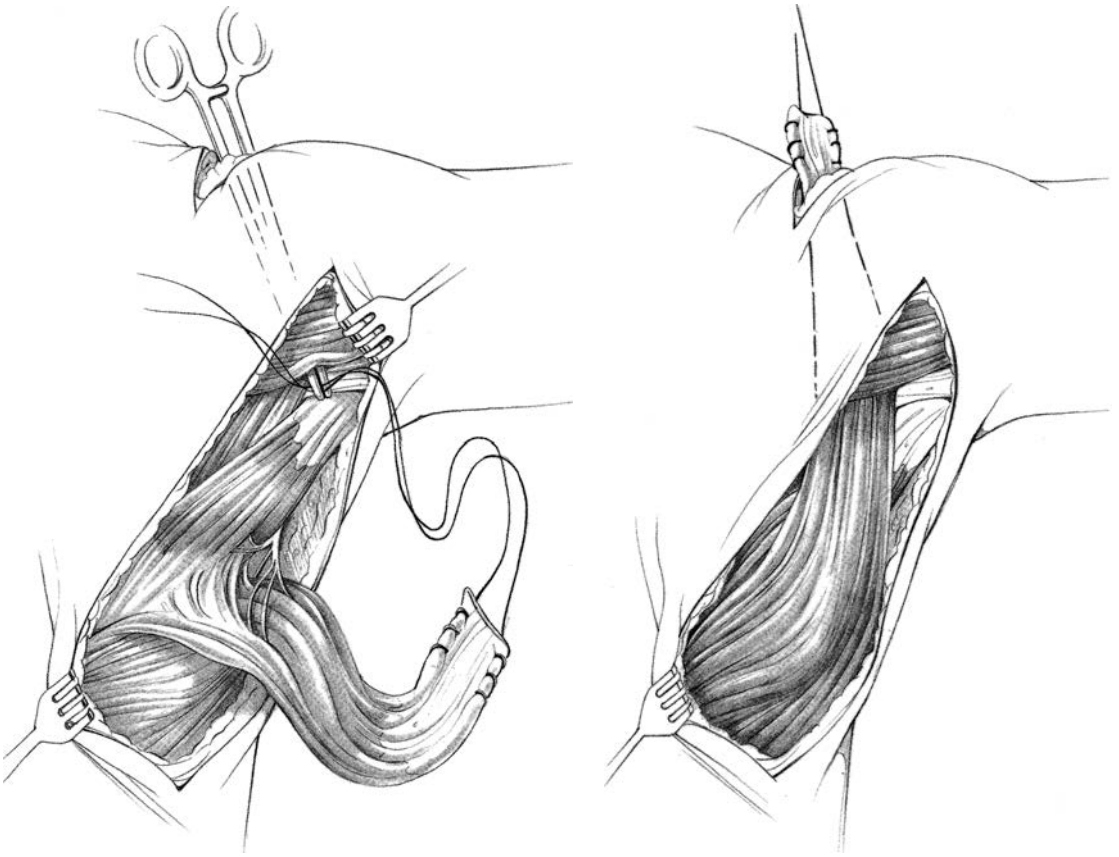
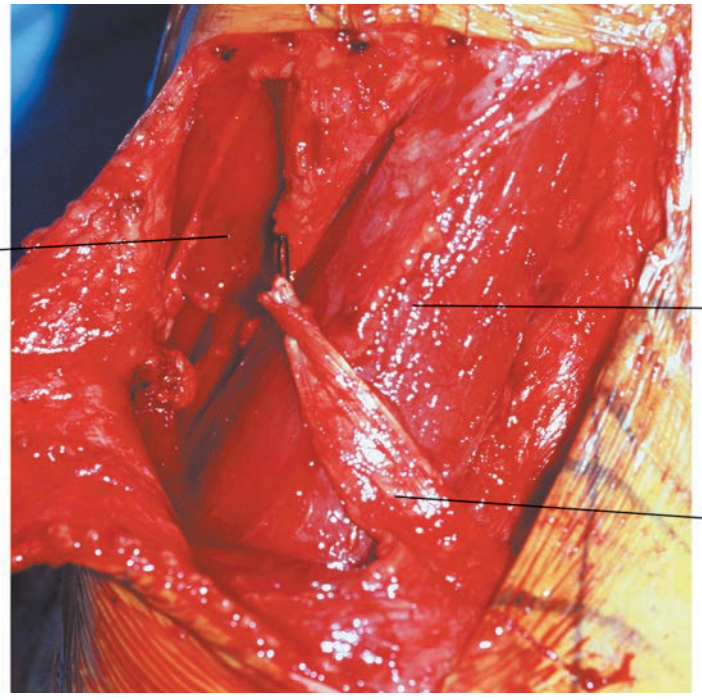
Figure 4-30 (*continued*)

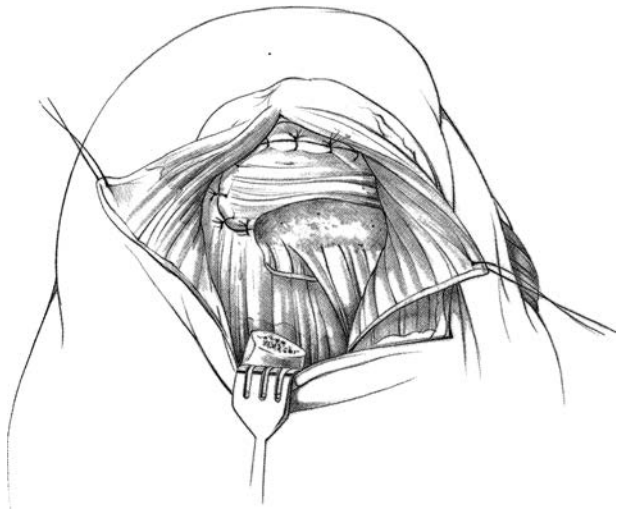


F G(I)



G(II)

Figure 4-30 (*continued*)



H

Figure 4-30 (*continued*)

Tenet Medical Engineering, Calgary, Alberta, Canada). An articulated hydraulic arm holder (Spider Arm Positioner, Tenet Medical Engineering, Calgary, Alberta, Canada) is used on the ipsilateral side of the operating table to facilitate proper arm positioning during exposure and tendon harvest and to enable proper tensioning of the tendon transfer (Fig. 4-33). A combined regional and general anesthetic is administered, and antibiotic prophylaxis is given in all cases prior to surgery.

An extended deltopectoral approach is performed to expose the inferior border of the pectoralis major tendon as well as the latissimus dorsi tendon. If, based on preoperative imaging or clinical examination, there exists suspicion of a supra- or infraspinatus tear, dissection is extended over the superior rotator cuff as previously described for the latissimus transfer. Repair of these structures, if warranted and feasible, is performed at this time.

The interval between the deltoid and the pectoralis major is developed. All subdeltoid adhesions are released. The subdeltoid bursa as well as any scar tissue overlying the lesser tuberosity is removed. Of note, scar tissue overlying the lesser tuberosity may be mistaken for native subscapularis tendon, which, in reality, has retracted medially beneath the conjoined tendon.

The biceps tendon usually shows signs of degeneration in the context of a subscapularis tear, or if intact, is usually unstable and medially subluxated or dislocated. For these reasons or to exclude this nidus as a possible pain generator postoperatively, the biceps is routinely tenodesed. The authors differ in their preferred methods of biceps tenodesis.

The subscapularis is then mobilized with the assistance of braided traction sutures (#2 Ethibond, Ethicon, Johnson & Johnson, Westwood, MA) that are placed through good-quality tissue in the retracted lateral edge of the subscapularis. The circumflex vessels are identified and controlled with suture ligature, and the axillary nerve is routinely identified and protected prior to mobilization of the retracted tendon. If the subscapularis is deemed irreparable following these measures, the decision is made to perform the pectoralis transfer. If only a portion of the subscapularis is reparable, most often the inferior one-third, this should be performed prior to pectoralis transfer as this may improve anterior stability of the humeral head (Fig. 4-34).

The sternal and clavicular heads of the pectoralis major tendon are identified at their humeral insertion (Fig. 4-35A,B). The tendon of the sternal head is invariably inferior and deep to the clavicular head, and the interval between these two portions can be easily developed at their insertion. The sternal head is detached (Fig. 4-35C) and tagged with braided nonabsorbable sutures in a modified Mason-Allen configuration. Medial dissection to facilitate transfer of the sternal head should not exceed 10 cm to avoid risk of denervation of the lateral pectoral nerve. Following dissection, the sternal head is passed beneath the clavicular head, superficial to the conjoined tendon, and anchored to the lesser tuberosity using either transosseous sutures or bone anchors in such a way that approximately 30 degrees of external rotation is possible (Fig. 4-35D). One of the authors (CG) uses a thin, seven-hole titanium plate (Stratec-Synthes, Oberdorf, Switzerland) to augment suture fixation at the inferolateral greater tuberosity.[53] The rotator interval is then closed by suturing the inferior edge of the supraspinatus to the superior border of the pectoralis transfer.

In the presence of a complete rupture of the subscapularis tendon in combination with a supraspinatus tear, a combined transfer of the teres major is performed in combination with the sternal head of the pectoralis major tendon (Fig. 4-36). In brief, the insertion of the latissimus dorsi tendon is identified and detached with a small cuff of tendon remaining on the humerus to enable later repair

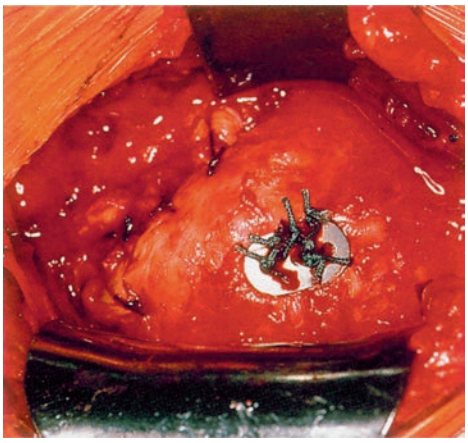


Figure 4-31 Direct repair of the lateral border of the tendon transfer is achieved with transosseous sutures, which are augmented using a titanium cortical bone augmentation device (Stratec/Synthes, Paoli, PA).

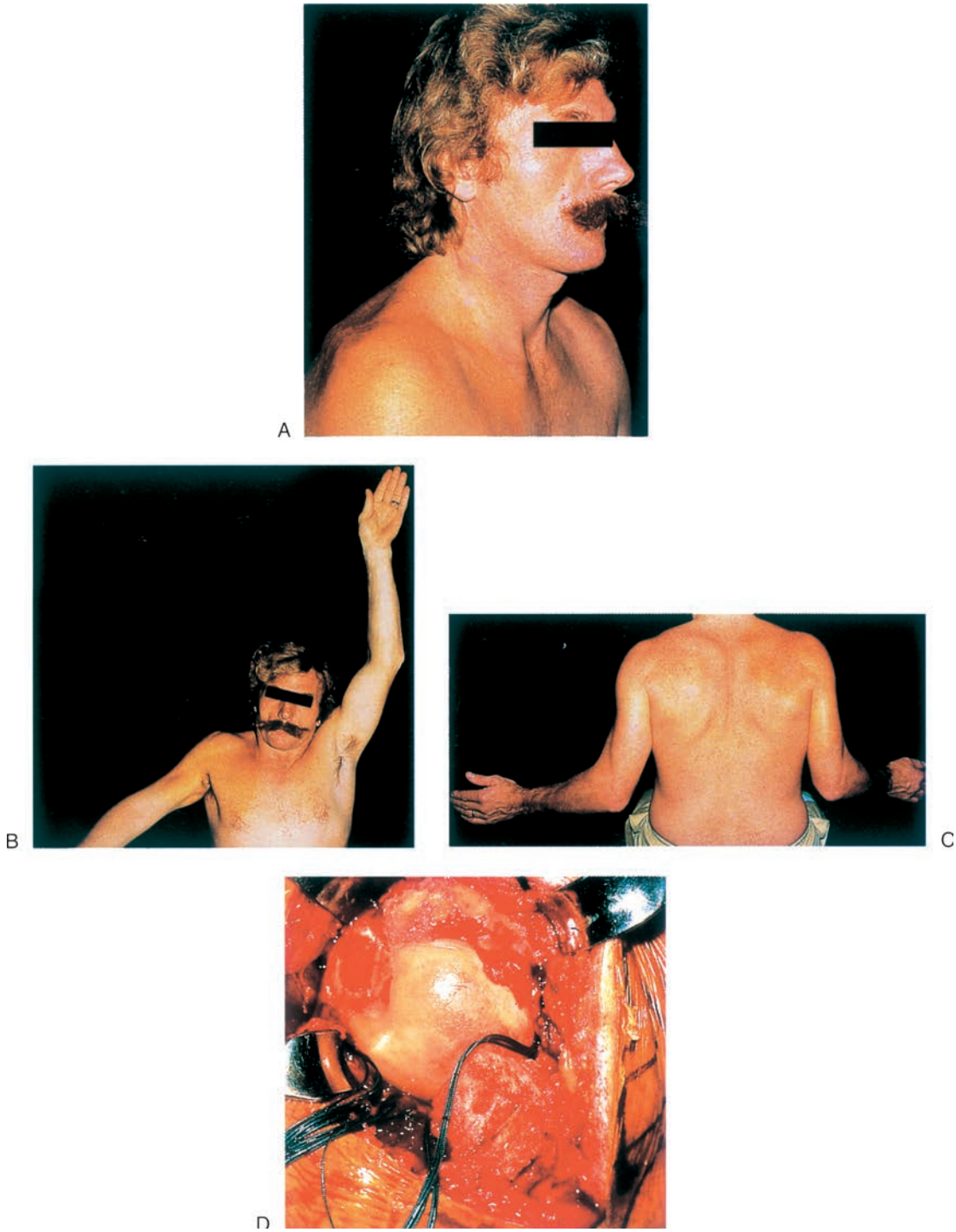


Figure 4-32 **(A)** A young patient with a massive tear and severe atrophy of the supra- and infraspinatus. **(B,C)** He has pseudoparalysis to scapular plane abduction and external rotation. **(D)** Intraoperatively, the residual supra- and infraspinatus tears are not reparable, and a latissimus dorsi tendon transfer is performed. **(E, F)** At 2 years postoperatively, the patient has recovered elevation and external rotation and is pain-free. **(G,H)** At 10 years postoperatively he maintains flexion and external rotation and is working as a truck driver.

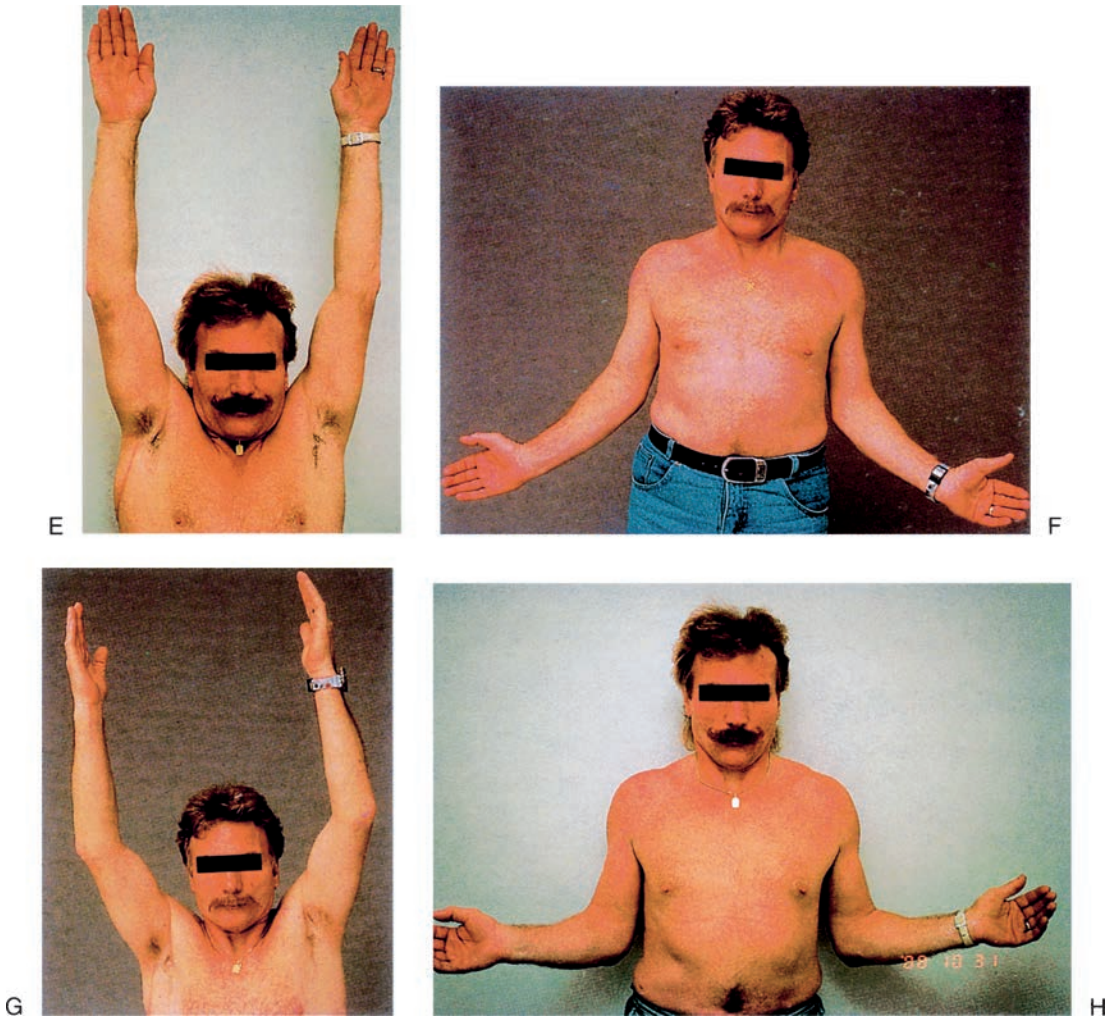


Figure 4-32 (*continued*)

(Fig. 4-36A). This procedure is facilitated with the arm in maximal external rotation. The interval between the teres major and latissimus is identified and developed laterally, with special care taken to avoid injury to the axillary nerve, which usually runs along the superior border of the teres major. Once the upper and lower borders of the teres major are released, the insertion is detached and tagged with nonabsorbable #2 suture (Fig. 4-36B). Care must be taken not to exceed 7 cm of medial dissection from the insertion in order to preserve the neurovascular pedicle supplying the muscle. The latissimus tendon is then repaired back to the stump left behind previously at the humeral insertion. The teres major and sternal head of the pectoralis major tendon are then passed beneath the clavicular head of the pectoralis major (Fig. 4-36C) and reattached to the lesser tuberosity using bone anchors (Fig. 4-36D).

Postoperative Care

Phase I (first 6 weeks) consists of continuous wearing of an abduction brace (UltraSlingII, Donjoy, Carlsbad, CA) and passive motion by the therapist, which brings the arm into adduction and internal rotation. Abduction and external rotation are not permitted. These measures ensure that the transferred tendon is able to move in a controlled fashion without compromising the integrity of the repair. The benefits of passive motion on tendon healing have been previously described.[29,32,118,119]

Phase II (second 6 weeks) consists of removal of the brace and active assisted range-of-motion as well as continued passive range-of-motion exercises. The patient is permitted to use the arm for activities of daily living at this time. Aquatherapy may be commenced to facilitate return of passive motion arcs.

Phase III (12 weeks to 16 weeks) consists of ongoing active assisted motion as well as gentle strengthening exercises using elastic bands. Phase IV (16 weeks) consists of strengthening exercises using free weights. Return to work or sports is not expected until 6 months after surgery. Completion of rehabilitation may take as long as 12 to 18 months.

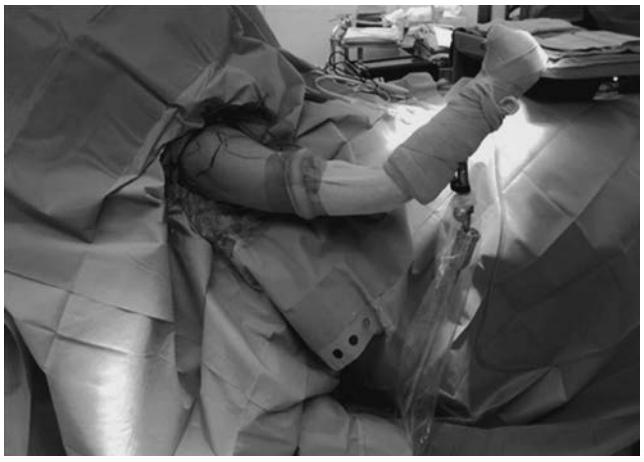


Figure 4-33 Beach-chair position for pectoralis major tendon transfer. An articulated hydraulic arm holder (Spider Arm Holder, Tenet Medical Engineering, Calgary, Canada) is used on the ipsilateral side of the operating table to facilitate proper arm positioning during exposure and tendon harvest as well as to enable proper tensioning of the tendon transfer. (Reprinted with permission from Gerber A, Clavert P, Millett PJ, Holovacs TF, Warner JJP. Split pectoralis major transfer and teres major tendon transfers for reconstruction of irreparable tears of the subscapularis. *Tech Shoulder Elbow Surg* 2004;5:5–12.)

Dr. Gerber's Technique for Pectoralis Transfer

Surgery is performed under general anesthesia combined with PCIA, which facilitates early passive mobilization postoperatively. The patient is placed in a beach-chair position using a full-length beanbag sufficiently lateral on the table to enable free access to the shoulder. A mechanical arm holder is not used during the operation.

An extended deltopectoral approach is made sharply through an approximately 12- to 15-cm incision (Fig. 4-37A). Extensive scar tissue, if present from previous surgery, is débrided as needed. The cephalic vein is generally retracted laterally with the deltoid, while the underlying conjoined tendon is gently retracted medially. Distal dissection is continued until exposure of the entire pectoralis major tendon insertion is identified. With the arm in gentle abduction and internal rotation, the subdeltoid adhesions are released sharply from the humerus followed by placement of a deltoid retractor.

At this point, the superior rotator cuff is assessed, and the proximal incision is extended to a superolateral approach if rotator cuff reconstruction is warranted.

In patients with subscapularis tears, the biceps tendon is frequently degenerated or subluxated, and for this reason, it is either tenotomized or tenodesed in the bicipital groove with an appropriate bone anchor (Super Anchor, Mitek, Ethicon, Johnson & Johnson, Westwood, MA) depending on the age and cosmetic demands of the patient.

The anterior humeral circumflex vessels are then identified and usually ligated or cauterized. The axillary nerve is routinely visualized by dissecting between the lower border of the subscapularis and circumflex vessels, and a vessel loop is placed around the nerve throughout the entire procedure. An attempt is then made to repair the subscapularis musculotendinous unit following mobilization and release from the rotator interval, the base of the coracoid, the brachial plexus, and the subscapularis fossa. The residual tendinous tissue is grasped with braided nonabsorbable suture (#2 Ethibond, Ethicon, Johnson & Johnson, Westwood, MA) with use of a modified Mason-Allen technique. Both strands of one suture are passed through the lesser tuberosity transosseously, into the medullary canal, and out through the greater tuberosity with the arm in neutral rotation. As described for latissimus transfer, a cortical augmentation plate is used to strengthen the repair (Stratec-Synthes, Paoli, PA). If only a partial repair of the subscapularis is possible, usually the inferior portion, this should be performed in addition to a pectoralis transfer (Fig. 4-37B).

The upper and lower borders of the pectoralis major tendon are identified at the tendon's insertion on the humerus (Fig. 4-37B). The entire tendinous insertion is released to maximize the length of tendon that is harvested from superior to inferior and tagged with three braided #3 Ethibond sutures (Ethicon, Johnson & Johnson, Westwood, MA) in a modified Mason-Allen configuration (Fig. 4-37C,D). The musculotendinous unit is then mobilized sufficiently to enable passage superficial to the conjoined

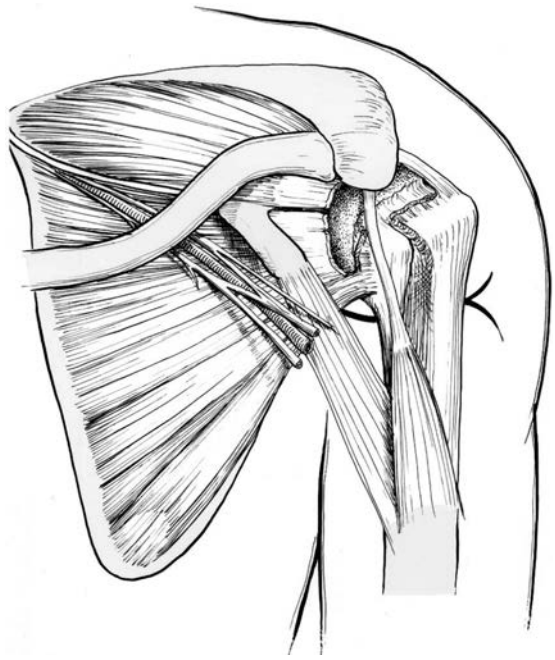
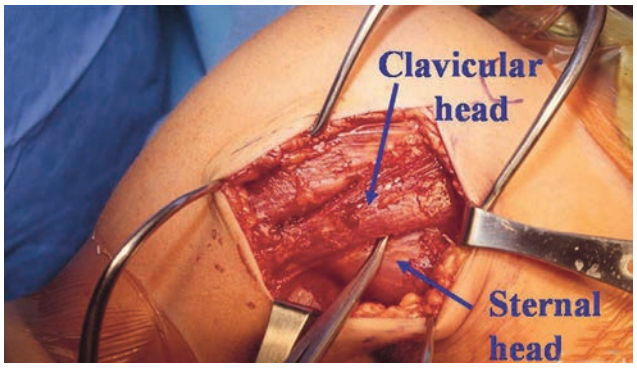


Figure 4-34 The inferior portion of the subscapularis is often intact or can be repaired prior to transfer of the pectoralis major tendon. (Reprinted with permission from Gerber A, Clavert P, Millett PJ, Holovacs TF, Warner JJP. Split pectoralis major transfer and teres major tendon transfers for reconstruction of irreparable tears of the subscapularis. *Tech Shoulder Elbow Surg* 2004;5:5–12.)



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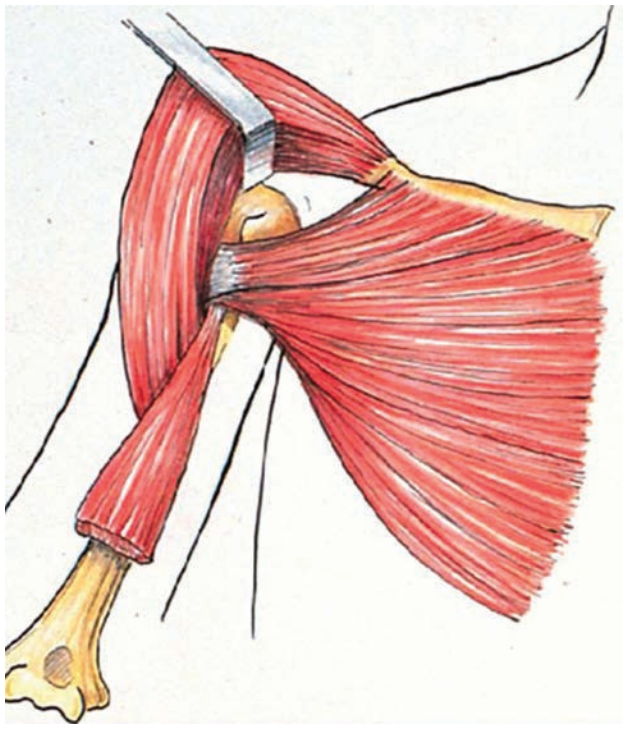

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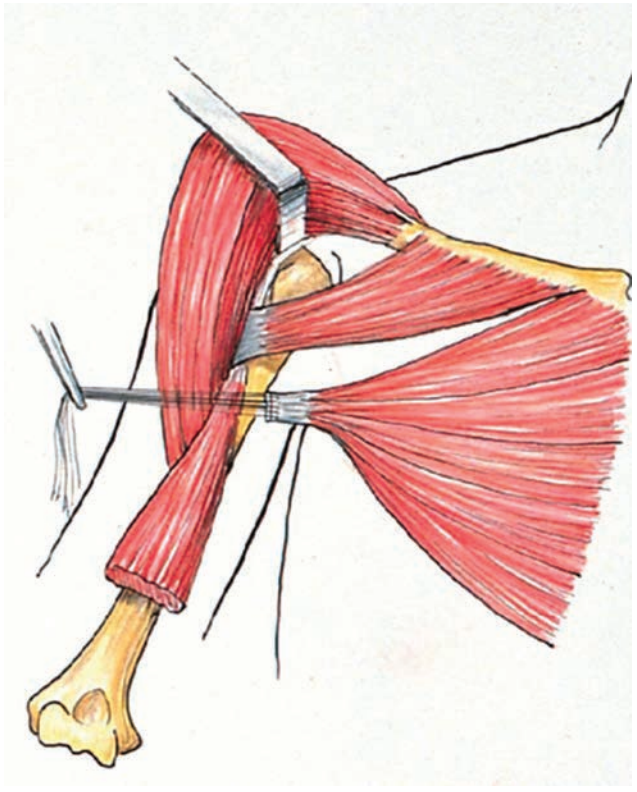

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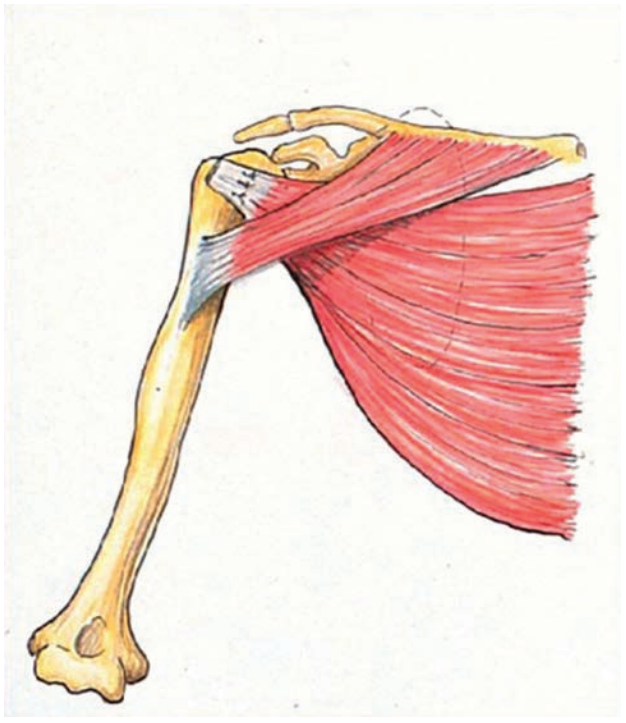


D

Figure 4-35 Technique of split pectoralis major tendon transfer (JPW). **(A,B)** Identification of the sternal and clavicular heads of the pectoralis major tendon in a right shoulder. The tendon of the sternal head is invariably inferior and deep to the clavicular head. **(C)** Detachment of the sternal head of the pectoralis major tendon and placement of traction sutures. The sternal head of the pectoralis is transferred underneath the clavicular head and **(D)** repaired to the lesser tuberosity using bone anchors.

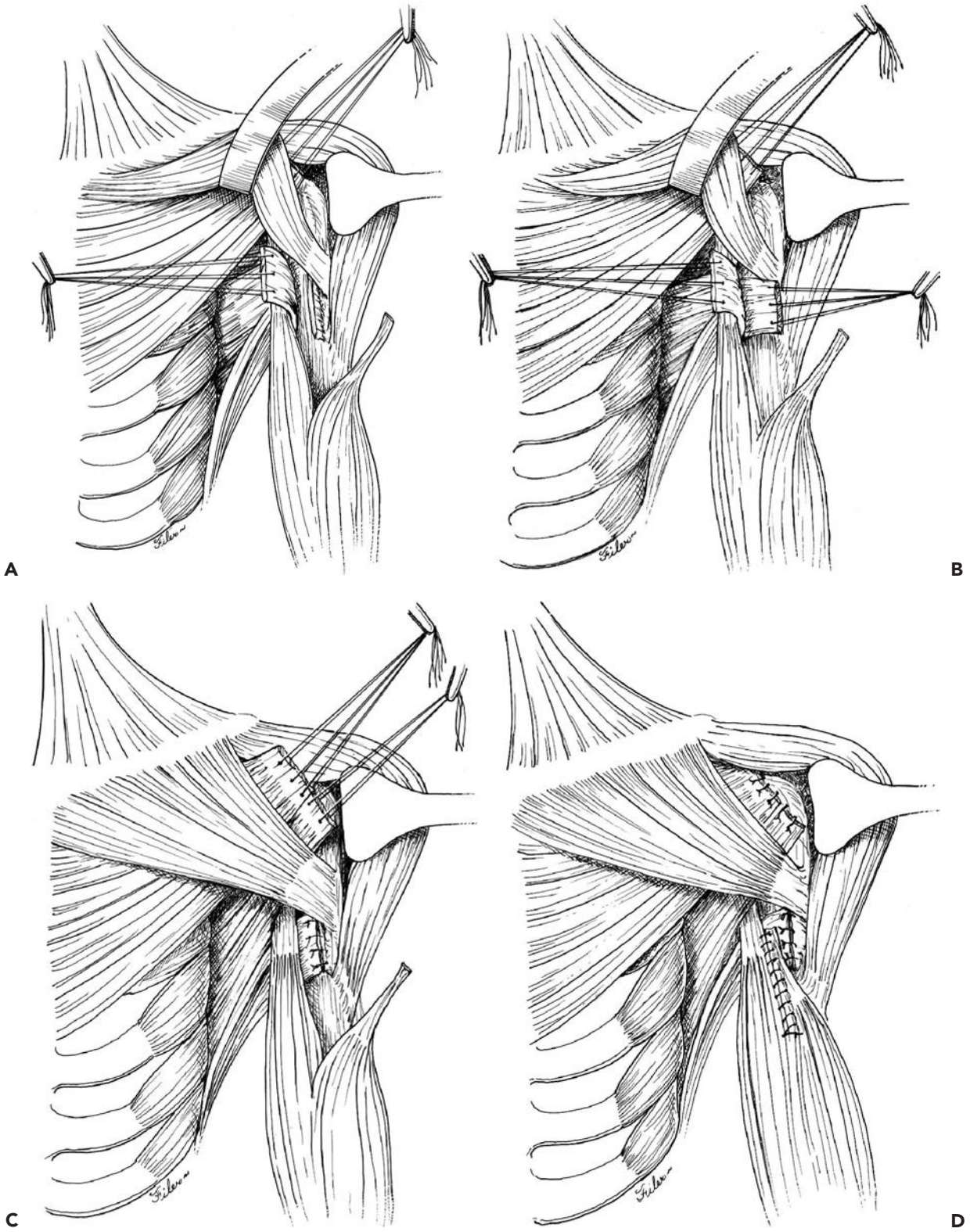


Figure 4-36 Technique of combined split pectoralis major and teres major tendon transfer (JPW). **(A)** The latissimus dorsi tendon is detached from the humerus to expose the teres major tendon. **(B)** The teres major tendon is detached from the proximal humerus. **(C)** Both tendons are transferred to the lesser tuberosity beneath the clavicular head of the pectoralis major tendon, which remains attached to its anatomic footprint. **(D)** Reconstruction of the upper part of the subscapularis with the pectoralis major and the lower part with the teres major. (Reprinted with permission from Gerber A, Clavert P, Millett PJ, Holovacs TF, Warner JJP. Split pectoralis major transfer and teres major tendon transfers for reconstruction of irreparable tears of the subscapularis. *Tech Shoulder Elbow Surg* 2004;5:5–12.)

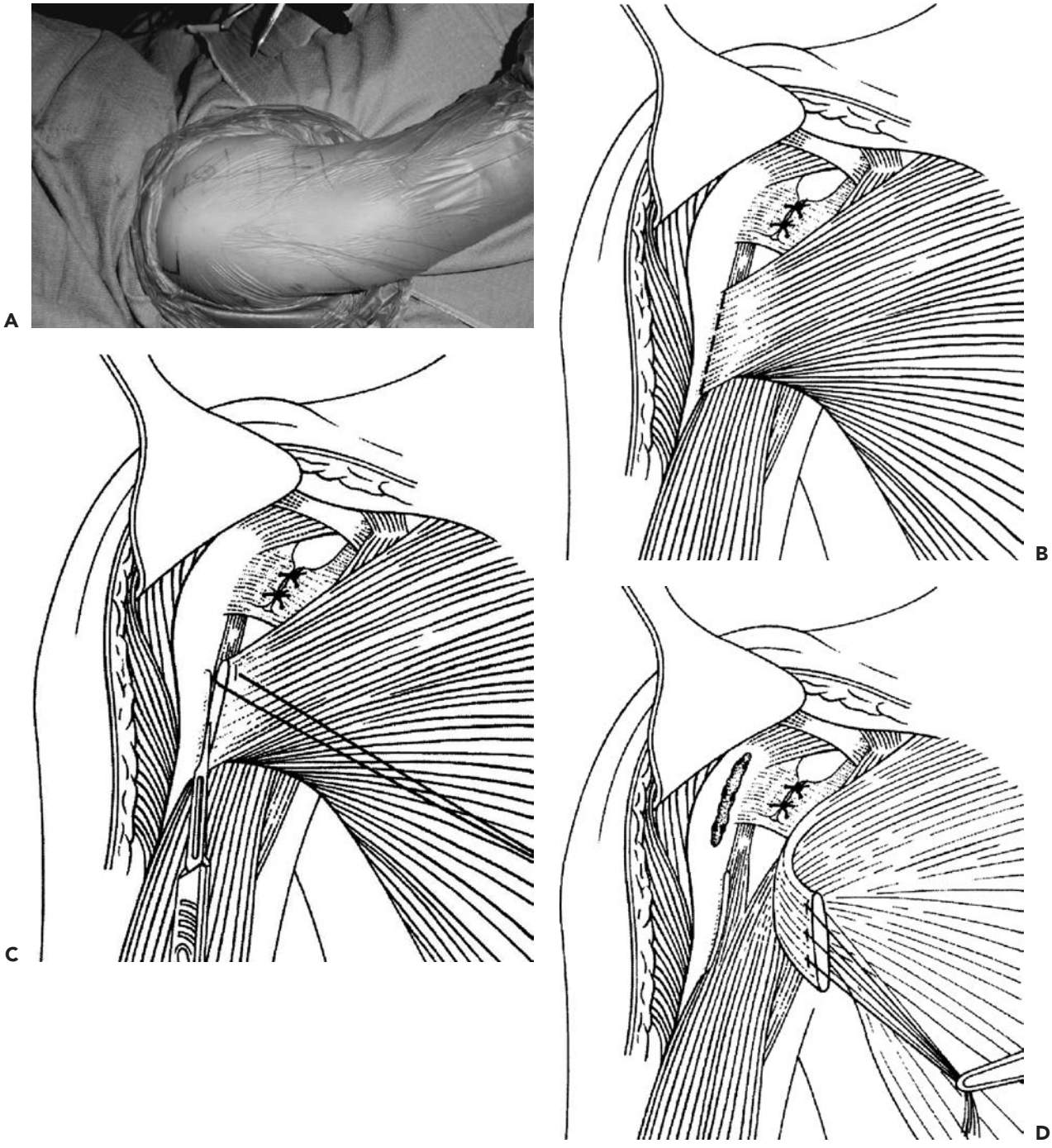
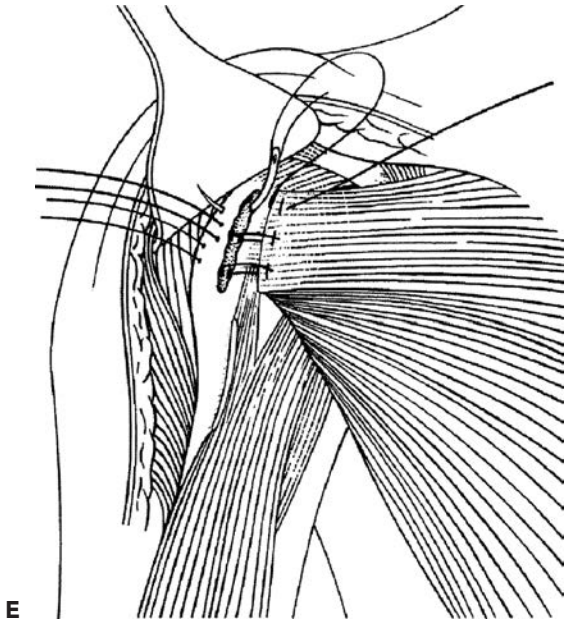
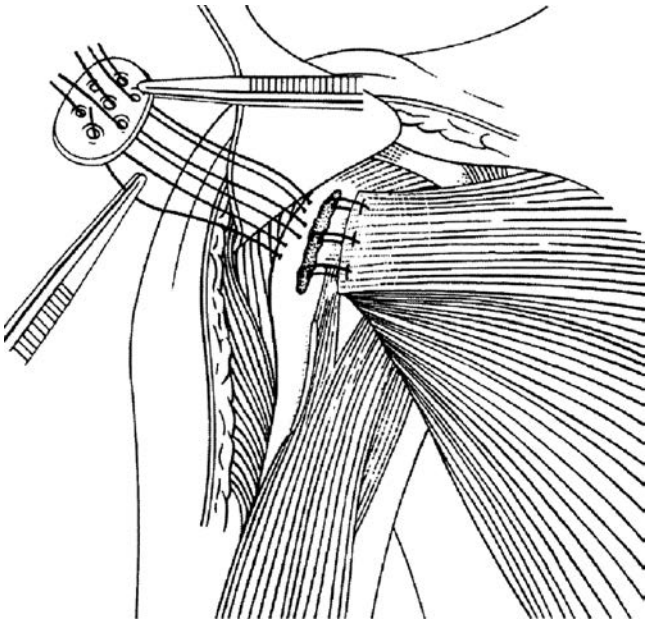


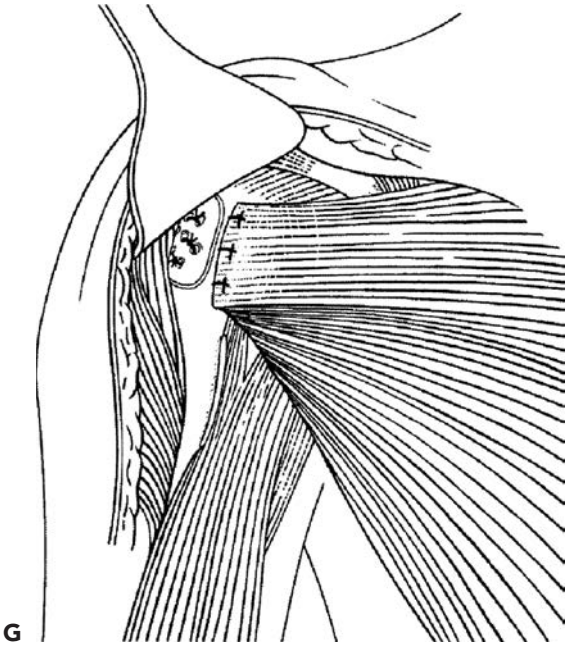
Figure 4-37 Technique of pectoralis major tendon transfer (CG). **(A)** Patient positioning in the beach-chair position. Skin incision for the extended deltopectoral approach is marked. **(B)** A partial repair of the subscapularis is attempted. Detachment of the entire pectoralis major tendon is marked. **(C)** The tendon is sequentially detached from inferior to superior and tagged with nonabsorbable sutures using the modified Mason-Allen technique. **(D)** A superficial trough is prepared at the medial aspect of the greater tuberosity using drill holes for transosseous fixation and the pectoralis major musculotendinous unit is mobilized. **(E)** The pectoralis is transferred over the conjoined tendon and repaired to the bone trough. **(F)** A titanium cortical bone augmentation device is used to reinforce the repair and prevent suture pullout. **(G)** Final repair of pectoralis major tendon. (Reprinted with permission from Jost B, Gerber C. Pectoralis major transfer for subscapularis insufficiency. *Tech Shoulder Elbow Surg* 2004;5:157–164.)



E



F



G

Figure 4-37 (*continued*)

tendon and just medial to the greater tuberosity (Fig. 4-37E). The pectoralis tendon transfer is not repaired to the lesser tuberosity for two reasons: (1) to not disrupt a possible prior repair of residual, native subscapularis tendon, and (2) to place the pectoralis major unit under adequate tension. The transfer is repaired transosseously into a bony trough that is prepared with a high-speed burr, and fixed over a cortical augmentation device (Fig. 4-37F,G).

Following the transfer, the upper portion of the pectoralis major is sutured to the inferior border of the supraspinatus with a running 0-PDS monofilament suture (Ethicon, Johnson & Johnson, Westwood, MA), constituting a partial lateral rotator interval closure.[71]

Closure is always performed over a suction drain that is removed 2 days following surgery.

Postoperative Care

Patients who have undergone an isolated repair of the subscapularis tendon with or without pectoralis major tendon transfer wear a sling for 6 weeks postoperatively. Patients with combined repairs of the subscapularis and supraspinatus are placed into an abduction brace in 45 degrees of abduction for 6 weeks (SOBER Abductor, Pharmap, Crolles, France).

Immediately after surgery, passive range-of-motion exercises are initiated under the supervision of a physical

Figure 4-38 A 54-year-old banker who suffered a complete, irreparable tear of the subscapularis following an injury playing tennis. Preoperatively, he suffered from significant pain and displayed positive lift-off and belly-press signs. Following pectoralis major tendon transfer and partial repair of the inferior portion of the subscapularis tendon, **(A)** he noted significant improvement in pain with an associated decrease in passive and active external rotation, which is common following this procedure. **(B)** Postoperative photograph of a different patient who has undergone pectoralis major transfer of the right shoulder displays an elevated anterior axillary fold, which invariably occurs using this technique. (Reprinted with permission from Jost B, Gerber C. Pectoralis major transfer for subscapularis insufficiency. *Tech Shoulder Elbow Surg* 2004;5:157–164.)

therapist within a range found to be safe at the time of surgery, generally with restricted passive external rotation beyond 10 degrees and no restrictions to forward elevation and internal rotation. Aquatherapy is a standard part of the postoperative regimen, beginning on postoperative day 2. Active range-of-motion exercises are permitted 6 weeks following surgery, and strengthening is allowed at 3 months postoperatively (Fig. 4-38).

Complications

The primary complications that have been reported with tendon transfer for irreparable rotator cuff tears include infection, nerve injury, and rupture of the tendon transfer.

If one reviews all published series on pectoralis major transfer for irreparable anterosuperior rotator cuff tears, the overall complication rate is 10%.[36,42,70,73,77,111,112,138] The most frequent complication was rupture of the tendon transfer, which occurred in 6% of all cases. Treatment of documented tendon rupture consists of immediate revision and repair of the transfer. Infection occurred in 2% of all cases, which is comparable to that found in revision shoulder surgery.

Another, more subtle complication of pectoralis major transfer can include subcoracoid impingement due to static and/or dynamic anterior subluxation of the humeral head following the transfer.[73] The only report of this phenomenon was reported by Jost and Gerber in 3% of their 30 transfers.[70] There have also been reports of transient musculocutaneous nerve injury following subcoracoid transfer of the pectoralis major tendon in 4% of such cases.[36,42,77,111,112]

In cases of latissimus transfer, injury to the axillary nerve may occur during passage of the tendon transfer beneath the deltoid. In these cases, electrodiagnostic studies are helpful in making a diagnosis and a decision of whether to surgically explore and repair the nerve if needed. Furthermore, as described earlier, revision cases are associated with a higher risk of rupture of the tendon transfer and require immediate revision.[134] In rare cases, deltoid dehiscence can occur and should be approached with immediate reconstruction, as delayed repair is associated with significantly worse outcomes.[116]

In Gerber's review of 69 cases of latissimus transfer with an average follow-up of 53 months, no infections were noted, but three patients experienced transient postoperative dysesthesias that resolved spontaneously in 6 months. Two patients required a second surgery for acromioclavicular joint excision. One patient was successfully revised for a postoperative deltoid disinsertion.[48]

CONCLUSIONS

The indications and techniques for tendon transfer in the setting of irreparable rotator cuff tears continue to evolve. An increased understanding of the natural history of irreparable and massive rotator cuff tears as well as the underlying biochemical mechanisms that instigate fatty infiltration, muscle atrophy, and tendon degeneration is helping to guide treatment decisions. Currently, the treatment of irreparable anterosuperior defects with pectoralis major transfer and its modifications results in improved pain and function in a large proportion of patients. The use of latissimus dorsi tendon transfer for the treatment of posterosuperior, irreparable lesions has proven to be an effective method of treatment and is time tested. The

management of combined antero- and posterosuperior defects continues to be a difficult problem, although some recent literature has suggested the benefits of combined pectoralis with latissimus dorsi tendon transfer. The most important element for success is proper patient selection as well as compliance with a strict postoperative rehabilitation regimen. Future studies and surgical advancements will enable the surgeon to better define the indications for tendon transfer based on a more thorough knowledge of response to treatment using these methods.

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5 Complications of Rotator Cuff Surgery

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INTRODUCTION

Rotator cuff pathology represents the most common source of shoulder pain, and acromioplasty the most common surgical procedure of the shoulder. Age-old debates persist regarding intrinsic and extrinsic causes of rotator cuff

TABLE 5-1
CAUSES OF PERSISTENT SHOULDER PAIN AFTER ROTATOR CUFF SURGERY

- Extrinsic shoulder pathology
  - Brachial plexopathy
  - Cervical radiculopathy
  - Long thoracic neuropathy
  - Neoplasm
  - Reflex sympathetic dystrophy
  - Spinal accessory neuropathy
  - Suprascapular neuropathy
  - Thoracic outlet syndrome
- Intrinsic shoulder pathology
  - Intraarticular
    - Adhesive capsulitis
    - Articular cartilage defect
    - Bicipital tendinitis
    - Instability
    - Labral tears
    - Osteoarthritis
  - Extraarticular
    - Acromioclavicular arthropathy
    - Deltoid insufficiency
    - Rotator culf defect
    - Subacromial impingement

From Williams G. Painful shoulder after surgery for rotator cuff disease. *J Am Acad Orthop Surg* 1997;5:97–108, with permission.

TABLE 5-2
INCIDENCE OF POSTSURGICAL COMPLICATIONS FOLLOWING ROTATOR CUFF SURGERY*

Complication	No. of Shoulders (%)
Failed tendon repair	182 (6.2)
Nerve injury	33 (1.1)
Infection	31 (1.1)
Deltoid avulsion	16 (0.5)
Frozen shoulder	16 (0.5)
Suture granuloma	14 (0.5)
Wound hematoma	11 (0.4)
Dislocation	3 (0.1)
Reflex dystrophy	2 (0.1)
Greater tuberosity fracture	1
Acromion fracture	1
Total	310 (10.5)

* Complications reported in 40 series of patients undergoing surgical repairs for rotator cuff tears. The series were published between 1962 and 1995; they included 2948 operated shoulders.
From Mansat P, Cofield RH, Kersten TE, Rowland CM. Complications of rotator cuff repair. *Orthop Clin North Am* 1997;28:205–213, with permission.

disease. However, most authors agree that rotator cuff surgery is generally successful. Rotator cuff surgery is routinely performed in the outpatient setting[43] and generally consists of an anterior acromioplasty, rotator cuff repair, or a combination of both. Recently, debate about the need for acromioplasty has resurfaced. The literature is replete with articles describing the diagnosis and treatment of rotator cuff pathology, espousing new techniques and technology. It is interesting, then, that there is a relative paucity of data regarding complications of such common surgical practices. There are a number of causes of persistent shoulder pain following rotator cuff surgery that are either intrinsic or extrinsic to the shoulder girdle (Table 5-1). Persistent shoulder pain and poor results are not necessarily complications of surgery, just as complications do not always equate to a poor result or persistent pain. The complications discussed in this chapter, although not all-inclusive, are directly related to the surgical procedure and relate to preoperative, intraoperative, and postoperative etiologic factors. These complications include acromial fracture, persistent subacromial impingement, heterotopic ossification, postoperative stiffness, infection, suprascapular nerve injury, axillary nerve injury, deltoid detachment, recurrent rotator cuff tear, and anterosuperior humeral head subluxation.

Complications following rotator cuff surgery are probably underreported or underappreciated. Although several authors have studied the results of operative treatment of failed rotator cuff repairs,[12,49,156] Mansat and colleagues have published the most definitive study regarding complications following rotator cuff repair (Table 5-2).[131] In their series of 116 rotator cuff repairs, the combined medical and surgical complication rate was 38% (44 shoulders). While the surgical complication rate was 33% (38 shoulders), complications that affected the final surgical outcome occurred in 16% of patients (23 complications in 19 shoulders). Complications included failure of tendon healing (17), frozen shoulder (3), deep infection (2), and anterosuperior humeral head dislocation (1). Failure of tendon healing was inferred clinically by an inability to actively flex the arm farther than 120 degrees in the absence of stiffness. The actual rate of recurrent tearing may have been higher if postoperative imaging studies had been obtained. In Mansat et al.'s extensive literature review, as well as their reported study, the rate of revision surgery was approximately 3.5%.

There have been no recent studies specifically addressing the incidence of surgical complications following rotator cuff repair. Most of the studies describe surgical results following reoperation for a particular complication, and the authors are not able to offer knowledge of the true incidence of surgical complications. To a certain extent, each complication has its own incidence and associated etiologic factors. These will be discussed individually in the following sections.

The evaluation of patients with persistent pain and dysfunction following rotator cuff surgery requires a thorough knowledge of the potential postsurgical complications. The most important diagnostic components are history

and physical examination. In addition to obtaining a history from the patient, clinical records and imaging studies from prior surgeries should be reviewed whenever possible. Potential adjunctive studies may include magnetic resonance imaging, ultrasonography, arthrography, computed tomography, scintography, and electrodiagnostic tests. The history, physical findings, and relevance of various adjunctive tests vary according to the complication(s) present.

The treatment options for patients with complications following rotator cuff surgery vary according to the specific complication. Furthermore, multiple complications may coexist in the same patient and, therefore, require combined treatment strategies. In general, the results following treatment of complications of rotator cuff surgery are inferior to the results that would have been obtained following uncomplicated primary rotator cuff surgery. However, when patients are selected carefully and the complications treated appropriately, the symptoms associated with their complications can often be improved.

ACROMIAL STRESS FRACTURE

Etiology and Prevention

Acromial fracture has been reported in association with both open and arthroscopic acromioplasty.[135,138,165] Although the reported incidence of fractures is quite low, the overall incidence is unknown and may be underreported or undetected. Etiologic risk factors that have been identified include osteopenia and overzealous bone resection. The higher prevalence of fracture during arthroscopic acromioplasty is probably related to technical error. The technique of arthroscopic acromioplasty is difficult to master and carries with it a significant learning curve.[4,35,52,121,165] Three-dimensional perception of the direction and depth of bone resection is often difficult to appreciate arthroscopically, when visualizing in a two-dimensional field. This holds true even for experienced surgeons.

It has been stated anecdotally in the literature that removal of greater than 50% of the acromial thickness increases the likelihood of fracture.[138] With this in mind, the risk of acromial stress fracture following arthroscopic acromioplasty can be mitigated by maintaining as much of the thickness of the acromion as possible while still removing the subacromial spur. There is no predetermined amount of bone to resect among all patients with subacromial impingement. Rather, the amount of bone resection will vary depending on the size of the spur as well as the size of the acromion (i.e., patient). Several studies suggest that the amount of bone resection required to relieve abnormal subacromial contact may be rather small.[56,114] There probably exists an optimal range of bone resection that will both relieve impingement and minimize the risk of postoperative acromial fracture. Bone resections on either side of that range may result in poorer results due to either acromial stress fracture or persistent impingement.

Preoperative radiographic assessment of acromial morphology (shape and thickness) should be used to provide an estimate of the amount of bone to be removed intraoperatively. Although the interobserver reliability of the commonly used classification system of acromial morphology (types I, II, and III) has been called into question,[227] the supraspinatus outlet view has been advocated by many authors as the best view to evaluate subacromial spurring.[143,147,148,153] Others have favored a standing anteroposterior (AP) view with 30-degree caudal tilt to assess the anterior prominence of the acromion.[42,180] Since the acromial spur occurs in two planes (anterior and inferior), use of both the outlet and 30-degree caudal tilt views is recommended to provide sufficient preoperative information regarding spur size and acromial thickness. Determination of acromial thickness should also be measured at the junction of the middle and anterior thirds of the acromion, which indicates the true thickness of the acromion. This measurement can then be subtracted from the measured thickness of the spur to provide an estimate of the desired bone resection.

Although Neer cautioned against shortening of the anterior acromion, subsequent authors have noted the importance of the anterior prominence in the impingement syndrome. Rockwood and Lyons[180] formally described the two-step acromioplasty in which any portion of the acromion projecting anterior to the clavicle is removed, followed by removal of the inferior aspect of the acromion. This may be performed with either an osteotome or oscillating saw, followed by feathering of any residual ridge with a burr or nasal rasp. Some authors recommend using only the burr.[138] These techniques are reliable and reproducible with few reported acromial fractures in the literature.[138]

Arthroscopic acromioplasty may be performed via anterior or posterior approaches.[4,35,52,187] While overzealous bone resection may occur during either technique, the mechanism differs depending on which operative portal is used. Techniques that solely use the posterior portal for acromial resection use the undersurface of the posterior acromion as a template to progressively flatten the acromion from posterior to anterior.[35,187] A drawback of this technique is the inability to visualize the thickness of the acromion. This can be overcome by performing the acromioplasty in two stages. Through an anterolateral portal, a full-thickness resection of a small portion of the anterior acromion, determined by extending a line laterally from the anterior cortical border of the clavicle, is performed. The coracoacromial ligament is subperiosteally elevated from the anterior aspect of the acromion, and the hood of the burr is turned toward the deltoid fascia to maintain an intact deltoid origin. This is followed by the posterior cutting block technique, while visualizing the

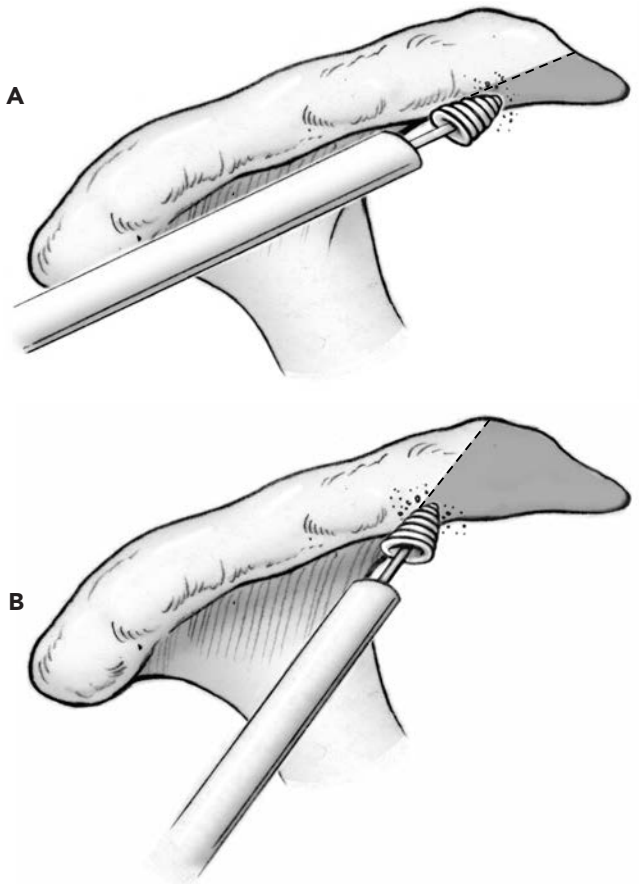


Figure 5-1 **(A)** Arthroscopic acromioplasty via a posterior approach should maintain the burr parallel to the undersurface of the acromion to produce a flat acromial surface, while maintaining maximal thickness. **(B)** If the burr meets the acromion in a convergent manner, the acromion may be amputated or excessively thinned, leaving it prone to stress fracture.

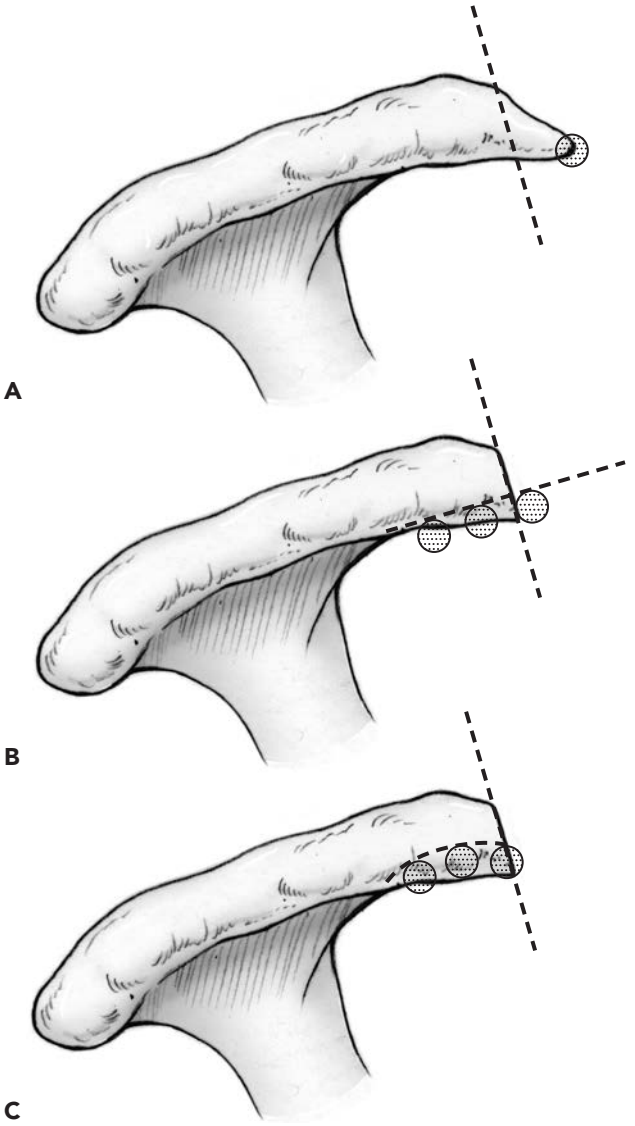


Figure 5-2 Arthroscopic techniques that use an anterolateral portal for bone resection may also result in inappropriate bone resection. **(A)** The initial step involves resection of a small portion of the anterior acromion. **(B)** The remaining acromion is then beveled from anterior to posterior to produce a flat undersurface. **(C)** When the resection is not tapered from anterior to posterior, a dome-shaped acromion will result, which is prone to stress fracture.

thickness of the acromion through the anterolateral portal. The most common error occurs when the surgeon fails to keep the burr parallel to the undersurface of the acromion. When the posterior acromion and arthroscopic burr are convergent, rather than parallel, the anterior acromion may be excessively thinned or amputated (Fig. 5-1). This problem may occur if the surgeon has placed the posterior portal too low, or may be related to an inferior prominence of the posterior acromion. The surgeon can compensate by replacing the instrument through a separate, more parallel portal, or by adjusting the angle or amount of resection accordingly. Under-resection can be easily addressed by making a second pass of the burr while over-resection is not a correctable error. The known diameter of the burr is used to properly measure the depth of the bone resection as well as the thickness of the residual anterior acromion, so that approximately 50% of the thickness remains following acromioplasty.

Acromial resection through a midlateral or anterolateral portal requires that the burr sweep from anterior to posterior. The amount of bone resection, or depth of penetration of the burr, is greatest at the anterior acromial margin and is progressively tapered to zero at the posterior border of the acromioclavicular joint. Failure to taper the resection depth will result in a thinner, dome-shaped acromion that may be prone to stress fracture (Fig. 5-2). Care should also be taken to preserve at least 50% of the thickness of the acromion. We try to accomplish this by performing the acromioplasty in two stages. The first stage is performed as described above. This is followed by beveling of the acromion from anterior to posterior, starting laterally and progressively working toward the medial acromial facet.

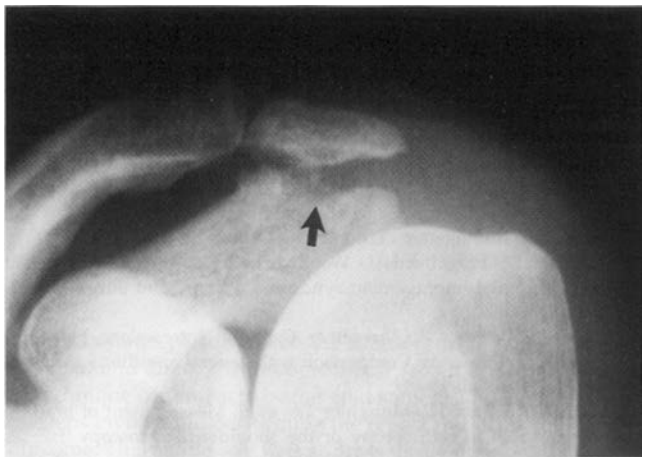


Figure 5-3 Mesacromiale iatrogenesis. (Courtesy of Mark Lazarus, MD.)

Evaluation

Although acromial fracture following anterior acromioplasty occurs either intraoperatively or within the first several months following surgery, there is often a delay in diagnosis due to a low index of suspicion. While the inciting event may be rather minor, the patient will often report a sudden, marked increase in pain, associated with swelling. There will be marked point tenderness over the dorsal aspect of the acromion, corresponding to the fracture site. This typically occurs at the junction of the anterior and middle thirds of the acromion, in line with the posterior border of the clavicle and acromioclavicular joint. The diagnosis is confirmed with routine radiography including an axillary view, a supraspinatus outlet view, and an anteroposterior view with 30-degree caudal tilt (Fig. 5-3).[42,153,180] If these studies are not sufficient, magnetic resonance imaging will confirm the diagnosis.

Treatment

Nonoperative treatment of patients with acromial stress fracture following anterior acromioplasty is generally unsuccessful. Patients frequently develop a painful nonunion as a result of micromotion from deltoid contracture. This is similar to patients with a painful os acromiale. Surgical treatment options include excision, open reduction and internal fixation, and bone grafting.[135,167] The anterior fragment, which has already been partially resected, is often thin and irregular. Under these circumstances, it is usually difficult or impossible to perform adequate internal fixation of the fragment, and excision is preferred. Open excision requires detachment of the deltoid, which is prone to postoperative dehiscence. Careful arthroscopic excision of the fragment may be less likely to lead to dehiscence of the deltoid if the deltotrapezial aponeurosis is left intact at the time of surgery. Open reduction, internal fixation, and possible bone grafting is preferred when the fragment is large enough.

Arthroscopic excision is delayed for 6 to 12 weeks following the initial surgery to allow the fracture and soft tissue envelope to recover from the hemorrhagic stage of the acute injury. The resulting healed fibrous tissue surrounding the fracture site is theoretically less likely to fail after the nonunited anterior fragment has been excised. Arthroscopic excision is performed in a manner analogous to excision of a painful preacromion or mesacromion.[223] The subacromial space is viewed through the posterior portal and the burr is placed through an accessory anterolateral portal. The soft tissue on the undersurface of the acromion is removed using an electrocautery or radiofrequency device, and any remaining coracoacromial ligament is subperiosteally elevated from the anterior aspect of the fragment. The fragment is then removed using a hooded arthroscopic burr, sweeping from the anterior lip of the nonunited fragment to the nonunion site (Fig. 5-4). The resection begins laterally and proceeds medially. Eventually, all that remains is a thin dorsal cortex that can be carefully peeled away from its fascial envelope, leaving an intact sleeve of deltotrapezial aponeurosis that is in continuity with the remaining acromion and the anterior deltoid. If the surgeon feels that he or she risks disruption of the fascia, it is more advisable to leave a small amount of the dorsal periosteum than to penetrate the fascial layer.

Excision of fragments that are larger than a typical meso os acromiale may result in unacceptable deltoid weakness. If the fracture line is posterior to the posterior aspect of the acromioclavicular joint, open reduction and internal fixation should be strongly considered.

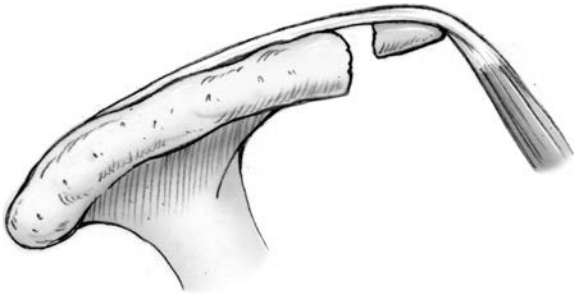
Postoperatively, it is important to emphasize full passive motion exercises, beginning on the day of surgery. An overhead pulley is added at 4 weeks. Active assisted motion is allowed 6 weeks postoperatively. A therapy regimen that is too aggressive risks disruption of the thin deltotrapezial aponeurosis. Subsequent rotator cuff and scapular strengthening exercises are initiated 8 to 12 weeks following surgery.

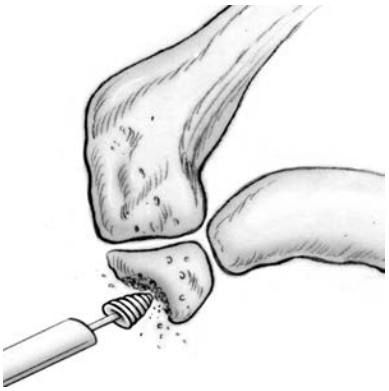
Results following treatment of postoperative acromial stress fracture have been sparsely reported. Excision of the anterior acromion does shorten the anteroposterior dimension of the acromion, thereby compromising the deltoid lever arm. This may potentially lead to weakness in forward elevation, fatigue in overhead activity, and inconsistent pain relief.[135] However, if the deltoid remains intact, an excellent result is possible.

PERSISTENT SUBACROMIAL IMPINGEMENT

Etiology and Prevention

The objective of anterior acromioplasty is to establish a tunnel beneath the anterior acromion and the acromioclavicular





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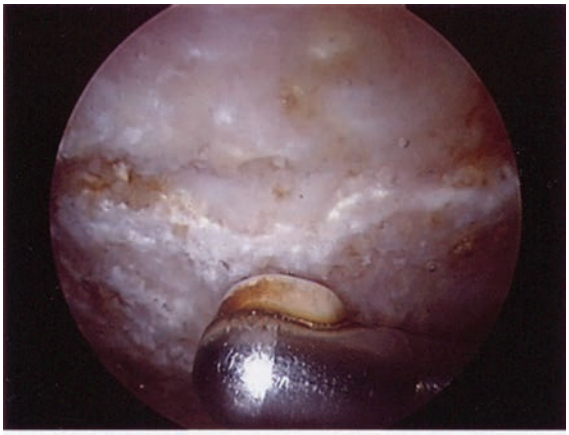

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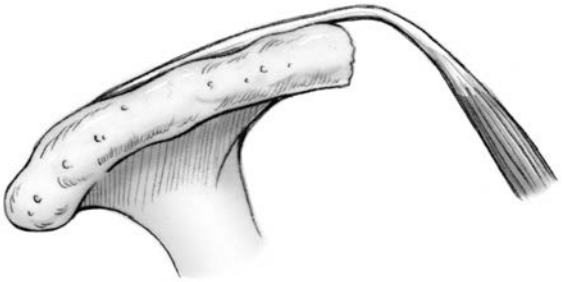

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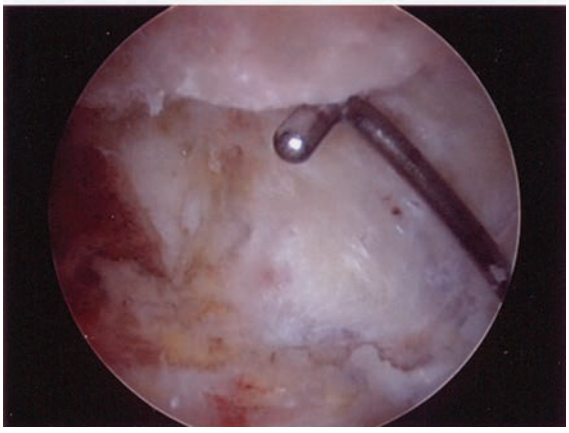

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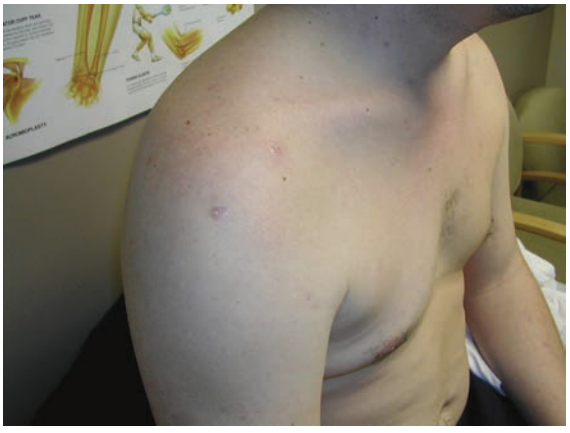

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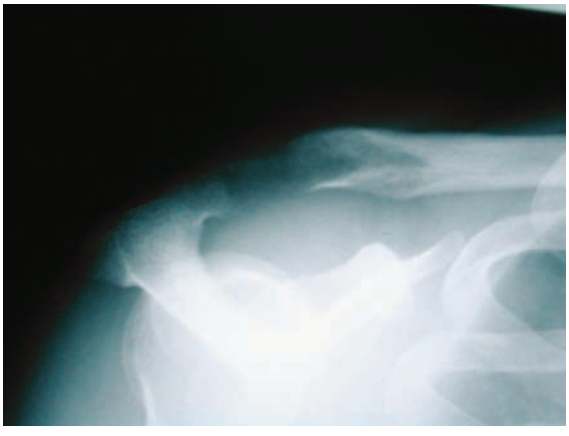


E

Figure 5-4 **(A)** The ununited anterior acromial fragment lies within the anterior deltoid aponeurosis and extends to the posterior border of the acromioclavicular joint. **(B)** Arthroscopic excision begins at the anterior edge of the anterior fragment and extends posteriorly toward the nonunion site, progressing from lateral to medial. **(C,D,E)** Complete excision leaves the deltotrapezial aponeurosis on the dorsal surface of the acromion in continuity with the anterior and middle deltoid. **(F)** Atraumatic excision maintains a normal deltoid contour.

joint, thereby enlarging the supraspinatus outlet and freeing the supraspinatus tendon. Persistent impingement following rotator cuff surgery, therefore, is the result of inadequate supraspinatus outlet decompression. This has been reported in association with residual anterior spurring,[58,161,182] inferiorly projecting acromioclavicular osteophytes,[148] and persistence or regrowth of the coracoacromial ligament.[148,161,182] Persistent impingement has been reported in 18% to 79% of patients with failed acromioplasty.[58,94,161,182] The true incidence of inadequate outlet decompression or persistent impingement following acromioplasty is unknown. Those who do not believe in acromioplasty in the management of rotator cuff disease undoubtedly also doubt the role of persistent impingement as a cause of persistent pain following rotator cuff surgery.

Debate continues regarding surgical indications, technique, and the optimal amount of acromial resection required to relieve supraspinatus outlet narrowing.[63,72,164] It is believed that Sir Reginald Watson-Jones performed the first lateral acromial excision for the treatment of supraspinatus tendon lesions.[6,216] Although several early authors advocated lateral or radical acromionectomy in cases of chronic supraspinatus syndrome,[6,21,83,137] these procedures are associated with significant complications[151] and have been largely abandoned. In 1972, Neer described the impingement syndrome, noting a characteristic ridge of proliferative spurs and excrescences on the undersurface of the anterior acromion.[148] He recommended anterior acromioplasty with removal of a wedge of bone, including the anterior edge and lateral portion of the undersurface of the acromion. While Neer cautioned against shortening of the normal anterior acromion,[147] it is clear that the intended purpose of his procedure was not only to flatten the undersurface of the acromion, but also to remove any anterior projection of the acromial spur.[147–149] Since the transition from the normal acromion to anterior spur is not always apparent, Rockwood and Lyons[180] selected an imaginary line extending laterally from the anterior cortical border of the clavicle and recommended removal of any acromial projection extending past this line. The likelihood of persistent impingement related to residual acromial spurring is minimized when the resection produces a smooth inferior acromial surface, with an anterior edge that approximates the anterior cortical border of the clavicle.[149,180]

The causal relationship between acromial morphology and rotator cuff disease remains elusive. Radiographic, histologic, and cadaveric studies indicate that acromial spurs are actually traction enthesophytes, a response to tension rather than compression.[36,163,196] The shape of the acromion changes with increasing age and in the presence of rotator cuff tearing.[159] While the correlation between type III acromions and the incidence of rotator cuff tears has been called into question, there appears to be a relationship between type III acromions and rotator cuff tear size.[103] Cadaveric studies clearly reveal that contact between the undersurface of the coracoacromial arch and the rotator cuff is a normal phenomenon.[56] Animal studies have demonstrated that abnormal subacromial pressure or prominences will induce an impingement lesion or bursal-sided tearing, but does not seem to result in the articular-sided and intratendinous lesions that are most often seen in clinical practice and microscopic studies.[188,192] Some authors have advocated a subacromial smoothing to remove an area of arch prominence that may create point pressures on the bursal side of the rotator cuff, rather than a nonanatomic flattening of the undersurface of the acromion. The current trend in the literature seems to be toward a more conservative acromioplasty, and in some cases, no acromioplasty.[23,63,72] Therefore, the significance of residual subacromial spurring in the persistence of pain following rotator cuff surgery is difficult to confirm. Practically speaking, if persistent postoperative pain is relieved with subacromial injection and other causes of persistent pain, such as stiffness, have been eliminated, removal of any residual subacromial prominence is reasonable.

Inferiorly projecting acromioclavicular osteophytes may represent a source of persistent impingement pain.[147] This problem is prevented by identifying, and addressing, acromioclavicular pathology at the time of the original surgery.[47] Distal clavicle excision is appropriate in patients who have symptoms related to the acromioclavicular joint. Coplaning of the medial acromial facet and inferior surface of the distal clavicle theoretically removes contact pressure on the rotator cuff, and is based on preoperative imaging studies, intraoperative palpation, and arthroscopic visualization. Technical points that are worth mentioning include preservation of the superior and posterior acromioclavicular ligaments when performing the distal clavicle excision and minimal disruption of the joint when performing distal clavicle coplaning. The inferior acromioclavicular ligament should be preserved in patients without preexisting osteoarthritis, since disruption may lead to a subtle but painful instability. The interested reader is referred to Chapter 32 for a detailed discussion of acromioclavicular joint abnormalities.

Persistence or regeneration of the coracoacromial ligament has been described in the literature and may represent a cause of persistent impingement following subacromial decompression.[84,123,147,161,182] Since the persistent coracoacromial ligament is often associated with a persistent acromial spur,[182] it is difficult to attribute the importance of the ligament to the patient's symptoms. It is important to remember that contact between the rotator cuff and coracoacromial ligament occurs in normal shoulders. In addition, preservation of an intact coracoacromial arch has been increasingly emphasized as a humeral head containment mechanism in rotator cuff–deficient shoulders.[57,122,219] Codman emphasized the importance of

the coracoacromial arch, stating that "evidently, the coracoacromial ligament has an important duty and should not be thoughtlessly divided at any operation." Among patients with an intact or small repairable cuff, resection of a small portion of the ligament is not likely to result in clinically significant anterosuperior subluxation. However, excision of the coracoacromial ligament in patients with an irreparable cuff tear or a large tear that is difficult to repair may increase the risk of postoperative anterosuperior escape of the humeral head.[219] Currently, most authors consider preservation of the coracoacromial ligament and direct repair to the acromion at the time of open repair of large rotator cuff tears. During arthroscopic acromioplasty, we will subperiosteally elevate the ligament from the anterior aspect of the acromion to remove the spur and will allow the origin of the ligament to regenerate.

Evaluation

The diagnosis of persistent subacromial impingement following rotator cuff surgery is made when physical findings associated with subacromial impingement are present, those findings improve substantially following a subacromial injection of local anesthetic (positive impingement test), and there is radiographic evidence of continued supraspinatus outlet narrowing.[42,148,153,180] Patients may complain of night pain and difficulty with overhead activities, and often note little or no postoperative improvement. The impingement sign and reinforcement tests (Hawkins, Jobe) elicit pain and may be associated with subacromial crepitus.[92,93,95,148]

Preoperative range of motion should be carefully evaluated to detect the presence of capsular contracture. Posterior capsular contracture is manifest by decreased internal rotation of the arm in the abducted position (90 degrees of elevation in the scapular plane). This finding is commonly associated with the impingement syndrome and may contribute to continued subacromial impingement following rotator cuff surgery.[132–134]

Supraspinatus outlet views and 30-degree caudal tilt radiographs[17,42,180] should demonstrate evidence of supraspinatus outlet narrowing (Fig. 5-5 and 5-6). Some patients whose postoperative radiographs reveal little or no evidence of continued anatomic narrowing of the supraspinatus outlet may have continued pain and physical findings suggestive of subacromial impingement. Presumably, their persistent symptoms are on the basis of a persistent coracoacromial ligament, subacromial scarring, or some other less obvious problem such as early glenohumeral arthritis. A diagnostic subacromial injection of lidocaine is often very helpful in localizing pain to the subacromial space (assuming the cuff is intact) and is used routinely as a confirmatory test in patients with suspected continued subacromial impingement following rotator cuff surgery.[90]

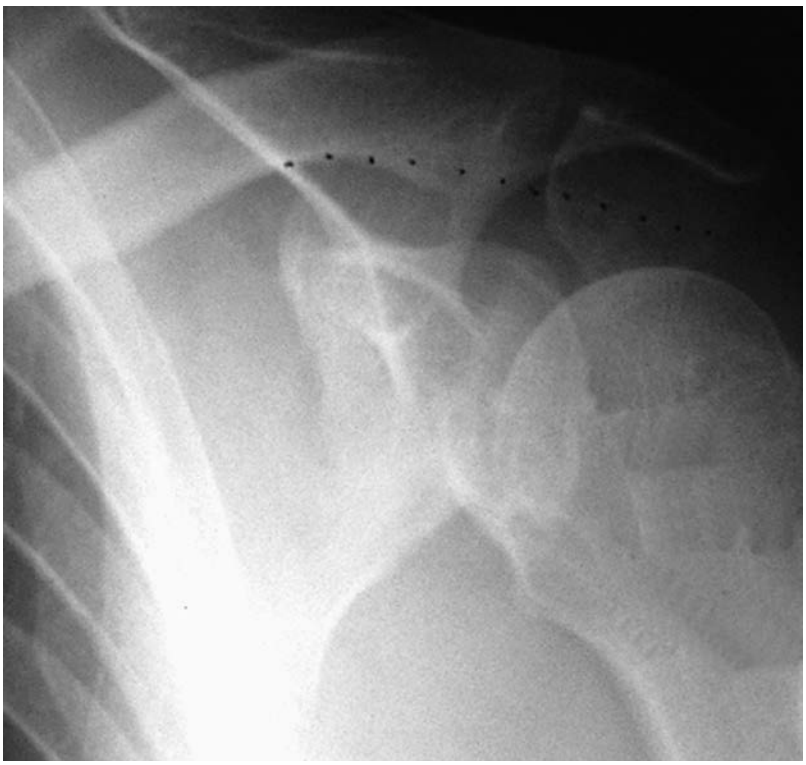


Figure 5-5 A standing anteroposterior radiograph with 30-degree caudal tilt in a patient with persistent pain following previous arthroscopic acromioplasty. Note the persistence of an anterior acromial spur.

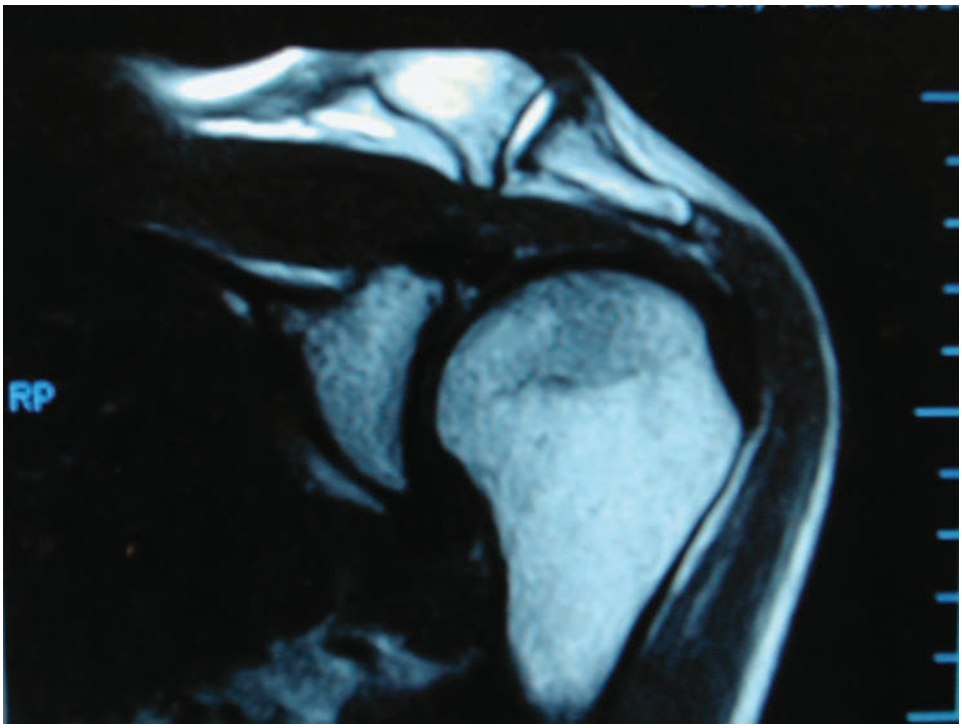


Figure 5-6 A coronal magnetic resonance imaging scan reveals persistent subacromial impingement from an inferior distal clavicular osteophyte.

Treatment

Persistent supraspinatus outlet narrowing from residual acromial spurs, persistent coracoacromial ligament, or inferior acromioclavicular osteophytes may be managed by repeat arthroscopic or open subacromial decompression.[58,90,162,182] The surgical techniques and postoperative rehabilitation for revision acromioplasty do not differ significantly from the analogous primary procedure in a nonoperated shoulder (Fig. 5-7). These techniques are extensively described in Chapters 3 and 39, regarding primary rotator cuff repair and rehabilitation.

The overall results of revision subacromial decompression are inferior to those achieved following primary subacromial decompression.[58,90,161,182] This holds true for both open and arthroscopic procedures. Satisfactory results have been reported in 10% to 75% of patients undergoing revision subacromial decompression.[58,90,161,182] Although some authors advocate open techniques for revision acromioplasty, it is not clear that this offers a more favorable result than arthroscopic surgery. Factors associated with inferior results include open workman's compensation claims and factors associated with secondary gain,[90] postoperative subacromial scarring, unrealistic patient expectations, and unrecognized concomitant pathology. The importance of strict patient selection on the basis of continued radiographic supraspinatus narrowing and positive subacromial injection tests cannot be overemphasized.

HETEROTOPIC OSSIFICATION

Etiology and Prevention

Heterotopic ossification is a poorly understood condition featuring the formation of bone in periarticular regions. Although the pathogenesis is unclear, ectopic bone formation in general is most often related to burns, brain injury, or spinal cord injury. In the shoulder, heterotopic ossification has also been noted following acromial surgery and distal clavicle excision.[6,10,11,83,121,222] The reported incidence of heterotopic ossification following anterior acromioplasty varies between 3% and 30%, and is associated with poorer postoperative results.[6,10,11,83,121,222] While Berg and Ciullo[10,11] noted no significant difference between open and arthroscopic methods, Lazarus and collegues[121] reported a much higher incidence of heterotopic bone formation following arthroscopic acromioplasty (30%) as compared to open acromioplasty (10%).

The etiology of heterotopic ossification following acromioplasty is probably multifactorial. Neer[149] warned against the use of a power burr or handheld rasp because he felt that it would disseminate morselized bone throughout the subacromial space. Some authors who exclusively use the power burr during open acromioplasty have not noted any problems with heterotopic ossification. Lazarus et al.[121] proposed that the use of an arthroscopic burr and pressurized pump forces bone particles into the soft tissues,

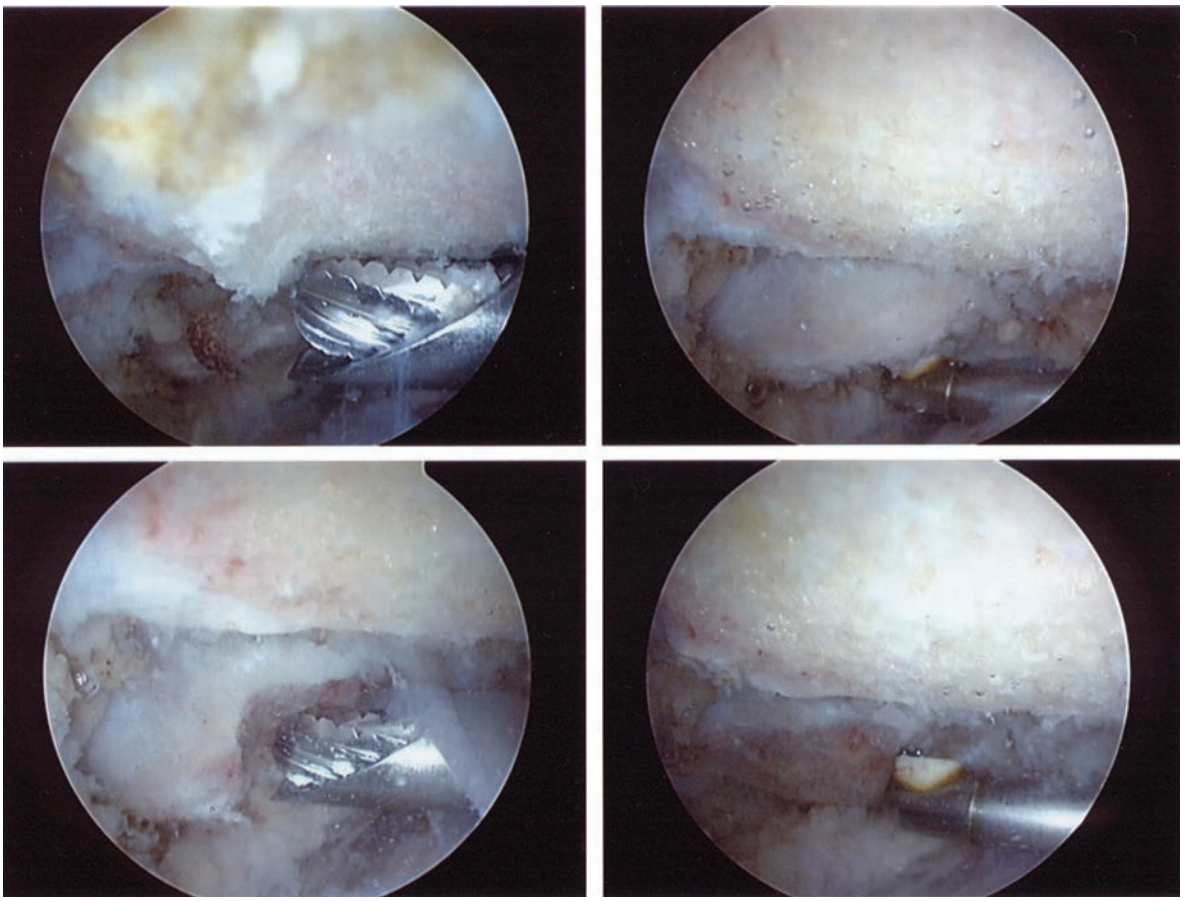


Figure 5-7 A conservative acromioplasty is performed along with resection of the inferior clavicular osteophyte.

increasing the risk of heterotopic bone formation following arthroscopic acromioplasty. However, since immediate postoperative radiographs were not taken, they may have mistaken inadequate bone resection with the formation of heterotopic bone. Berg and Ciullo[10] obtained radiographs within 8 weeks of surgery, noting no evidence of heterotopic bone. The overall incidence of heterotopic ossification in their series was 3.2%, and these authors noted a strong association between postoperative heterotopic ossification and a history of chronic pulmonary disease.[10]

While complete prevention of postoperative heterotopic ossification following open or arthroscopic acromioplasty would be preferable to its treatment, this may not be possible. However, some technical considerations may mitigate the occurrence of this complication. The use of an osteotome during open acromioplasty may produce less bone particulate debris than a power burr or handheld rasp.[149] When the latter instruments are used, copious irrigation is recommended to reduce the amount of bone debris that has been created. During arthroscopic acromioplasty, frequent use of the suction attachment on the burr will minimize the spread of bone particles into the subacromial space. The subacromial space should be carefully inspected at the completion of the procedure, and any visible bone should be removed using the suction. Berg and Ciullo[10] have recommended prophylactic treatment with indomethacin or radiation among patients with a history of ankylosing spondylitis, hypertrophic pulmonary osteoarthropathy, chronic pulmonary disease, smoking, or hypertrophic arthritis.

Evaluation

Recurrent pain attributable to heterotopic ossification will usually develop within 3 to 6 months following surgery. Heterotopic ossification most commonly occurs in the subacromial space, within the acromioclavicular interval, or at the deltoid attachment site. The impingement sign, impingement test, and impingement reinforcement tests are frequently positive,[92,93,148] and passive arcs of motion are usually not significantly limited.[10,121] Severe periarticular heterotopic ossification occurs rarely and is associated with a dramatic loss of passive glenohumeral motion, resembling a frozen shoulder. Erythema and warmth may be present, mimicking infection. Occult infection may provoke the development of ectopic bone and should be carefully investigated.

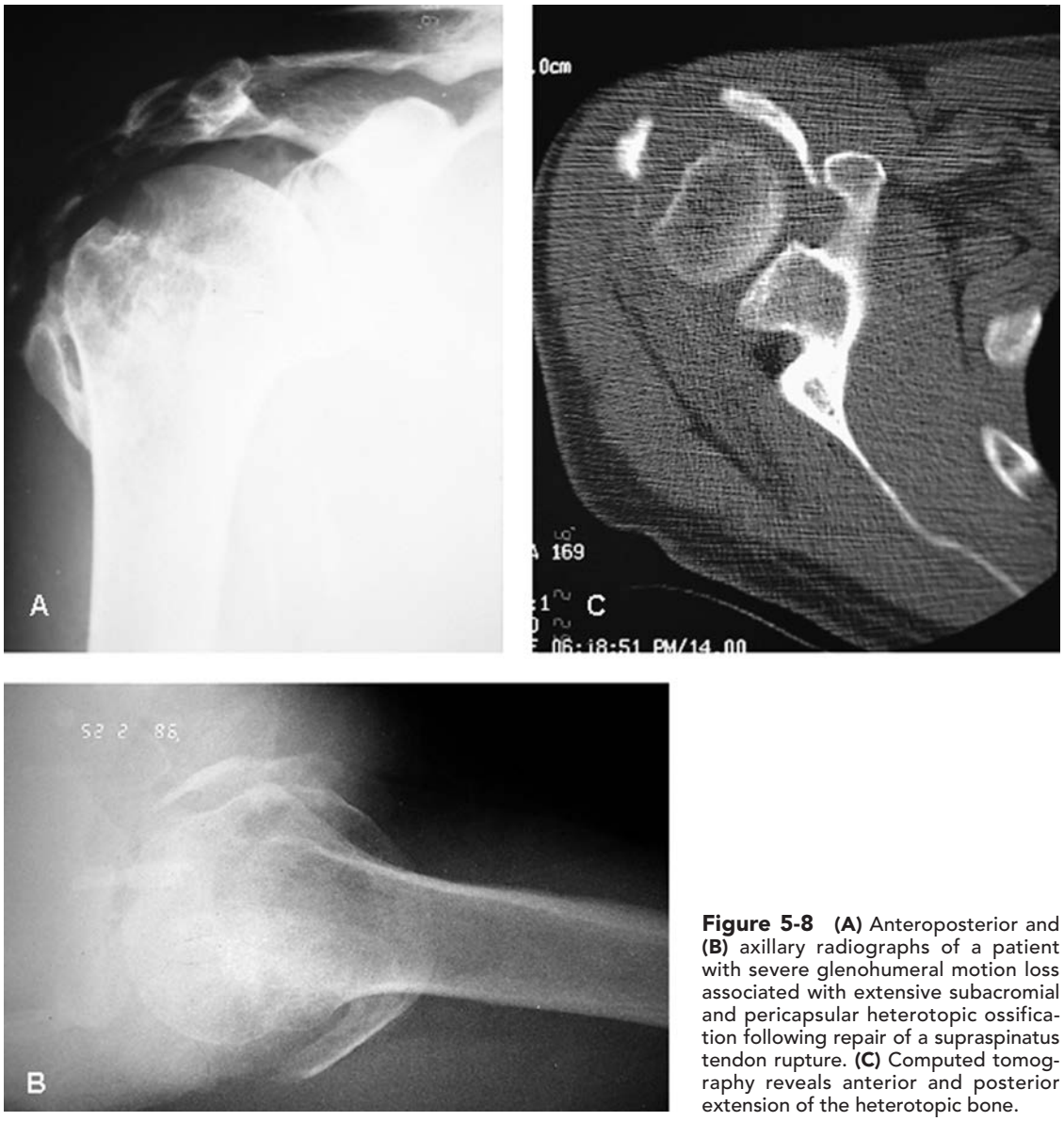


Figure 5-8 **(A)** Anteroposterior and **(B)** axillary radiographs of a patient with severe glenohumeral motion loss associated with extensive subacromial and pericapsular heterotopic ossification following repair of a supraspinatus tendon rupture. **(C)** Computed tomography reveals anterior and posterior extension of the heterotopic bone.

The classification system of Booker and colleagues has not been extensively applied to the condition in the shoulder. Radiographic analysis, including anteroposterior, axillary, supraspinatus outlet, Zanca, and 30-degree caudal tilt views, will facilitate the diagnosis.[42,153,180] Computed tomography may be helpful in identifying multiple sites of heterotopic bone and may assist in preoperative planning (Fig. 5-8).

Treatment

Once the process of heterotopic ossification has begun, it is doubtful whether any of the available preventative measures can affect the outcome. Experience with this problem about the shoulder is limited and recommendations are based on the reported experience and treatment of heterotopic bone formation about other joints. A major concern relates to the timing of surgical intervention, with some authors indicating that the arbitrary 12- to 18-month delay may be unnecessary.[136,177] They suggest that the heterotopic bone associated with a central nervous system injury behaves differently than that seen in postoperative patients.[178] Surgical treatment is performed as early as 3 to 4 months, at which time the heterotopic bone often has a well-defined margin and trabecular pattern on standard radiographs.[177] It does not appear to be necessary to wait for the radionucleotide bone scan or serum alkaline phosphatase level to normalize.[136] Early excision of heterotopic bone in the elbow has offered significant benefits and yielded favorable results.[136] Advantages include the ability to more easily peel away immature bone from the tissue planes as well as accelerated functional recovery. Early

restoration of motion may provide beneficial effects to the articular cartilage, and may help prevent secondary soft tissue contracture and muscular atrophy, hopefully maximizing functional recovery.[136,178]

The treatment of heterotopic ossification following rotator cuff surgery is based on the location and severity of the process. Prior to removal, the precise distribution of bone, the optimal surgical approaches, and the potential anatomic hazards should be reviewed. Isolated deposits within the subacromial space may be débrided by open or arthroscopic methods. Open débridement requires detachment of the deltoid but may be associated with a lower recurrence rate and allow a more thorough excision than arthroscopic débridement.[121] Extensive pericapsular ossification requires an open approach, which is directed toward the location of the largest amount of bone. It usually occurs anteriorly and is accessed through a deltopectoral approach. When possible, excision is preferred through a muscle splitting rather than muscle detaching approach. Anterior and inferior pericapsular bone is excised through a subscapularis splitting incision. When this is not possible, the subscapularis is divided 1 to 2 cm medial to its insertion and reflected medially to expose the heterotopic bone. Heterotopic bone within the superior capsule and supraspinatus can then be excised by sharply dividing the interval between the supraspinatus and the superior capsule. Posterior heterotopic bone may require a second, more posterior incision for adequate exposure. Single low-dose radiation or a 6-week course of indomethicin[10] is administered postoperatively to prevent recurrence.

FROZEN SHOULDER

Etiology and Prevention

The literature currently lacks a standardized definition of frozen shoulder in the postoperative setting. Therefore, the incidence of postoperative stiffness following rotator cuff surgery is unknown. However, there is generalized agreement that the hallmark of postoperative capsular contracture involves a commensurate decrease in both active and passive arcs of motion, which can involve one or more planes of motion. This motion loss is asymmetric in comparison to the contralateral, uninvolved shoulder. In our clinical practice, we have defined this as a 20-degree loss of passive motion in any plane as compared to the opposite side. Symptomatic loss of motion following rotator cuff surgery may be attributed to a variety of etiologies including preexisting medical conditions, surgical technique, and postoperative rehabilitation. The likelihood of developing postoperative stiffness is highest in patients who exhibit significant capsular contracture preoperatively.[149] Patients with comorbid conditions known to be associated with adhesive capsulitis, including diabetes mellitus and hypothyroidism,[155,213] are also at increased risk of developing postoperative stiffness.

Frozen shoulder that occurs following rotator cuff surgery is caused by a combination of capsular contracture and extracapsular scarring. Capsular involvement may be localized or generalized. Localized posterior capsular contracture is commonly associated with the subacromial impingement syndrome and rotator cuff disease.[132–134] Failure to address this through preoperative stretching, or intraoperative capsular release, will result in persistent postoperative stiffness, pain, and dysfunction. Generalized capsular contracture develops when there exists an element of preoperative generalized adhesive capsulitis, following prolonged postoperative immobilization, or in association with predisposing factors such as diabetes mellitus, hypothyroidism, or occult glenohumeral arthritis.

Extracapsular adhesions may develop in the humeroscapular motion interface following rotator cuff surgery, interfering with normal shoulder function and motion.[186] During normal shoulder motion, the upper proximal humerus and rotator cuff slide beneath the smooth undersurface of the coracoacromial arch, deltoid, coracoid process, and conjoined tendons. Postoperative adhesions may form in this interface, especially between the raw cancellous undersurface of the acromion and the rotator cuff.[142,149] The subdeltoid and subacromial adhesions capture the deltoid and humeral head, altering the normal biomechanics of the glenohumeral joint. Subdeltoid adhesions may effectively tenodese the deltoid, requiring a greater effort from the supraspinatus tendon to achieve shoulder abduction. Iatrogenic tightening of the rotator cuff interval and overadvancement of the rotator cuff tendon are examples of operative techniques that may lead to loss of motion by capturing the shoulder.[210]

In general, rotator cuff surgery should be avoided in patients with evidence of a generalized frozen shoulder.[132,148,149,180] Capsular contracture should be addressed through preoperative capsular stretching exercises. In cases of recalcitrant stiffness, consideration may be given to closed manipulation or arthroscopic release,[169,213] prior to performing rotator cuff surgery. An alternative approach in smaller rotator cuff tears associated with stiffness is to combine capsular release with cuff repair. Under this scenario, rehabilitation should be directed primarily at the frozen shoulder, realizing that there might be a greater chance of cuff rerupture.

Evaluation

A careful history of the patient with suspected postoperative frozen shoulder may reveal the presence of predisposing risk factors. Since the hallmark of frozen shoulder is a symmetric decrease in both active and passive arcs of motion,[154,155,158,213,214] motion should be evaluated in all

planes and compared to the contralateral, normal shoulder. While this can be performed in the sitting or supine positions, some patients are better able to relax in the supine position with gravity eliminated.

The clinical manifestation of capsular contracture depends on which portion of the capsule is affected. An isolated rotator interval contracture will limit external rotation with the arm at the side, while having less of an effect on rotational motion with the arm at 90 degrees of scapular elevation.[89] Posterior capsular contracture will result in decreased terminal elevation, cross-body adduction, internal rotation behind the back, and decreased internal rotation with the arm at 90 degrees of elevation in the scapular plane.[132] Generalized capsular contracture will result in a global loss of motion.

Patients with acute synovial inflammation may be particularly difficult to examine. This obstacle may be mitigated with an intraarticular injection of lidocaine (with or without cortisone). The injection may be both diagnostic and therapeutic, and should facilitate the examination. Other patients with secondary gain may demonstrate subjective complaints out of proportion to their expected objective findings. While examination under anesthesia represents a reliable method of determining the presence or absence of true capsular contracture, some simple measures may be applied to the conscious patient. When passive supine external rotation at the side equals, or approximates, the opposite side, no superior capsular contracture is present. The arm is then brought to 90 degrees in the scapular plane and passive internal and external rotation is measured. No inferior capsular contracture exists when this arc approximates the opposite side. If the arm cannot be passively forward elevated beyond 90 degrees under these circumstances, the cause is likely to be voluntary guarding, rather than contracture.

Radiographic evaluation of patients with frozen shoulder is usually normal, although it should be performed to exclude the presence of pericapsular heterotopic ossification or glenohumeral arthritis. While arthrography has been recommended in the past, it is not necessary to make the correct diagnosis of frozen shoulder. Magnetic resonance imaging does not assist in making the diagnosis of frozen shoulder, but may be indicated when the patient has regained full motion and continues to complain of persistent pain.

Treatment

Some loss of motion following repair of a large rotator cuff tear may be inevitable due to loss of tendon tissue during local transplantation of the tendon. The acceptable amount of passive motion loss associated with acceptable results has not been defined in the literature, but some patients are obviously not bothered by painless loss of motion, provided that function and strength are improved. Symptomatic motion loss following surgery is frequently related to dense adhesions in the humeroscapular motion interface,[142] as well as capsular contracture.[213,214] Nonsurgical joint mobilization techniques are continued for 3 to 6 months, provided that the patient continues to demonstrate improvement. The judicious use of intraarticular steroid injections will usually provide significant pain relief and facilitate participation in the therapy program. However, when postoperative stiffness occurs following rotator cuff repair, the merits of intraarticular steroid placement must be balanced against the possibility of delayed tendon healing and tendon damage.[19,203,218]

Closed manipulation alone is often unsuccessful in patients with postoperative shoulder stiffness, but may be attempted. Arthroscopic capsular release has been shown to be a successful technique in regaining motion in shoulders that are recalcitrant to nonsurgical measures or closed manipulation.[87,214] The technique is somewhat of a misnomer in that the authors address both the capsular and extracapsular sources of shoulder stiffness at the time of surgery.[210,214] The arthroscopic sheath and blunt obturator are placed into the subacromial space through a posterior or posterolateral portal. An attempt is made to pass the sheath lateral to the tuberosity and into the lateral subdeltoid recess. If this is unsuccessful, the arthroscope is placed and an accessory anterolateral portal is established, through which an arthroscopic resector or radiofrequency device is placed. The dense adhesions in the humeroscapular interface are then resected with careful attention directed at avoiding damage to the rotator cuff, deltoid, or axillary nerve. The resection is complete when the arthroscope can pass freely from the subacromial space, over the greater tuberosity, into the lateral subdeltoid recess (Fig. 5-9). Adhesions between the coracohumeral ligament and underlying rotator cuff are débrided to the level of the coracoid process, and any anterior adhesions between the subscapularis and deltoid are also débrided.

The arthroscope is then placed into the glenohumeral joint through the standard posterior portal. Release of the capsular structures is then performed according to the preoperative evaluation. These techniques are described extensively in Chapter 17 on the diagnosis and management of the stiff shoulder. Open releases are indicated in patients who have failed arthroscopic release or have had iatrogenic tightening of tendinous structures.

Several authors have reported excellent recovery of motion following arthroscopic treatment of postoperative stiffness. However, despite improvements in motion, pain relief and functional improvement are generally less favorable than in patients with idiopathic frozen shoulder and patients who did not develop postoperative stiffness.[67,105,214] Therefore, caution should be exercised in predicting the outcome of arthroscopic release among patients with postoperative frozen shoulder, and patients should be counseled regarding the possibility of persistent pain despite the successful return of motion.

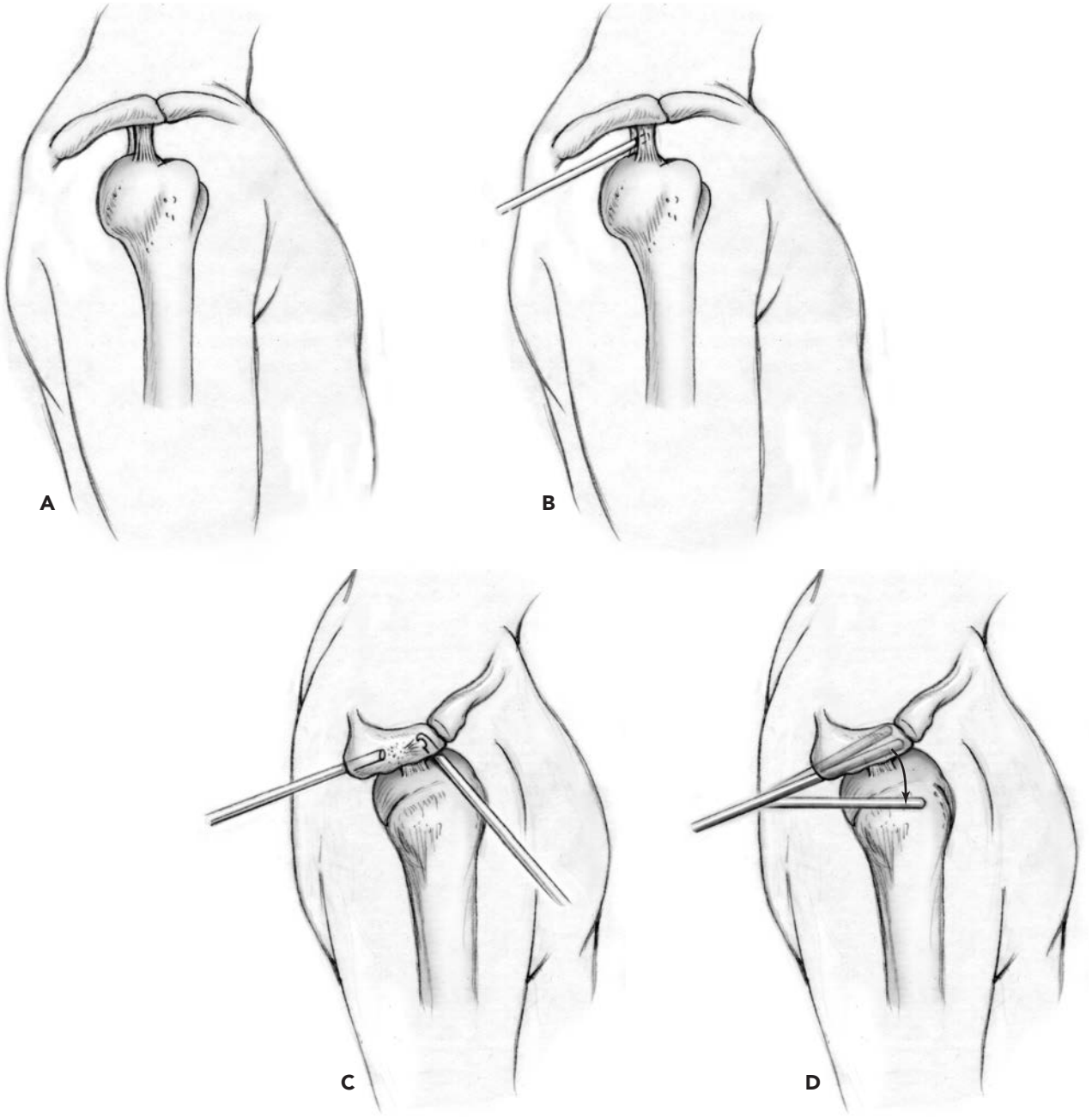


Figure 5-9 Frozen shoulder following previous rotator cuff surgery is often characterized by dense adhesions between the undersurface of the acromion and the superficial surface of the rotator cuff **(A).** A blunt obturator placed into the subacromial space is prevented from moving over the humeral head into the subdeltoid bursa by the adhesions **(B).** After the adhesions have been resected and the subacromial space has been reestablished **(C),** the blunt obturator can easily pass from the subacromial space into the lateral subdeltoid recess and back again **(D).**

INFECTION

Etiology and Prevention

Deep infection following rotator cuff surgery is relatively uncommon, but represents a potentially devastating complication in terms of functional outcome. The incidence of infection following shoulder arthroscopy has been reported to be approximately 0.4%,[7,13,199] while that of open and mini-open rotator cuff repair has been reported to range between 0.27% and 1.9%.[98,131,195] Risk factors may include patient age, preexisting medical conditions such as hypothyroidism and diabetes mellitus, local irradiation, smoking and alcohol intake, instrument sterilization problems, and

increased operative times.[37,141] It is not clear whether steroid injections administered prior to surgery may increase the risk of infection.[102] A low index of suspicion, related to the infrequency of this complication, often leads to a delay in diagnosis.[97,141] Deceptively innocuous-looking wound problems may mask soft tissue loss, rotator cuff and deltoid dehiscence, and osteomyelitis. Extensive soft tissue destruction and a delay in diagnosis are both associated with a worse prognosis. While aggressive surgical débridement, combined with soft tissue coverage and intravenous antibiotics, will usually control the infection, permanent functional deficits frequently persist.[97,141] Obviously, prevention of this complication is preferred to treatment.[48,189]

The use of preoperative antibiotic prophylaxis is strongly recommended in the prevention of postoperative infection.[48,85,220] Although overuse of antibiotics is directly related to the development of resistant bacterial strains, the treatment costs and clinical morbidity associated with an infection following rotator cuff repair support the use of prophylactic antibiotics. The most commonly reported offending pathogens are *Staphylococcus aureus*, coagulase-negative *Staphylococcus*, and *Propionibacter acnes* species.[98,120,141] Due to their typical susceptibilities, the most common prophylactic antibiotic is a first-generation cephalosporin such as cefazolin.[189] With the emergence of methicillin-resistant *Staphylococcus aureus* as a community-acquired pathogen, Bactrim DS may be added to the prophylactic preoperative regimen when placement of an implant is planned.

Since most of the infecting organisms are present in normal skin flora, careful skin scrubbing, preparation, and draping may prevent contamination. Axillary hair should either be shaved prior to Betadine scrubbing or sealed off from the operative site during draping. Impermeable drapes and stockings should be used during arthroscopic procedures. Arthroscopic instruments should be either autoclaved or soaked in warm 2% glutaraldehyde for 20 minutes.[189] Operating time and operating room traffic should be minimized. Intraoperative conversion from arthroscopic to open methods may be associated with an increased risk of infection. Herrera and colleagues[98] reported a 1.9% infection rate (seven patients) following arthroscopic subacromial decompression and mini-open rotator cuff repairs. They were able to reduce this rate to zero by changing surgical gloves, applying a second preparation of Betadine, and placing a new extremity drape at the time of mini-open repair.

Evaluation

Shoulder infections are rarely diagnosed acutely. This is often the consequence of subtle physical findings and a low index of suspicion.[97,141] Following arthroscopic procedures, infection is usually manifest by a low-grade fever, as well as erythema and prolonged drainage from one or more of the portal sites.[120] Patients will complain of progressively increasing pain and a change in the nature of the drainage from a thin, serous fluid to a thick, yellow exudate. A white blood cell count obtained in the early postoperative period may remain within the normal range. While the diagnosis is confirmed by a positive culture aspirate from the portal site or subacromial space, a negative culture does not exclude the possibility of an infection, especially if antibiotics were administered prophylactically at the time of surgery or in the postoperative period.

The clinical symptoms of infection following open rotator cuff surgery may vary from pain, swelling, and erythema to wound dehiscence, drainage, general malaise, fever, and leukocytosis.[141] Untreated infections may develop a draining sinus or synovial-cutaneous fistula.[207] Patients who present to their postoperative visit (7 to 10 days) with an erythematous wound and the appearance of a subcutaneous hematoma should be suspected of having a deltoid detachment, deep wound infection, or both. Hematoma formation is a reasonable diagnosis when the fluid collection occurs within the first 24 to 48 hours following surgery and is not accompanied by erythema. Aspiration of a hematoma should produce organizing clot, while the expression of serosanguineous fluid is more likely to represent an infection. The diagnosis of infection is confirmed by a positive culture of the aspirate. It should be emphasized that the growth of *Propionibacter* species is not necessarily a contaminant and must be considered an infecting organism.[98,141]

Routine radiographs may reveal soft tissue swelling. The presence of subacromial air at 7 to 10 days postoperatively may indicate the presence of a gas-forming organism. Magnetic resonance imaging during the immediate postoperative period will be of limited value due to postoperative artifact. In addition, the presence of deltoid or rotator cuff deficiency will be identified at the time of surgical débridement of the infection. In the chronic setting, magnetic resonance imaging as well as scintigraphy may be of value in identifying the presence of osteomyelitis.

Treatment

Deep infection following rotator cuff surgery is uncommon, with relatively few reports in the literature addressing the management of this complication.[97] The most important factors in effectively treating deep infections are a high index of suspicion, early diagnosis, and aggressive surgical treatment.[98,141] There is a tendency to treat patients with postoperative wound problems (such as mild erythema, drainage, or late hematoma formation) with oral antibiotics. While these methods may occasionally be successful, the preferred management of a deep, postoperative wound infection is surgical drainage and débridement.

Early infection (within 4 weeks of surgery) following arthroscopic acromioplasty may be adequately treated by arthroscopic irrigation and débridement. The subacromial

space should be aggressively débrided prior to entering the glenohumeral joint. The subacromial space often contains loculations of infected tissue, which may be débrided with an aggressive arthroscopic resector. Copious amounts of irrigant should then be allowed to flow through the subacromial space to decrease the bacterial load. Once the subacromial space has been adequately débrided and irrigated, the arthroscope is placed in the glenohumeral joint, and the joint is irrigated with 3 to 6 L of antibiotic-impregnated irrigant. It is helpful to establish an outflow portal to facilitate flow of the fluid through the joint. One drawback of the arthroscopic approach is difficulty in adequately débriding the biceps tendon sheath. The biceps tendon should be grasped intraarticularly and pulled into the joint to break up any infected adhesions in the tendon sheath.

Open irrigation and débridement is preferred for infections following open or mini-open rotator cuff surgery, allowing inspection of the deltoid repair as well as direct access to the biceps tendon sheath. Serial débridements may be required.[98] If the deltoid repair is intact, the deltoid is split in line with the previous split, and the subacromial space is inspected. If the rotator cuff is intact, and the loculations within the subacromial space can be adequately débrided, the procedure is performed without detachment of the deltoid repair.

When the deltoid repair has failed, or the subacromial space cannot be adequately débrided, the deltoid is released and retracted anteriorly to access the subacromial space. Once the necrotic debris has been removed and the subacromial space has been irrigated, the rotator cuff is inspected. If the rotator cuff repair is intact, the rotator cuff and sutures are left in place. Small incisions are then made in the rotator interval and biceps tendon sheath to permit irrigation of the glenohumeral joint.

When the rotator cuff repair is disrupted, the necrotic tendon edges are débrided and all visible suture and suture-anchoring material is removed.[120] The revision rotator cuff repair is delayed until the time of the final irrigation and débridement, or until the infection has cleared (6 weeks). However, if the tendon defect is particularly large, consideration should be given to early repair at the time of initial or secondary débridements. In all cases, the deltoid should be securely repaired to the bone of the acromion at the time of the final débridement. Unrepairable rotator cuff defects may be associated with persistent synoviocutaneous fistula formation. I (BDC) have had anecdotal success in treating such cases with aggressive débridement, sinus tract excision, deltoid repair, partial rotator cuff repair, and placement of a patch graft (Restore Patch, Depuy Orthopedics, Inc. Warsaw, IN). Other authors have described muscle transfers to "seal off" the joint.

Antibiotics are withheld until intraoperative cultures have been obtained. Broad-spectrum antibiotic coverage for skin organisms such as *Staphylococcus aureus* and *Propionibacter acnes* is then administered. This usually includes vancomycin or a first-generation cephalosporin such as cefazolin. The antibiotics are changed appropriately as indicated by the culture sensitivities. The duration of antibiotic coverage is individualized, but generally includes 1 to 4 weeks of intravenous treatment, followed by 1 to 4 weeks of oral treatment. If there is any suspicion of osteomyelitis, antibiotic treatment may continue for 6 weeks.

Neglected infections following rotator cuff surgery often result in a long interval (i.e., months) between the index procedure and subsequent surgical intervention.[97,141] These late or chronic infections often present with draining sinus tracts, synoviocutaneous fistulas, severe soft tissue loss, or exposed bone (humeral head or distal clavicle). Complete eradication of the infection requires radical débridement of all necrotic debris, excision of synovial tracts, removal of all retained suture or suture-anchoring devices, and aggressive débridement of any affected bone. Multiple débridements are often necessary, and the rotator cuff is left unrepaired until the infection has been successfully cleared. Vascularized rotational myocutaneous flaps from the latissimus dorsi or pectoralis major muscles may be required to facilitate wound closure and restore the soft tissue envelope.[86,97,141] Intraoperative specimens of fluid, soft tissue, and bone shavings should be sent for aerobic, anaerobic, fungus, and acid-fast bacillus culture. Parenteral antibiotics are continued for at least 6 weeks following débridement.

Postoperative rehabilitation includes immediate passive mobilization in all cases. In patients who require a revision deltoid or rotator cuff repair, an abduction brace is used to protect the repair for 3 to 4 weeks. The brace is removed only to allow passive motion exercises several times daily. Active assisted range of motion is allowed 6 weeks postoperatively and strengthening is initiated 8 to 12 weeks following surgery.

Patients with an intact rotator cuff and deltoid at the time of débridement are placed in a postoperative sling. If the index and revision surgeries were performed arthroscopically, and did not include a rotator cuff repair, active motion and strengthening are allowed according to the patient's symptoms. If either surgery included a rotator cuff or deltoid repair, passive motion exercises are performed for 6 weeks. Thereafter, active motion and strengthening exercises are added.

Postoperative wound infection following rotator cuff surgery clearly has a negative effect on overall outcome.[97,120,141] The clinical results are directly related to timely intervention, with a delay in treatment resulting in the worst outcome. All patients, regardless of rotator cuff or deltoid integrity, seem to have more pain than their counterparts who have not been infected. The pain is usually not disabling, nor is it usually severe enough to require analgesics. Early intervention will frequently facilitate a successful deltoid and rotator cuff repair. Most patients who ultimately possess an intact deltoid and rotator cuff demonstrate good overhead function. Even patients with

an intact deltoid and a small rotator cuff defect may possess overhead function, although they may demonstrate some degree of fatigue. Overall, the clinical outcome of this complication is not as poor as those reported for spontaneous septic arthritis of the shoulder in adults.[65,116]

DELTOID DETACHMENT

Etiology and Prevention

Detachment of the deltoid origin represents a potentially devastating complication of rotator cuff surgery.[12,49,80,151,156,197] To our knowledge, it has only been reported following open and mini-open rotator cuff surgery, but in our clinical practice we have seen deltoid detachment occur as a result of arthroscopic rotator cuff surgery. Postoperative deltoid detachment may occur as the result of poor intraoperative technique, overzealous acromial excision, postoperative infection, and inappropriate postoperative physiotherapy.[80,151,156,197] Poor prognostic factors include lateral acromionectomy, involvement of the middle deltoid, and a recurrent or massive rotator cuff tear.[12,151,197]

Anatomic and histologic studies clearly indicate a direct attachment of the tendon to the anterior and lateral acromion, rendering the release of deltoid fibers inevitable during any method of acromioplasty.[112] Arthroscopic acromioplasty, which removes 4 or 6 mm of bone, will release 43% and 72% of the deltoid origin, respectively.[206] Although some detachment of the anterior deltoid fibers occurs during arthroscopic acromioplasty, functional compromise has not been shown in clinical studies. Several authors have described arthroscopic excision of a mesoacromion. This procedure releases all of the anterior and lateral tendinous attachments of the deltoid and emphasizes preservation of the superior deltoid fascia. Again, functional compromise has not borne out in these clinical studies. Other authors have shown regrowth of the coracoacromial ligament following subperiosteal release of the ligament during arthroscopic acromioplasty. Fibrous reattachment of the released deltoid fibers has not been studied but remains an area of further investigation.

The fibers of the middle deltoid are arranged perpendicular to the acromion, while those of the anterior deltoid arise obliquely from the anterior acromion. Conservative deltoid splitting approaches exploit this interval by dividing the tendinous raphe between the middle and anterior thirds of the deltoid (Fig. 5-10). This may result in fewer deltoid detachments and less tendinous retraction when deltoid detachment does occur.[132,138,198] Deltoid releasing approaches should extend onto the dorsal surface of the acromion to ensure a strong distal musculocutaneous cuff of tissue for reattachment to bone.[180] While all attempts should be made to avoid disruption of the middle deltoid,

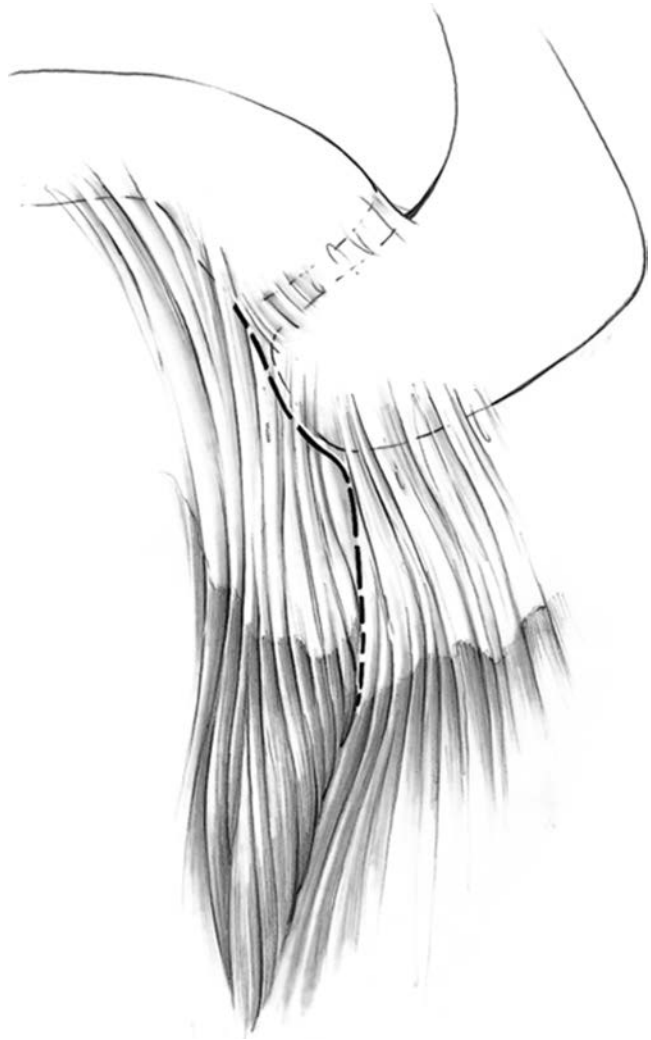


Figure 5-10 The anterior deltoid fibers arise obliquely from the anterior acromion, and the middle deltoid fibers arise perpendicularly from the lateral acromion. Incisions that exploit this interval may be less likely to result in postoperative deltoid detachment and retraction.

this may not be possible when attempting to repair a large or massive rotator cuff tear that extends into the infraspinatus or teres minor tendons.[14] We have not found detachment of the middle deltoid to be necessary during open rotator cuff repair. However, if more posterior exposure than is attainable without middle deltoid detachment is required, the middle deltoid should be released and reattached in a similar fashion to the anterior deltoid origin. Repair of the deltoid origin includes identifying and incorporating the deep deltoid fascia, which has a tendency to retract distally when released. The deltoid split should be closed with tendon-to-tendon sutures, and the deltoid origin should be repaired to bone or to an adequate soft-tissue cuff using nonabsorbable sutures.

Deltoid detachment that occurs in the setting of prior lateral or radical acromionectomy represents an extremely

disabling and often irretrievable condition.[12,151,180,197] While good results have been described following radical acromionectomy and acromion excision,[6,21,83,137] these procedures are clearly not required to relieve impingement,[56,148] and fortunately have been largely abandoned.

As with any repaired tendon, the deltoid should be protected postoperatively. Acromioplasty by any method theoretically weakens the deltoid origin[112,206] and the degree of postoperative protection is dependent on the surgical procedure as well as the quality of repair. Following arthroscopic acromioplasty, we allow activities of daily living immediately, but restrict lifting to the weight of the arm for 2 to 4 weeks. Following open acromioplasty, the deltoid repair is protected, allowing only pendulum and passive motion exercises for 6 weeks.

Evaluation

While failure of the deltoid repair occurs early in the postoperative period, there is often a long delay prior to diagnosis or intervention.[197] The clinical presentation is variable and depends on the size and location of the deltoid defect, the status of the coracoacromial arch, the amount of remaining acromion, and the status of the rotator cuff. Most cases are heralded by a sudden onset of increased pain and localized swelling or hematoma formation over the anterior acromion. All patients who present with a sudden onset of pain and localized swelling or hematoma formation over the acromion within the first couple of weeks following rotator cuff surgery should be suspected as having a deltoid detachment, infection, or both.

Physical examination will reveal the presence of a visible and palpable defect at the detachment site, with the deltoid retracted distally (Fig. 5-11). Since the fibers of the middle deltoid attach to the acromion perpendicular to their line of action, postoperative detachment of the middle deltoid results in a greater degree of distal retraction than detachment of the anterior deltoid. The findings associated with a small detachment may be subtle and the examiner can verify the diagnosis by having the patient gently abduct or forward elevate the arm against resistance while palpating the border of the acromion with the other hand. When the rotator cuff is intact and there is no capsular contracture, overhead elevation is often preserved in all but the largest deltoid detachments. Patients with concomitant rotator cuff deficits, especially in the presence of a deficient coracoacromial arch, may present with severely compromised overhead function.[12,49,80,156,197,219]

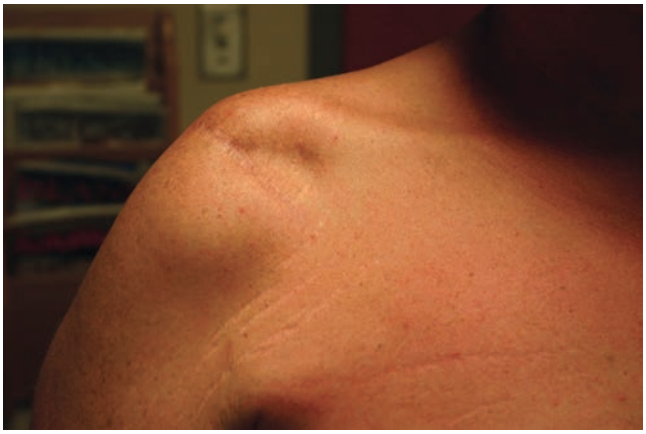


Figure 5-11 Postoperative deltoid detachment is characterized by distal retraction of the detached deltoid, which can easily be recognized as a mass or prominence in the anterior or lateral arm.

Routine radiographic evaluation should include anteroposterior, axillary, supraspinatus outlet, and 30-degree caudal-tilt views[42,153,180] to assess the amount of remaining acromial bone. Magnetic resonance imaging will reveal the deltoid detachment, but is most valuable in assessing the integrity of the rotator cuff (Fig. 5-12). If a concomitant deep wound infection is suspected, aspiration of the subacromial space or glenohumeral joint will provide a specimen for culture.

Treatment

Detachment of the deltoid following rotator cuff surgery is best managed by early detection and prompt deltoid repair. While small (1 to 1.5 cm) detachments may not be symptomatic or clinically relevant, they should be closely monitored for propagation of the defect or functional impairment. Larger deltoid detachments should be repaired as soon as they are recognized. When the detachments are detected early (within 4 to 6 weeks), the deltoid can usually be easily repaired. Firm repair to the bone of the acromion as well as the deltotrapezial fascia is recommended. The rotator cuff should also be carefully inspected for evidence of a recurrent tear, which should be repaired at the time of deltoid repair.

Deltoid detachments are frequently overlooked in the immediate postoperative period and may persist for prolonged periods of time.[80,197] Sher and collegues[197] reported on a series of deltoid detachments that were undetected for an average of 17 months. Mobilization and repair of the chronically retracted deltoid is extremely difficult, especially when it involves a significant portion of the middle deltoid. Full-thickness skin flaps are created and the skin is undermined to identify the intact portions of the deltoid as well as the retracted margins of the deltoid. Invariably, a thin layer of scar tissue occupies the interval between the retracted deltoid and the acromion. Incision of the scar tissue should begin at the anterolateral border of the acromion and extend distally toward the border of the normal deltoid. The subdeltoid adhesions should be carefully released with a combination of blunt and sharp dissection to establish the humeroscapular interface. Digital palpation of the deep and superficial surfaces will identify the

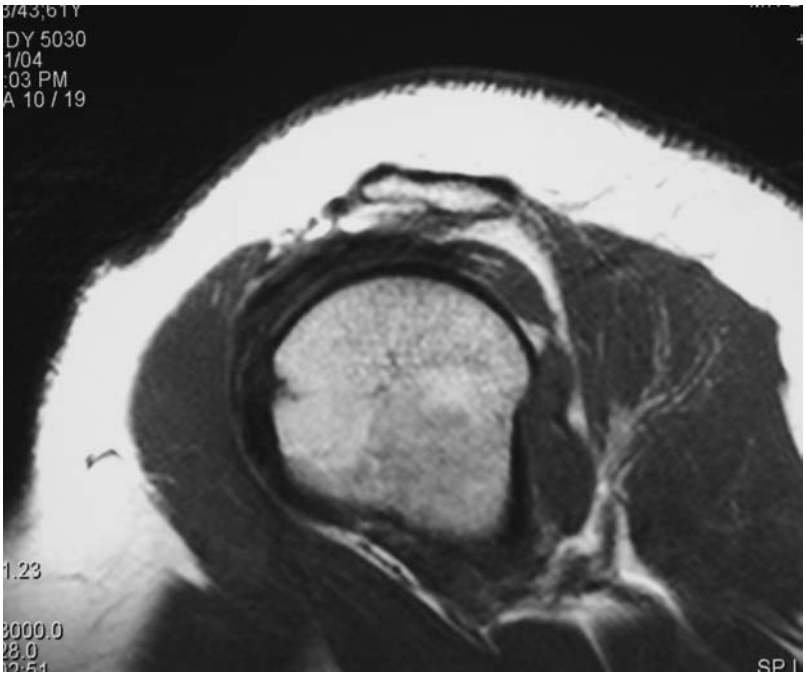


Figure 5-12 Sagittal magnetic resonance imaging scan of a postoperative deltoid detachment.

thicker area of the intact portions of the normal deltoid. Once the intact portions of the anterior and middle deltoid have been identified, the intervening scar tissue can be released from the acromion (Fig. 13A). The split at the anterolateral acromion can then be extended to the level of the axillary nerve, which is easily palpated on the deep surface of the deltoid fibers. A circumferential release of the retracted portions of the deltoid can then be performed to mobilize the muscle proximally toward the acromion (Fig. 5-13B). The retracted portions of the middle and anterior deltoid are then convergently mobilized toward the anterolateral border of the acromion, creating a V–Y repair configuration (Fig. 5-13C,D). The intervening scar tissue is then excised and sutures are placed in the corners of the anterior and middle deltoid that are to be approximated to the anterolateral border of the acromion. It is important to incorporate the deep deltoid fibers to facilitate a full-thickness repair. The acromion is then lightly decorticated and the anterior and middle portions of the deltoid are then sutured to the acromion through drill holes using heavy, nonabsorbable sutures. It may be helpful to retain a small portion of the scar tissue, which can be sutured to the deltotrapezial fascia, allowing reinforcement of the repair. The remaining split in the deltoid is then closed using interrupted absorbable sutures (Fig. 5-13E).

When primary repair is not possible, a rotational deltoidplasty may be performed by transposing a portion of the adjacent intact deltoid into the defect,[197] attaching it to the acromion. The new defect in the deltoid is then closed in a side-to-side fashion. As the complexity of the required reconstruction escalates, the expected results deteriorate.

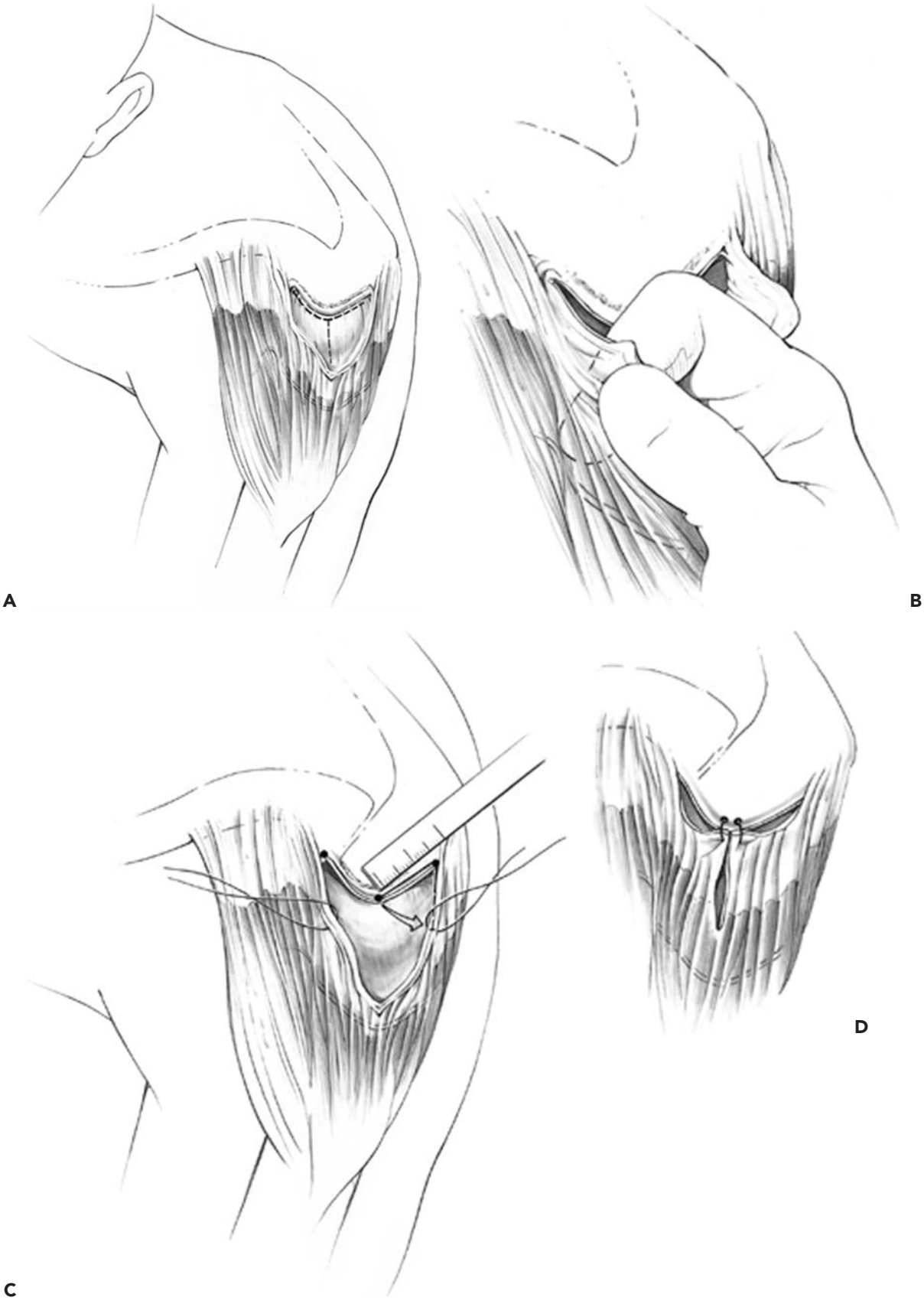
Postoperatively, the deltoid reconstruction or repair is protected for 3 to 6 weeks in an abduction orthosis. Passive motion exercises are initiated within the first week, followed by active motion and strengthening at 6 and 12 weeks, respectively.

Reported results for surgical treatment of deltoid detachment are sparse and generally carry a poor prognosis.[12,156,197] Sher and collegues[197] reported 67% unsatisfactory results among 24 patients who were followed for deltoid repair or reconstruction following postoperative deltoid detachment. The poorest results occurred in patients with an associated large rotator cuff tear who demonstrated weakness in external rotation, prior acromionectomy, and a large residual deltoid defect. Predictive variables for a favorable result include an intact or repairable rotator cuff, early recognition and treatment, little or no middle deltoid involvement, and no acromial insufficiency.[197]

AXILLARY NERVE INJURY

Etiology and Prevention

Axillary nerve injury represents the most common single nerve injury to the shoulder.[166] The course of the axillary nerve makes it vulnerable during any operative procedure involving the inferior aspect of the shoulder. Injury by direct laceration or overzealous traction may occur during any of the operative approaches, denervating the entire deltoid distal (anterior) to the point of injury.[22,31,147–149] This usually results in substantial disability.[80]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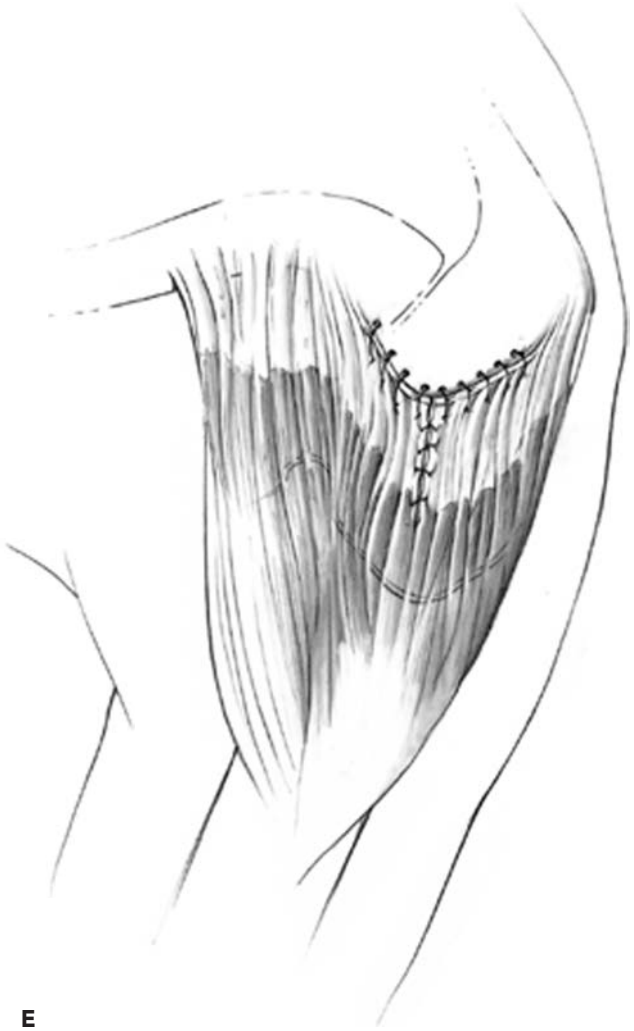

E

Figure 5-13 (*continued*).

The nerve courses posterior to the coracoid process and crosses the inferolateral border of the subscapularis, 3 to 5 mm medial to the myotendinous junction. Throughout its course it rests an average of 2.5 mm from the inferior glenohumeral ligament, passing within 12.4 mm of the glenoid rim at the 6 o'clock position.[173] At this point, the posterior branch separates from the main anterior circumflex branch and lies closest to the capsule and glenoid. The superior–lateral brachial cutaneous nerve and the nerve to the teres minor always originate from the posterior branch.[8] This anatomic relationship is of note because loss of sensation over the deltoid may indicate loss of teres minor function. After passing through the quadrangular space, the axillary nerve courses from posterior to anterior along the deep surface of the deltoid, which it innervates.[22,31,127] The posterior deltoid is variably innervated by both the anterior and posterior branches of the axillary nerve, with the nerves consistently entering the muscle directly inferior to the posterolateral corner of the acromion.[8]

The distance from the lateral edge of the acromion to the axillary nerve is generally thought to be about 5 cm,[127] but is subject to variation depending on the size of the arm (i.e., patient). Burkhead and collegues[31] have shown the axillary nerve to lie as close as 3.5 cm from the edge of the acromion in a cadaver specimen, and have shown that the distance decreases as much as 30% with increasing abduction of the humerus.

Axillary nerve injury during the superior, deltoid splitting approach can be avoided by limiting the extent of the deltoid split to 3 cm or less.[31] When more distal exposure is required, the axillary nerve can be easily palpated on the deep surface of the deltoid, and protected (Fig. 5-14). Neer recommended placing a suture at the apex of the deltoid split to prevent distal propagation and inadvertent nerve injury during retraction of the deltoid during rotator cuff repair.[147–149] During the anterior approach for subscapularis repair, the axillary nerve should be identified at the inferior border of the subscapularis muscle and may be gently protected with a blunt retractor. External rotation of the arm will also relax the nerve when performing the intraarticular capsular releases required to mobilize the tendon. Posterior approaches for latissimus dorsi or teres major transfer should maintain the subdeltoid dissection medial to the posterolateral border of the acromion to avoid denervating the posterior deltoid.[8] Arthroscopic approaches should avoid excessively inferior placement of any of the portals[166] and pay careful attention to avoiding excessive fluid extravasation into the arm. Arthroscopic capsular releases should be performed with the knowledge that the axillary nerve is in close proximity to the inferior glenohumeral ligament.[173] Abduction, external rotation, and perpendicular traction may increase the zone of safety during arthroscopic capsular releases near the 5 to 7 o'clock positions.[208]

Figure 5-13 The interval between the detached deltoid and the acromion will be traversed by a thin layer of scar tissue. The scar tissue is released from the margins of the acromion, and is then incised distally **(A).** Digital palpation of the anterior and posterior flaps will reveal the junction of the thick retracted deltoid edge and the thinner scar tissue **(B).** Palpation of the axillary nerve defines the distal extent of the longitudinal split. The respective distances from the intact portions of the middle and anterior deltoid to the anterolateral corner of the acromion are measured. Sutures are placed within the fibrous, retracted edges of the deltoid at the points corresponding to the previously measured distances **(C).** These two sutures are then passed through drill holes in the anterolateral corner so that they come to rest adjacent to one another **(D).** The remaining portions of the deltoid are also reattached to the acromion through drill holes **(E).**

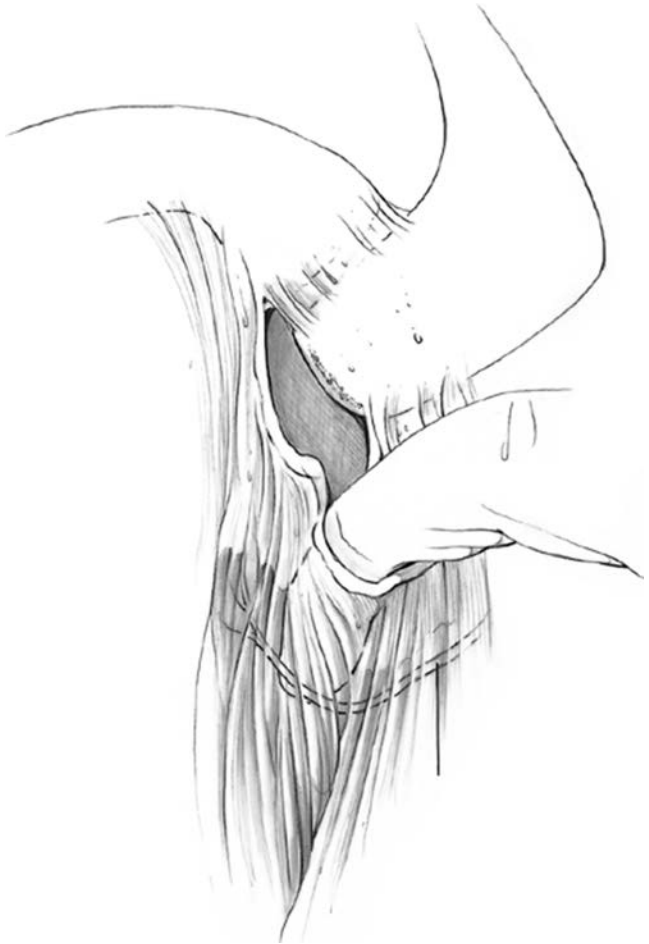

Figure 5-14 Digital palpation of the deep surface of the deltoid can be used to identify the axillary nerve so that the deltoid split does not extend distal enough to cause injury to the nerve.

Evaluation

The clinical features associated with axillary nerve injury vary according to the location of the injury. In some cases, the location of the previous skin incision will suggest an axillary nerve injury. The initial presentation usually includes weakness in shoulder abduction and elevation. However, deltoid weakness may be masked by a strong and competent rotator cuff. Numbness and paresthesias in the lateral arm will be present when the injury is proximal to the superior lateral brachial cutaneous nerve.[8] Since the sensory nerve usually arises proximal to the common zone of injury and the cutaneous distribution of the axillary nerve and associated cutaneous nerves overlap substantially, the absence of sensory deficits is often unreliable in making the diagnosis. Generally, a characteristic pattern of deltoid atrophy will develop distal (anterior) to the site of injury (Fig. 5-15).[22,31,32,51,119,124,127] Other causes of atrophy should be excluded at the time of physical examination. The absence of a palpable defect at the border of the acromion will help to differentiate a nerve injury from a deltoid detachment. Disuse atrophy will affect all portions of the deltoid, mimicking the atrophy seen with a proximal nerve injury. However, disuse atrophy should not present with associated sensory deficits.

Deltoid dysfunction in a suspected proximal nerve lesion may be assessed clinically by performing the deltoid lag test.[101] Since no other muscle can compensate for the posterior part of the deltoid in a position of maximal shoulder extension, testing in this plane is highly specific for the deltoid and, consequently, axillary nerve function. With the patient seated, the physician places the arm into full extension and asks the patient to actively maintain this posture. If the deltoid is weak, the arm will drop. The suspected

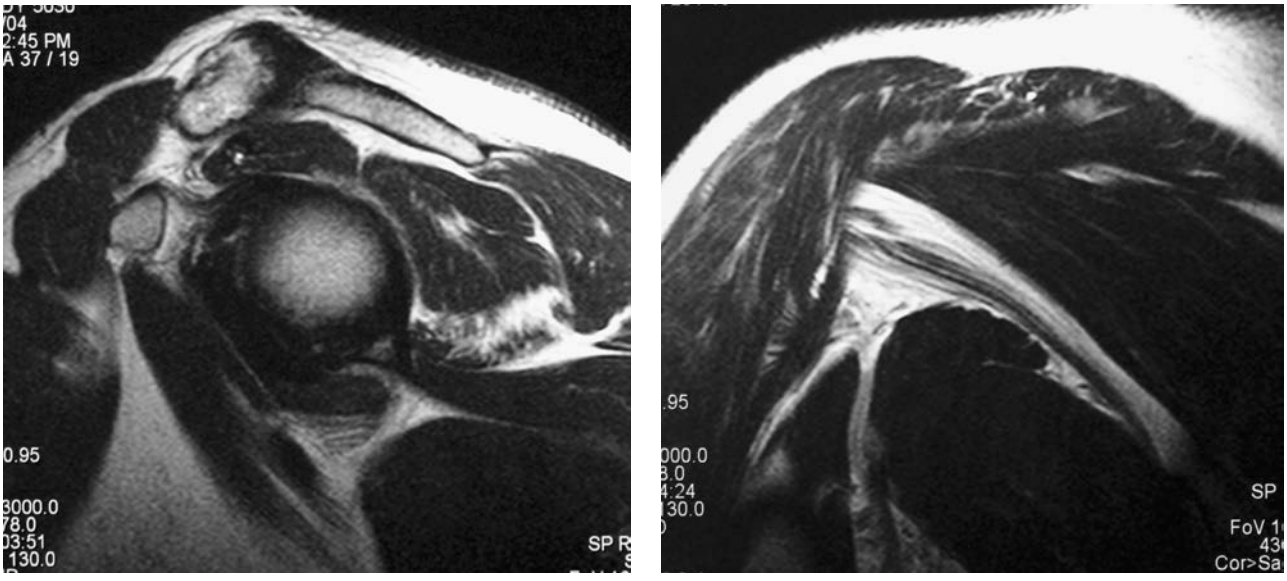

Figure 5-15 Axillary nerve injury during rotator cuff surgery is the result of direct incision of the nerve or traction neuropathy. Fatty atrophy is seen within the teres minor muscle.

diagnosis of an axillary nerve injury is confirmed by electromyography. All three portions of the deltoid should be tested to ensure that a false-negative study is not produced from isolated testing of an intact portion of the deltoid. Experienced electromyographers may be able to provide an opinion on the type of nerve injury (i.e., whether the nerve is likely to be in continuity). Serial electrophysiologic studies may be used to follow progressive regeneration of the nerve. Magnetic resonance imaging is helpful in determining the integrity and reparability of the rotator cuff.

Treatment

Treatment of an axillary nerve injury depends on the degree of functional impairment, as well as the status of the rotator cuff and coracoacromial arch. When the injury is proximal or posterior, exploration may be considered if there is no clinical or electrophysiological recovery by 3 to 6 months following injury. However, the nerve injury typically occurs at the level of the anterolateral acromion, whereby the caliber of the nerve and terminal branches often precludes neurolysis, nerve repair, or grafting. Therefore, available treatment options include activity modification and rehabilitation, muscle transfers, and arthrodesis. Although active abduction may be limited after acute injury, most patients with an intact rotator cuff are able to compensate for the loss of deltoid function with time and are willing to pursue nonoperative treatment. Some authors have reported excellent functional shoulder recovery among patients with an isolated axillary nerve injury, despite a complete paralysis of the deltoid muscle.

Management of an axillary nerve injury in the presence of a recurrent rotator cuff tear depends on the size and reparability of the rotator cuff defect and on the integrity of the coracoacromial arch. When the rotator cuff defect is small or reparable, consideration is given to bipolar latissimus transfer.[110] Alternatively, Leffert has described rotational deltoidplasty with excision of the denervated portion of the muscle to restore anterior deltoid function. These procedures are not performed, however, until the revision rotator cuff repair has been performed and the subsequent final results have been evaluated. The location of the axillary nerve injury may spare enough of the middle deltoid that affected patients are willing to live with the axillary nerve injury if the repeat rotator cuff surgery has been successful.

Axillary nerve injury in association with a massive or irreparable rotator cuff tear is usually not a correctable problem. When coracoacromial arch insufficiency is added to this clinical vignette, the shoulder is nearly flail. The likelihood of restoring normal function to any portion of this triad is abysmal, leaving the patient with a choice between acceptance of the deficits or arthrodesis. Patients considering arthrodesis should fully comprehend that they will lose all rotational motion of the shoulder, even with the arm at the side. The potential pain relief and stability provided by arthrodesis may not justify the loss of this last remaining function of the shoulder.

SUPRASCAPULAR NERVE INJURY

Etiology and Prevention

Numerous studies exist describing the course of the suprascapular nerve, as well as pathologic entities associated with it. Interestingly, iatrogenic injury to the suprascapular nerve during rotator cuff surgery has been reported only a handful times in the literature.[76,226] It is usually the result of aggressive lateral mobilization of a large or massive chronically retracted rotator cuff tear. The nerve either becomes kinked along the lateral margin of the suprascapular notch or is directly injured by surgical dissection, which extends too far medial to the glenoid rim. All of the above reported cases were confirmed using electrodiagnostic studies. Since postoperative electrodiagnostic studies are not routinely performed, the true incidence of suprascapular nerve injury may be higher than reported.

While suprascapular neuropathies of the shoulder can present with concomitant rotator cuff tears, the clinical presentation may be confusing, and the rotator cuff tear may mask a neuropathy.[209] Clinical examination alone will not differentiate between rotator cuff tears and suprascapular neuropathy. Since the clinical manifestation of suprascapular nerve entrapment is pain and associated external rotation weakness, the significance of the neuropathy may be lost in the presence of a rotator cuff tear. It is more likely that an electromyography study is obtained following rotator cuff repair to determine the cause of persistent pain or weakness. However, if a nerve injury is uncovered at this point, it is not possible to elucidate whether it occurred iatrogenically or was present preoperatively.

Postoperative suprascapular neuropathy may actually represent a failure in diagnosis, with the injury having been present preoperatively. A recent study by Vad[209] and colleagues utilized preoperative electromyography to show a 28% incidence of neurologic injury occurring in the presence of large rotator cuff tears with associated atrophy. Albritton and colleagues[2] have shown in a cadaveric study that 2 to 3 cm of medial retraction of the supraspinatus tendon will change the course of the suprascapular nerve and place it under tension. They postulate that supraspinatus and infraspinatus atrophy, following isolated supraspinatus tears and massive tears, may be associated with suprascapular nerve injury. Lateral mobilization of the muscle during rotator cuff repair may actually be beneficial in relieving tension on the nerve and may assist in reversal of muscle atrophy in the supraspinatus. The infraspinatus muscle appears to be less resistant to nerve injury.

The course of the suprascapular nerve predictably leaves the superior trunk of the brachial plexus and reaches the

dorsal surface of the scapula by passing through the suprascapular notch, beneath the transverse scapular ligament.[16,211] Two motor branches typically innervate the supraspinatus muscle, with the first motor branch being the larger of the two and originating under or just distal to the ligament. The nerve continues through the spinoglenoid notch, where three to four motor branches innervate the infraspinatus muscle. The inferior transverse scapular ligament appears to be a variable structure under which the nerve may become compressed.[108] Superiorly, the nerve is 2.5 to 3 cm medial to the supraglenoid tubercle, whereas the branches to the infraspinatus lie 1 to 2 cm medial to the midportion of the posterior glenoid rim.[16,211]

The anatomy of the suprascapular nerve leaves it vulnerable to either direct or indirect injury.[16,211] Capsular releases passing medial to the above described "safe zone" risk direct injury to the nerve. Limiting the amount of medial dissection through the capsulotomy may avoid injury to the nerve branches. Indirect injury occurs when traction is placed on the nerve branches during lateral mobilization of the rotator cuff tendon. Since the nerve occupies a relatively fixed position on the floor of the supraspinatus fossa and at the notch, lateral advancement of the tendon risks tethering the nerve as it crosses through the notch while the motor branches pivot around the pedicle of the nerve.[211] Cadaveric dissections demonstrated tethering of the nerve and its branches when the tendon was advanced laterally further than 1 cm using conventional repair techniques.[78,211] Warner and colleagues have shown that this safe zone may increase to 3 cm when the muscle is released from its respective fossa and allowed to advance laterally with the tendon.[211]

The clinical relevance of cadaverically derived safe zones for advancement is not entirely clear. Warner and colleagues[211] obtained their data using cadavers without retracted rotator cuff tears. Lateral advancement of greater than 1 cm may be safely performed when a large, acutely retracted musculotendinous unit is restored to its original, premorbid length. Conversely, it may not be possible to gain even a centimeter of length in a chronically retracted musculotendinous unit without placing significant tension on the neurovascular pedicle. In general, the concept of limiting the amount of lateral advancement of a chronic, retracted rotator cuff tear during repair is valid, although the clinical safe limit for advancement is not entirely known.

Evaluation

Determining the cause of persistent pain and external rotation weakness following rotator cuff repair may be difficult. A protracted recovery may be expected in association with surgical repairs of large, chronically retracted rotator cuff tears.[64,107,184] The additional effects of an associated nerve injury may be difficult to quantify. However, the presence of significant external rotation weakness with the arm at the side and at 90 degrees of abduction should alert the surgeon to the possibility of either a recurrent rotator cuff tear or a suprascapular nerve injury. Preoperative and surgical records should be reviewed to determine the integrity of the rotator cuff. If the rotator cuff was intact at the time of surgery, a suprascapular nerve entrapment or nerve injury should be suspected. Similarly, when the size of a preoperative rotator cuff defect is not sufficient to explain the amount of atrophy or external rotation weakness, the diagnosis of nerve injury is entertained. Under these circumstances, electrodiagnostic testing is indicated. In all cases of suspected suprascapular nerve injury, testing should be included for the possibility of superimposed cervical radiculopathy. If these studies indicate that the suprascapular nerve and the brachial plexus are normal, then magnetic resonance imaging is performed to identify a persistent or recurrent rotator cuff tear.

Preoperative magnetic resonance studies may supply the clinician with valuable information regarding the suprascapular nerve.[109] Studies depicting atrophy of the supraspinatus and infraspinatus muscle bellies, in the presence of a supraspinatus tear and an intact infraspinatus, should indicate the possibility of a preoperative suprascapular nerve entrapment (Fig. 5-16). The presence of a ganglion cyst in the suprascapular or spinoglenoid notches may also cause entrapment of the nerve at its respective location.[168]

Treatment

Treatment options for a suprascapular neuropathy include observation and rehabilitation, nerve exploration and decompression, and muscle transfers.[45] Management depends on the degree of functional impairment, the age and activity level of the patient, and the status of the supraspinatus and infraspinatus muscles. Since most injuries represent a traction neurapraxia or axonotmesis, nonoperative treatment should be considered in all patients with a suprascapular nerve injury. Activity modification to avoid additional trauma and irritation of the nerve is balanced with a scapular and rotator cuff conditioning program to maximize muscular function while the nerve is healing. While some patients may improve, the overall success rate of nonoperative treatment is not known. Failure to follow an appropriate rehabilitation program and continued high-demand activities may actually lead to worsening of symptoms.[45] Even when the nerve does not completely recover, many patients are willing to modify their activities and accept the limitations associated with the suprascapular nerve injury.

The presence of a suprascapular nerve lesion in association with a retracted tear involving the supra- and infraspinatus may represent a special situation. As mentioned above, retraction of the supra- and infraspinatus changes

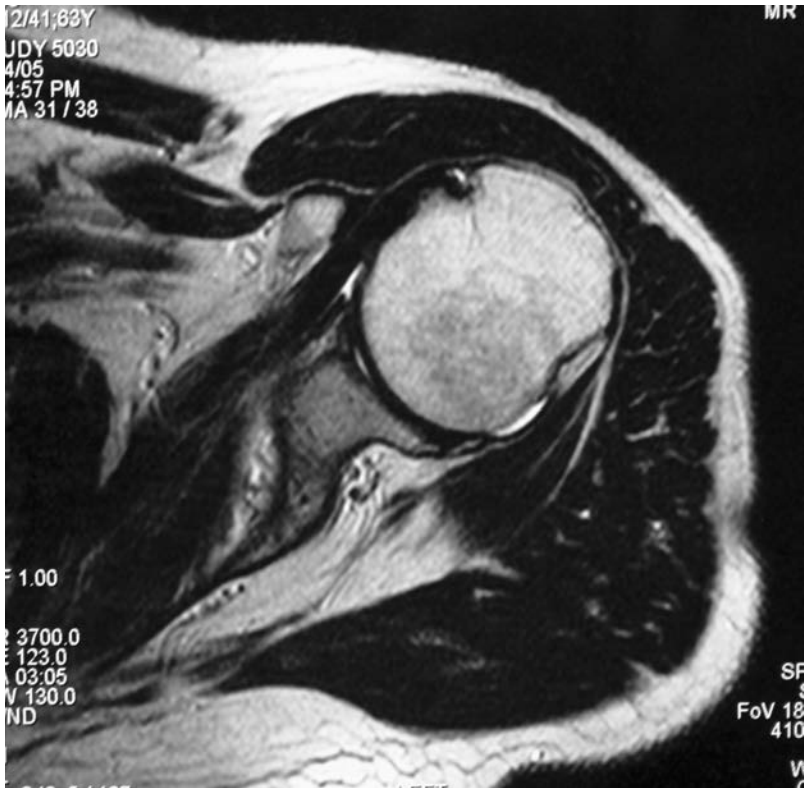

Figure 5-16 Preoperative magnetic resonance imaging scan of a patient with suprascapular neuropathy reveals severe increased signal in the infraspinatus muscle in the presence of an intact tendon.

the course of the nerve and may actually cause the nerve to be tented over the scapular spine. Therefore, repairing the tendon may relieve the tension on the nerve. This phenomenon may be an indication for relatively early cuff repair to facilitate nerve recovery.

Operative intervention may be considered in the young or active patient who presents with suprascapular neuropathy following mobilization and repair of a chronic, retracted rotator cuff tear. Nerve exploration and decompression is indicated when there exists significant functional impairment, no electromyographic or clinical improvement has been shown over a 6-month period, the rotator cuff repair is intact, and the supraspinatus and infraspinatus muscles have not undergone significant fatty degeneration. In a proximal lesion, decompression of the nerve is performed at the suprascapular notch and includes division of the transverse scapular ligament with lateral enlargement of the notch as necessary.[5,172,174] With isolated involvement of the infraspinatus, the nerve should be approached on both sides of the scapular spine. When present, the spinoglenoid ligament should be released. The spinoglenoid notch should not be deepened further than 1 cm to avoid creating a stress riser at the base of the acromion.

Tendon transfers are considered in patients who have either failed decompression of the nerve or are not candidates for nerve decompression. However, it is highly unlikely that a patient with an intact rotator cuff repair will have significant enough functional impairment related to the suprascapular neuropathy to warrant tendon transfers to augment posterior rotator cuff function. In this rare circumstance, tendon transfers are performed as described for irreparable posterior rotator cuff insufficiency in Chapter 4.

Postoperative suprascapular neuropathy, which occurs in conjunction with an intact rotator cuff, probably represents a failure in preoperative diagnosis. This situation should be treated as a primary suprascapular neuropathy.

RECURRENT ROTATOR CUFF TEAR

Etiology and Prevention

Since Codman's initial treatise on the surgical treatment of rotator cuff tears in 1911,[39] improved operative techniques have been responsible for a high success rate, with enduring patient satisfaction.[1,40,62,96,185] However, recurrent or persistent rotator cuff defects have been reported to occur in 20% to 90% of cases,[18,34,60,64,74,88,118,126,202] with the risk of recurrence increasing relative to the size of the initial tear. The failure rates between arthroscopic and open repairs appear to be equal when the tear is small and involves minimal retraction of the musculotendinous unit. The incidence of failure is highest among elderly patients with chronic and retracted tears involving two or three tendons. Arthroscopic and open repairs under these conditions

represent the highest retear rate, with early reports suggesting that arthroscopic repair may be associated with higher recurrence rates.[18,60] Associated risk factors for recurrent tears include advanced age, tear size, fatty degeneration, chronicity and atrophy, poor tendon quality, poor bone quality, inappropriate rehabilitation, inadequate subacromial decompression, smoking, steroid injections, and diabetes.[15,49,64,74,88,126,156,202] Persistent defects are not necessarily the sine qua non for failure, since the presence of a persistent rotator cuff defect is compatible with a good postoperative result following rotator cuff repair.[34,64,88,118,126] This process of converting a symptomatic tear into an asymptomatic retear is not entirely clear, although it may involve adequate subacromial decompression,[24,25,164,181] débridement, biceps tenotomy, partial healing of the rotator cuff, and adequate postoperative rehabilitation. The quality of functional results, however, depends on the size of the persistent defect, associated atrophy of the rotator cuff muscles, integrity of the deltoid and the coracoacromial arch, and functional demands of the patient.[53,88,107,148,181,219] Patients with persistent rotator cuff defects will be capable of overhead function when the deltoid is intact and the anterior and posterior portions of the rotator cuff are intact and balanced.[24,25,28,148,181] However, they will generally complain of fatigue with overhead activities and limitation in activities that require vigorous or sustained overhead strength, as compared to patients with an intact rotator cuff.[64,88] Therefore, the goal of rotator cuff repair is long-term restoration of a functional, healed musculotendinous unit. While this may not always be attainable in primary rotator cuff repair, the development of recurrent rotator cuff tears may be mitigated through a combination of careful preoperative patient selection, meticulous surgical technique, and attention to appropriate postoperative protection and rehabilitation.

With the understanding that the correlation between postoperative subjective and functional results and anatomic results (i.e., rotator cuff integrity) is variable,[64,88,126] outcome studies have begun to focus on patient satisfaction in terms of patient-derived subjective assessments of symptoms and function.[104,140,160,183] While it is true that, on average, postoperative shoulder performance scores are better in patients with intact rotator cuff repairs than in those with reruptures, even patients with rerupture often have improved pain and function. Preoperative and surgical variables that are associated with poorer patient satisfaction include age less than 55 years; smoking; débridement of massive, irreparable rotator cuff tears; chronic or unrepairable subscapularis tears; and patients with larger or massive supraspinatus and infraspinatus tears.[40,69,91,104,107,130,160,185,200,215,217] Objective postoperative variables that are associated with poorer patient satisfaction include diminished and weakened forward elevation, impingement signs, and acromioclavicular joint pain and tenderness. Subjective variables associated with poorer patient satisfaction include persistent pain, functional impairment, and work disability.[160,217]

Preoperative variables exist that will have a bearing on the ability to obtain long-term tendon-to-bone healing. In the presence of an acute rotator cuff tear, the biologic potential for healing appears greater when the repair is performed within 3 weeks of injury.[9] In long-standing tears or delayed repairs, muscle atrophy and fatty infiltration may develop, which are variably reversible[190] and appear to have a negative effect on the outcome following rotator cuff repair.[74,75] There appears to exist a direct relationship between tear size and degree of atrophy.[202,225] These changes may be graded using computed tomography scanning or magnetic resonance imaging and increase with elapsed time from the tendon rupture.[59] Additionally, chronic retraction and scarring of the musculotendinous unit may preclude the surgeon from obtaining an adequate tendon-to-bone repair. Therefore, in the presence of an acute or acute-on-chronic rotator cuff tear with retraction of the tendon, early repair is more likely to result in long-term tendon-to-bone healing as compared to late repair. This potential advantage should be considered in the context of appropriate patient selection criteria such as age, physical demands, comorbidities (diabetes, smoking), and motivation (willingness to comply with rehabilitation), prior to recommending surgical intervention.

The surgical principles that most likely reduce postoperative recurrent tears or persistent defects include adequate mobilization of the tendon to the greater tuberosity, preparation of the tendon and bone interfaces, and secure fixation of the tendon to bone. These principles hold true for all repair methods. The superficial surface should be free from the overlying deltoid, acromion, subdeltoid bursa, scapular spine, coracoid, and the trapezius muscle. Capsular releases are frequently required to release the tenodesis effect of the underlying capsule.[228] Interval releases may also be required to allow full excursion of the contracted tendon.[14,148–150] A thorough understanding of the various tear configurations will enable the surgeon to perform the proper releases, repair longitudinal tears in a convergent manner, and repair the tendon to bone with minimal tension. A repair that has been overly tensioned will eventually fail.[30] Inability to adequately mobilize the tendon may be due to substantial intramuscular scarring and the development of fatty atrophy. Tendon convergence and advancement methods are discussed extensively in Chapter 3, while the management of the irreparable rotator cuff tear is discussed in Chapter 4.

Débridement of the greater tuberosity, along with abrasion of the cortical surface at the proposed site of tendon attachment, may enhance tendon healing. While open rotator cuff techniques often include the creation of a shallow cancellous trough, most arthroscopic techniques repair the tendon to the cortical surface of the greater tuberosity. The rates of tendon healing between a shallow

cancellous groove or trough, and a cortical bone surface appear to be equal.[201] Complete removal of the cortical surface of the humeral attachment site in long-standing rotator cuff tears will often reveal a virtually hollow greater tuberosity and should be avoided.[139] While excessive débridement of the tendon edges is not necessary or advisable, excision of the friable, necrotic edges will reveal a healthy tendon edge for suture placement.[73] Rotator cuff repair failures frequently occur when the suture pulls through the tendon.[46] Tendon grasping suture techniques such as the Mason-Allen technique provide excellent pullout strength and presumably a more durable repair during open rotator cuff surgery, as compared to simple or mattress sutures.[70,193] The holding strength of arthroscopically tied horizontal mattress sutures appears to be higher than that of the Mason-Allen suture. The massive cuff stitch, which includes a horizontal mattress stitch that is reinforced with a vertical loop, may act in a similar manner to the Mason-Allen suture technique.[128] Despite these data, there is no evidence to show that tendon grasping suturing techniques produce any higher healing rates than multiple simple sutures; many surgeons still prefer multiple simple sutures over mattress or other tendon grasping techniques.

Strength of fixation is related to the pullout strength of anchoring devices, knot stability, suture elongation, the number of bone anchors, quality of bone and rotator cuff tissue, passive tension, and physiologic cyclic loading; the interested reader is directed to Chapter 3 on rotator cuff repair. While the holding strength that is required for successful tendon-to-bone healing is unknown, data exist supporting both the use of open transosseous techniques as well as arthroscopic fixation using bone anchors.[38,175] The advent of improved suture anchoring devices has led to their widespread acceptance and use in rotator cuff repair procedures. Advantages include the ease of use, decreased operating time, and decreased surgical exposure and morbidity. Suture anchors can provide equivalent fixation strength to transosseous tunnels, and under cyclic loading conditions may be somewhat better.[26,38,175] While most arthroscopic rotator cuff repair failures occur at the tendon–suture interface, anchors may subside without actually pulling out of the bone, leading to gap formation, poor tendon-to-bone healing, and rotator cuff repair failure (knot loosening or loop elongation will also contribute to failure). Pullout strength varies according to the anchoring device and bone quality of the humeral head.[139] Tingart and colleagues[204] have shown that the higher bone mineral density in the proximal–medial and proximal–anterior regions of the greater tuberosity (i.e., closer to the articular surface) is associated with increased pullout strength of suture anchors. While suture anchors may represent an attractive option in young patients with high bone density, caution should be exercised among patients with poor bone quality such as postmenopausal women, smokers, and elderly patients, as these devices may not provide adequate pullout strength.[193] Osteoporosis of the proximal humerus will also develop in the presence of a chronic rotator cuff tear, regardless of age.[139] Under these circumstances, passing sutures through bone tunnels and tying the sutures over a lateral bone bridge may provide superior strength.[70,193] This may be augmented with a thin plate or button to provide additional resistance to pullout of the sutures.[33]

Proper postoperative protection and rehabilitation play an important role in preventing postoperative recurrent or persistent rotator cuff defects. While the clinical importance of early passive motion of the shoulder following rotator cuff repair is well established,[111,117,149,150,152,180] the positions of extreme extension, adduction, and internal rotation should be avoided in the early postoperative period to minimize tension on the repair.[228] Arm position in this extreme range may not pose a problem with smaller tears. However, in larger posterior–superior tears, passive forward elevation exercises should be combined with slight abduction of the arm (15 to 30 degrees) to offset the effects of flexion. A protective orthosis may be placed with the arm at the side in a small amount of abduction to decrease passive tension and reduce bone–tendon gap formation.[176] Chronic or massive tears may require the use of a larger abduction pillow or orthosis, depending on the amount of tension that is observed at the time of surgery.[99] It should be emphasized that an abduction orthosis, which is placed to relieve tension, is unlikely to induce healing in the presence of an overtensioned repair. Tension overload will predispose to failure of the repaired fibers under physiologic cyclic loading conditions.[26,30] Active motion exercises should be instituted between 6 and 8 weeks postoperatively,[111,117,149,150,152,180] avoiding resisted vigorous strengthening and isokinetic exercises until 3 months following repair. The initiation of early active motion and the use of weights in the early postoperative period have been associated with failure of the tendon repair.[156]

Evaluation

Since the risk of recurrent rotator cuff tears is highest among elderly patients with chronic tears involving two or more tendons,[88] the index of suspicion for this complication should be high when these patients complain of persistent pain and functional impairment. Physical findings vary according to the size and location of the tear, the integrity of the coracoacromial arch, and the presence of soft tissue contracture. Lack of both active and passive elevation is more likely to represent capsular contracture rather than residual rotator cuff insufficiency. It is extremely difficult, if not impossible, to determine the presence and clinical relevance of recurrent rotator cuff tears in a stiff shoulder. Once the soft tissue contracture has been excluded or corrected, the physical findings associated with a recurrent rotator cuff tear will become more apparent.

Symptomatic patients will have subacromial crepitance and weakness on isolated muscle testing, and will often demonstrate a positive "lag sign" on physical examination.[100] When pain appears to be a limiting factor during strength testing, a subacromial lidocaine injection will usually alleviate a significant amount of associated pain and increase the reliability of the examination. Recurrent defects in the posterior–superior portion of the rotator cuff (supraspinatus and upper infraspinatus tendons) generally demonstrate weakness of arm abduction and weakness of external rotation with the arm at the side. This external rotation weakness generally improves when the arm is brought into 90 degrees of elevation in the scapular plane. Conversely, a tear extending to the posterior–inferior portion of the rotator cuff (inferior infraspinatus and teres minor tendons) will usually demonstrate external rotation weakness with the arm at the side as well as in 90 degrees of elevation in the scapular plane. External rotation lag signs refer to the inability of the patient to actively maintain maximal external rotation of the arm when it has been passively placed in this position by the examiner. Positive external rotation lag signs with the arm at the side, or with the arm at 90 degrees of elevation in the scapular plane, indicate recurrent defects in the posterior–superior and posterior–inferior rotator cuff, respectively. External rotation lag signs at any level of abduction are not particularly reliable in the detection of weakness associated with an isolated supraspinatus tear.[106] Abduction of the arm in the scapular plane with the elbow extended and the humerus internally rotated is generally accepted to represent supraspinatus function.[100,111] Therefore, weakness in this position may indicate a recurrent or persistent tear of the supraspinatus.

Subscapularis insufficiency results in increased passive external rotation of the shoulder, as well as weakness of terminal internal rotation. Detection of a recurrent defect requires careful isolation of the muscle from other internal rotators of the shoulder girdle.[79] The internal rotation lag sign[100] represents the inability of the patient to maintain the dorsum of the hand away from the midlumbar spine after it has been passively placed in this position of maximal internal rotation by the examiner. Similarly, the lift-off test[69] is sensitive for detecting subscapularis insufficiency and describes the inability of the patient to actively lift the dorsum of the hand away from the lumbar spine. These tests require full passive internal rotation to place the arm in the appropriate position. In the presence of posterior capsular contracture, the abdominal compression test appears to be as specific as the lift-off test in determining subscapularis insufficiency.[205] A positive test occurs when the patient is unable to maintain the flexed elbow anterior to the coronal plane of the body while simultaneously maintaining the palm of the hand compressed against the abdomen. Scapular protraction is often difficult to control and will interfere with the performance of this test. When this occurs, it may be easier to control scapular protraction with the patient in the supine position.

The suspected diagnosis of a recurrent rotator cuff tear may be confirmed with ultrasonography, arthrography, or magnetic resonance imaging.[34,64,81,88,129,162] The presence of postsurgical artifact will interfere with the interpretation of imaging studies, and the diagnostic criteria for a full-thickness recurrent tear are more stringent than that for a shoulder that has not undergone surgery. Magnetic resonance arthrography may offer the most information including tear size, muscle atrophy and fatty infiltration, and concomitant biceps and labral pathology. While the clinical relevance of minor tendon signal abnormalities is uncertain (Fig. 5-17A),[144] a well-defined tendon gap that is traversed by fluid is a reliable indicator of a persistent or recurrent rotator cuff defect (Fig. 5-17B).[81,118,162,202]

Treatment

The treatment options for recurrent or persistent rotator cuff defects are the same as those for primary rotator cuff tears. They include rehabilitation and activity modification, débridement, revision repair or partial repair, tendon augmentation (utilizing allograft, autograft, or xenograft), and distant tendon transfer.[14,24,25,49,66,82,181] Specific treatment recommendations are dependent on patient age, activity level, motivation, tear size, functional impairment, and tissue quality. The mere presence of a recurrent tear of the rotator cuff is not by itself an indication for repair. Revision rotator cuff repair is much more challenging than primary rotator cuff repair, especially in the setting of deltoid detachment or coracoacromial arch insufficiency. Rehabilitation of the remaining portions of the rotator cuff, deltoid, and scapular stabilizers is a reasonable initial approach for many patients with a recurrent defect, and may eliminate the need for further surgical intervention. Postoperative stiffness from subacromial scarring or capsular contracture should also be identified and corrected prior to considering revision rotator cuff surgery, especially in patients with large recurrent defects. Among patients with capsular contracture and irreparable rotator cuff insufficiency, arthroscopic capsular release may improve their function sufficiently that they are able to tolerate the residual rotator cuff insufficiency and avoid a complicated reconstructive procedure.

Revision rotator cuff surgery may be considered when nonoperative treatment has failed. Early surgical intervention is considered in patients who are markedly symptomatic following a traumatic, inciting event, which results in an early retear of the rotator cuff repair. This is especially true when the patient's original surgery was performed to repair an acute, large tear and the original repair was felt to be of good quality. Symptomatic patients who demonstrate weakness upon isolated muscle testing, have positive lag signs, and whose imaging studies reveal a recurrent tear

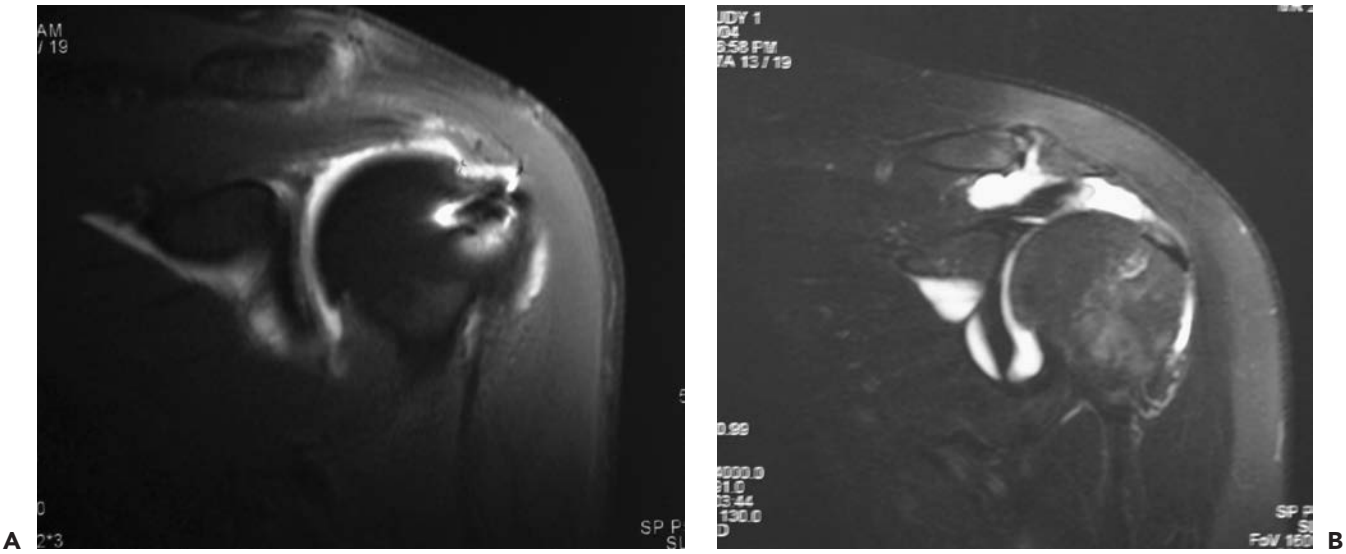

Figure 5-17 Criteria for the diagnosis of a recurrent full-thickness defect must be stringent to avoid false-positive results secondary to postsurgical artifact. **(A)** Subtle signal intensity abnormalities seen on magnetic resonance arthrography may represent scarring rather than a recurrent defect. **(B)** A well-defined tendon gap filled with fluid that communicates to the subacromial space, however, is a reliable sign of a persistent or recurrent rotator cuff tear.

that is commensurate with the physical findings are candidates for revision rotator cuff repair. The surgical principles of tendon mobilization and tendon-to-bone fixation are the same as that for primary rotator cuff repair. When the entire defect is not reparable, partial repair may decrease strain at the margins of the tear and result in improved function.[25,29] A decision must then be made regarding tendon augmentation, tendon transfer, or leaving a residual defect. The anterior and posterior portions of the rotator cuff should not be transposed superiorly to close the defect. In the presence of intact anterior and posterior force couples, a residual superior defect may be commensurate with intact overhead function, especially if this ability was intact preoperatively.[24,28] A patch graft represents a potential treatment option to augment the defect between the anterior and posterior cuff tendons, acting to resist inferior displacement of the tendons and therefore maintaining a more favorable orientation of the force couples.[145] However, its effectiveness appears to decline with increasing tear size.[194] Most patches are currently only approved for augmentation of a repair and not for spanning of large, irreparable defects. Latissimus dorsi transfer[66,71,212] may be considered for irreparable posterior cuff insufficiency, while pectoralis major or teres major transfer may be considered for anterior cuff insufficiency.[3,221] The coracoacromial ligament should be preserved in all cases and repaired to the acromion.

The reported results following revision rotator cuff repair are conflicting, with most authors describing disappointing results in about 50% of patients.[15,49,217] A satisfactory outcome is most dependent on an adequate subacromial decompression, repair of the rotator cuff with tendon-to-bone sutures, avoidance of weights in the early postoperative period, and an intact and functioning deltoid.[156]

ANTEROSUPERIOR HUMERAL HEAD SUBLUXATION

Etiology and Prevention

Normal shoulder kinematics in the presence of an intact rotator cuff, functioning deltoid, and intact coracoacromial arch will maintain the geometric center of the humeral head within 2 to 3 mm of the center of the glenoid during active elevation of the arm.[56,113,115,170] Although the greater tuberosity may contact the anterior acromion and coracoacromial ligament with elevation above 60 degrees,[56] the compressive force of the normal rotator cuff will actively center the humeral head into the glenoid fossa during deltoid muscle contraction and serves as the primary restraint to anterosuperior translation of the humeral head.[113,125] Under normal loading conditions, the static coracoacromial arch plays a limited role as a secondary passive restraint to anterosuperior subluxation, but assumes a crucial role in the presence of a dysfunctional rotator cuff.[57] In the presence of small rotator cuff tears, there are usually sufficient forces anteriorly and posteriorly to compensate for the loss of function that accompanies the tear. Although glenohumeral kinematics are altered,[224] the remaining rotator cuff muscles continue to serve as the primary restraint to superior translation, and

the coracoacromial arch probably plays a more important role as a passive restraint. When rotator cuff tears become too large or are "unbalanced," the remaining rotator cuff muscles are unable to compensate for the loss of joint compressive forces.[27] In these "uncompensated" rotator cuff tears, the humeral joint reaction force is not directed into the glenoid. Rather, the muscle imbalance between a dysfunctional rotator cuff and a strong deltoid directs the humeral head anterosuperiorly, and the intact coracoacromial arch becomes the only restraint to further anterosuperior subluxation. In the setting of uncompensated rotator cuff dysfunction, coracoacromial insufficiency from prior surgery will severely compromise overhead function.[219] The incidence of this complication following rotator cuff repair is unknown, but when it occurs, the results are devastating and have historically been unsalvageable.[54,57,219] Patients who have poor preoperative function will often indicate that it is worse following surgery that results in anterosuperior humeral head subluxation. Their pain and dysfunction is often intolerable, even with activity modification and analgesics.[54,219]

Prevention of anterosuperior humeral head subluxation involves preservation of the coracoacromial arch during acromioplasty and rotator cuff repair, particularly in the presence of a large rotator cuff tear or a decreased preoperative acromiohumeral interval. While the indications for coracoacromial ligament preservation and repair continue to evolve,[55] this generally includes patients with two and three tendon tears, in whom the risk of a recurrent rotator cuff defect is sufficiently high to justify restoration of the coracoacromial arch. To facilitate repair of the ligament to bone, the anterior length of the acromion should be preserved while smoothing the undersurface of the acromion.[24,28,147,148] Some authors have advocated rotator cuff repair without performing an acromioplasty to avoid disrupting the integrity of the coracoacromial arch.[23,63,72] Clearly, partial excision of the coracoacromial ligament during acromioplasty has met with good results among patients with small rotator cuff tears, suggesting that repair of the coracoacromial ligament is not required in all rotator cuff repairs.[14,88,96,148,150,157,180,181] Although there may be a growing trend toward coracoacromial ligament preservation during both open and arthroscopic acromioplasty, there is currently little data in the literature to support routine repair of the ligament in small tears.

The operating surgeon should proceed cautiously when evaluating patients with marginal overhead function and complete obliteration of the acromiohumeral interval, as these patients are at high risk for deteriorating function following subacromial decompression and rotator cuff repair. If surgical intervention is contemplated in these patients, imaging studies such as magnetic resonance imaging and ultrasound will provide critical information regarding reparability of the rotator cuff. In the setting of an irreparable rotator cuff tear, a more conservative deltoid sparing open approach or arthroscopic decompression may be considered. Arthroscopic subacromial decompression usually requires subperiosteal release of a portion of the coracoacromial ligament, although the most medial portion or the acromial attachment can often be preserved. Studies indicate that the coracoacromial ligament may regenerate or heal, provided that the anterior length of the acromion is preserved.[84,123] Conservative arthroscopic decompression with biceps tenotomy may represent a reasonable alternative to conventional arthroscopic acromioplasty in patients with massive rotator cuff tears, and preserves the coracoacromial arch.[191]

The coracoacromial ligament can be released and repaired in one of two ways during open subacromial decompression. First, the deltoid insertion, subacromial bursa, and coracoacromial ligament can be released in a single layer and reattached to the acromion at the completion of the procedure. Alternatively, the interval between the deltoid attachment and the coracoacromial ligament can be dissected, releasing the deltoid from the anterior acromion. The underlying ligament and bursa can then be subperiosteally released from the undersurface of the acromion to maximize the length of the ligament. The bursal roof and ligament are then immediately sutured to the deep deltoid fascia and ultimately repaired to the anterior acromion as a single layer (Fig. 5-18). Tuberoplasty, as described by Fenlin and colleagues, represents an alternative to conventional open acromioplasty in patients with massive rotator cuff tears and may assist in preserving the coracoacromial arch.

Evaluation

The manifestations of anterosuperior humeral head subluxation are often apparent on clinical examination. With the arm at the side, the humeral head rests within the glenoid cavity. However, when the patient attempts to actively elevate the arm, the humeral head will rise out of the glenoid into a subcutaneous position anterior to the acromion. Full passive forward elevation is usually possible unless the anterosuperior subluxation is chronic and fixed. Due to the massive rotator cuff tear, the patient will be unable to raise the arm overhead and will demonstrate weakness in external rotation with the arm at the side and with the arm at 90 degrees of elevation in the scapular plane. Although the subscapularis may be involved, some inferior fibers may remain intact, leading to negative abdominal compression and lift-off tests. However, internal rotation strength will often be weak and the internal rotation lag sign may be present.[69,100] This complication is often also accompanied by deltoid insufficiency,[219] consisting of thinning, detachment, or atrophy associated with denervation.[61]

Radiographic studies are obtained to evaluate acromial bone loss and humeral head morphology and include

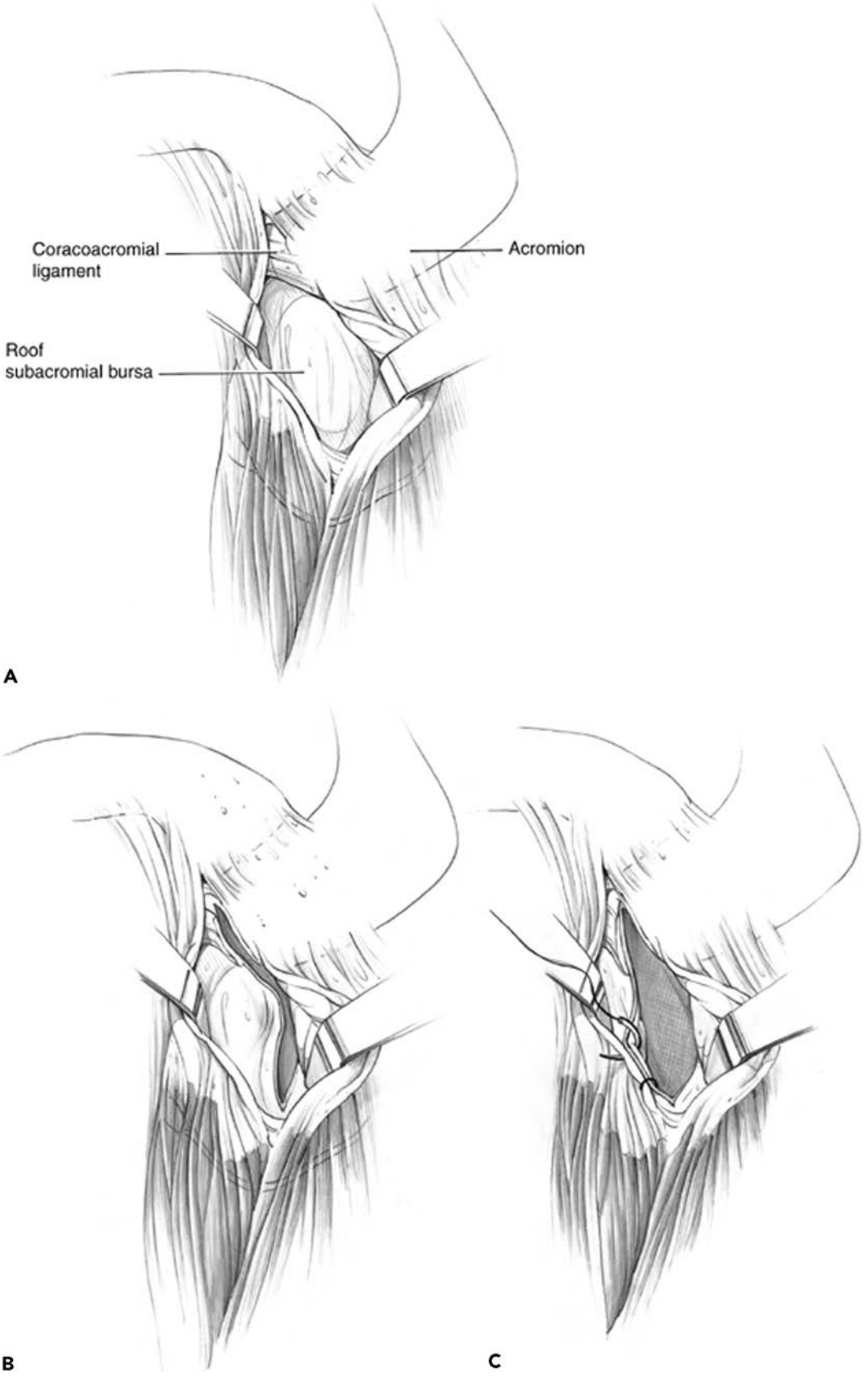

Figure 5-18 The coracoacromial ligament may be preserved during open rotator cuff surgery by reflecting the anterior deltoid in one layer, leaving the roof of the subacromial bursa and coracoacromial ligament attached to the acromion. **(B)** The roof of the bursa is then incised in line with the deltoid split, and the acromial attachment of the anterior portion of the bursal roof and the coracoacromial ligament are released from the anterior acromion. **(C)** The superior edge of the bursal roof and the acromial attachment of the coracoacromial ligament are sutured to the deep surface of the anterior deltoid fascia.

anteroposterior, axillary, 30-degree caudal tilt, and supraspinatus outlet views. Acromial bone loss is best appreciated on the 30-degree caudal tilt and axillary views, while the anteroposterior view will reveal humeral head articular changes associated with rotator cuff tear arthropathy, and static humeral anterosuperior subluxation. Magnetic resonance imaging is not required to confirm the diagnosis of anterior subluxation or recurrent rotator cuff tear, but will provide prognostic information regarding atrophy and fatty infiltration of the rotator cuff musculature. It will also facilitate treatment decisions regarding muscle transfer by indicating which muscles are still intact.[66,68]

Treatment

Surgical treatment of anterosuperior subluxation is at best a salvage procedure and consists of motion sparing and motion sacrificing options. Tendon transfers, coracoacromial arch reconstruction, and constrained arthroplasty are directed at preserving motion,[54,66,77,219,221] while arthrodesis is directed at relieving pain at the cost of glenohumeral motion.[41] A nonprosthetic motion sparing approach requires that the rotator cuff tear, coracoacromial insufficiency, or both be addressed. Since the rotator cuff tear is usually not repairable, reconstruction options to restore a balanced anterior and posterior force couple include partial repair, tendon transfers, or a combination of both. When the subscapularis is intact, latissimus dorsi transfer is performed to augment the posterior–superior force couple.[66] This transfer will not restore a balanced rotator cuff in the presence of subscapularis insufficiency and is therefore not indicated in this setting. Galatz and colleagues[61] have demonstrated success in treating anterosuperior subluxation that occurs in the setting of massive rotator cuff tear with subscapularis insufficiency. They described a dynamic transfer of the pectoralis major beneath the conjoined tendon to offset the loss of the subscapularis and the deltoid. Combined latissimus dorsi and pectoralis major transfers in the treatment of both anterior and posterior rotator cuff insufficiencies has been described,[3] but little data exist regarding this procedure in the treatment of anterosuperior subluxation. Coracoacromial ligament reconstruction has been described using either bone graft or autologous fascia lata.[54,219] Experience with either technique is relatively limited and the results uncertain. Flatow and colleagues[54] have combined soft tissue coracoacromial ligament reconstruction with appropriate tendon transfers to restore both dynamic and static restraints to anterosuperior subluxation. Patients were apparently not particularly ebullient regarding their results.

Although previous experience with constrained arthroplasty has met with disastrous results,[44,50,171] a new generation of inverse constrained prostheses have gained renewed interest in Europe, and more recently in the United States.[20,77,179] Indicated for anterosuperior subluxation or chronic pseudoparalysis, this reverse ball-and-socket joint appears to improve the moment arm of the intact deltoid, often allowing elevation above the horizontal plane. When this is combined with appropriate tendon transfers, patients may regain the ability to perform overhead functions and gain restoration of external rotation. Although technically demanding, this may prove to be a defining treatment option for patients with anterosuperior humeral head subluxation.

Arthrodesis will usually improve pain and increase stability and strength. It is reserved for patients with extreme pain who are willing to sacrifice glenohumeral rotation.[146] Although some patients with anterosuperior subluxation may be content to use the involved extremity as a helping hand with the arm at the side, others will perceive loss of glenohumeral rotation as a decrease in function. Patients should be carefully counseled regarding these limitations prior to recommending arthrodesis.

CONCLUSIONS AND TREATMENT ALGORITHM

Rotator cuff surgery is generally safe and efficacious. However, when complications occur, they are frequently accompanied by recurrent symptoms that may be worse than the original preoperative complaints that prompted the patient to seek medical attention in the first place. Reported results of surgical treatment of these complications are inconsistent and the incidence probably underreported. Surgical treatment of complications of rotator cuff surgery is demanding and requires both a motivated patient and a knowledgeable surgeon. It is impossible to establish rigid patient selection criteria and treatment protocols for all types and combinations of complications of rotator cuff surgery. However, adoption of a systematic approach that is based on known anatomic observations and sound surgical principles affords the best opportunity for a successful outcome. One algorithmic approach to the management of patients with complications following rotator cuff surgery is depicted in Fig. 5-19.

Several principles involved in the development of this algorithm deserve emphasis. First, the presence of significant stiffness interferes with the interpretation and clinical significance of any associated complications. Frequently, patients with documented recurrent rotator cuff defects will present with an inability to either actively or passively elevate their arm further than 90 degrees. If these patients had normal arcs of motion prior to their index procedure, restoration of normal or near-normal passive motion will frequently be accompanied by significant gains in active elevation. The patient may be satisfied with his or her shoulder function after simple arthroscopic capsular release and be willing to forego more complicated surgical procedures, particularly if his or her recurrent rotator cuff defect is irreparable.

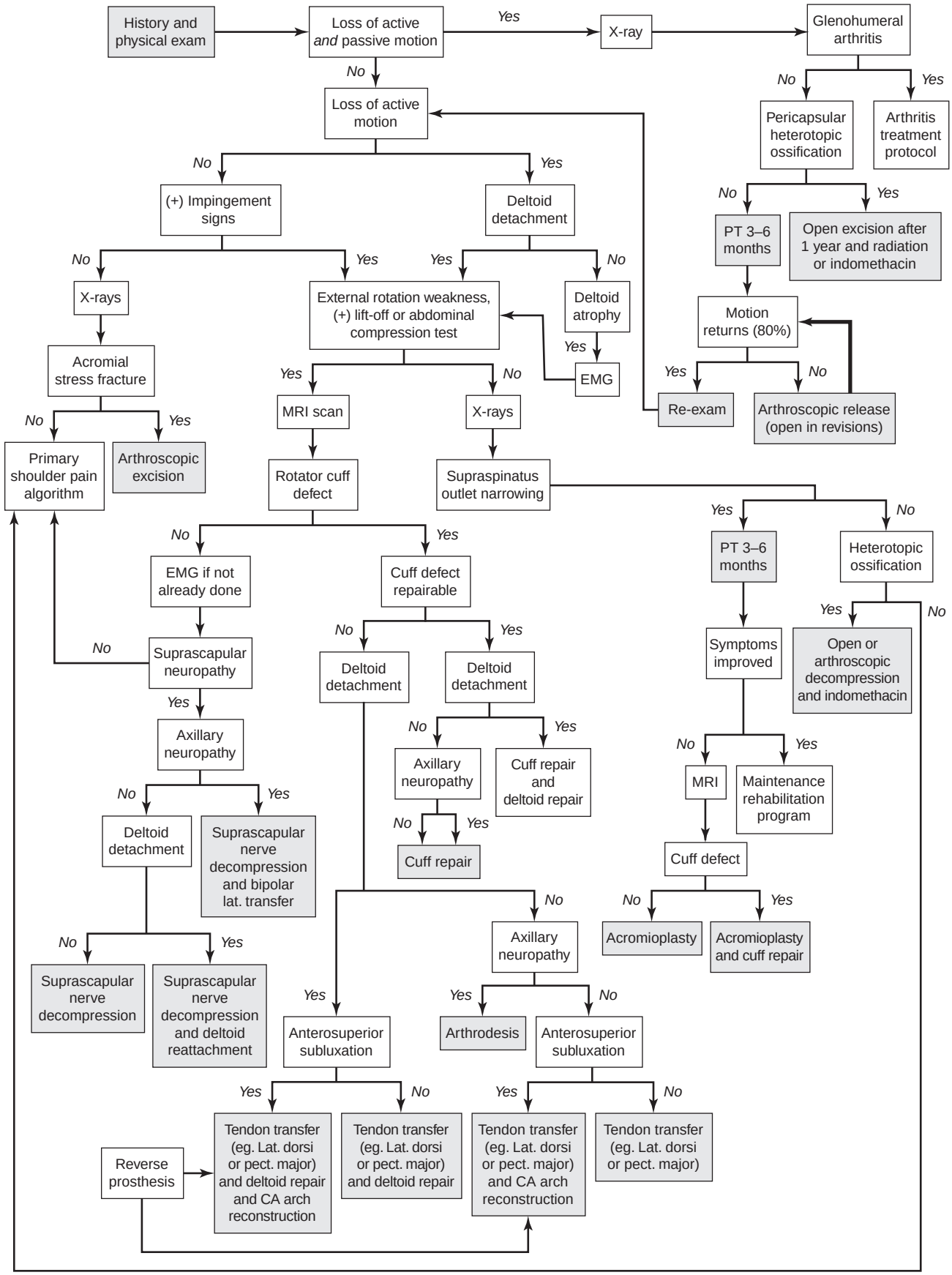

Figure 5-19 Treatment algorithm for the management of complications following rotator cuff surgery.

Loss of active elevation in the presence of normal or near-normal passive motion may indicate deltoid insufficiency, rotator cuff insufficiency, or both. Furthermore, the presence of pain and voluntary guarding will interfere with the interpretation of physical findings. This is particularly true for strength assessment of various portions of the rotator cuff. Subacromial lidocaine injections may decrease pain and improve the reliability of strength and functional assessments of the rotator cuff, and is frequently utilized in the diagnosis of patients with continued pain and dysfunction following rotator cuff surgery.

Ultimately, the final outcome among patients who have developed postoperative complications following rotator cuff surgery is affected by multiple factors. Assuming that the original diagnosis was correct, the goals of revision surgery are to reestablish passive motion; to restore a balanced anteroposterior force couple by rotator cuff repair, partial repair, or tendon transfer; and to preserve or restore deltoid function. The importance of coracoacromial arch preservation, especially among patients with massive rotator cuff tears, cannot be overemphasized. While the results of coracoacromial arch reconstruction have been disappointing, inverse prosthetic replacement, with or without tendon transfer, may represent a reasonable salvage procedure. Clearly, when reviewing the results following complications of rotator cuff surgery, the most efficacious method of ensuring patient satisfaction is prevention.

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Calcifying Tendinitis

6

Shawn P. Hennigan *Anthony A. Romeo*

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PATHOPHYSIOLOGY

Introduction

Calcifying tendinitis of the rotator cuff is a disease of unknown cause that is characterized by multifocal, cell-mediated calcium deposition in viable tissue. These deposits normally undergo spontaneous resorption followed by subsequent healing of the tendon. The clinical presentation is variable; patients may have little or no symptoms during the formative phase, or they may have acute symptoms during the resorptive phase.

Definition and Classification

Calcifying tendinitis of the rotator cuff tendons differs from dystrophic calcification or other rotator cuff tendinopathies in several ways. First, calcifying tendinitis occurs in vascularized, viable soft tissue, whereas tendinopathies tend to occur in dysvascular, nonviable soft tissues. Next, calcifying tendinitis is a disease that proceeds toward resolution. Degenerative tendinopathies tend to worsen over time. It is unusual to see other signs of degenerative changes in calcifying tendinitis, whereas they are commonplace in dystrophic calcification and tendinopathies. Another difference between calcifying tendinitis and dystrophic calcification is in the radiographic location and appearance of the calcification. In dystrophic calcification, the calcifying deposits tend to occur at the insertion site of the tendon into the bone and are commonly stippled in appearance. In calcifying tendinitis, the calcification tends to occur 1 to 2 cm proximal to the insertion site of the tendon, and within the midsubstance of the tendon (Fig. 6-1).

Duplay first recognized the subacromial bursa as a source of shoulder pain in 1871.[28a] He coined the term "scapulohumeral periarthritis." In 1907, Painter described subacromial calcium deposits, and felt that these deposits were the primary source of pathology.[101] There were later descriptions by Bergemann and Steida in 1908, who also believed that the subacromial bursa was the primary site of pathology.[10] In fact, this was the opinion of many authors at the beginning of this century. Codman, in 1909, was one of the first to demonstrate that the calcium deposits were located in the tendons of the rotator cuff, and not in the subacromial bursa.[21] In 1912, Wrede was

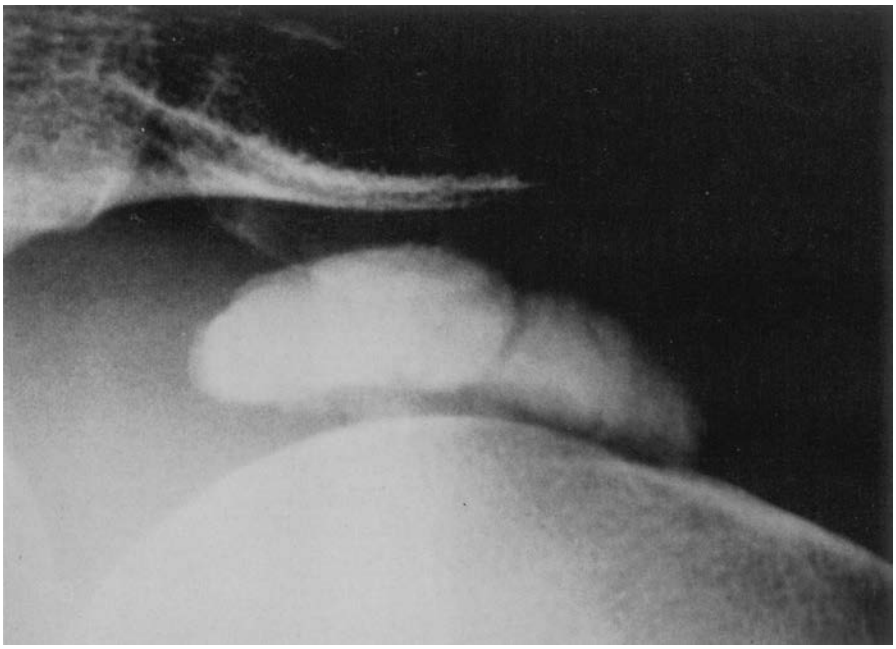

Figure 6-1 The typical location of the calcific mass is within the midsubstance of the tendon, not at its insertion site. Calcifying tendinitis most commonly affects the supraspinatus tendon. (Courtesy of Dr. Gilles Walch.)

credited with the first histologic description of the disease of calcifying tendinitis. He identified the presence of chondrocytes among tenocytes within the tendon.[149] The intratendinous location of the calcified deposits was later confirmed by other authors, such as Schaer in 1936,[126] Sandstrom in 1938,[123] and McLaughlin in 1946.[81] These authors all believed that calcifying tendinitis was the result of a degenerative process. Bateman observed that calcium deposits occurred close to the site of tendon attachment that is now known as the "critical zone," an area that may have diminished vascularity. He thought that "abnormal aging of collagen fibers initiated the calcification mechanism."[8] This belief persisted despite the observation in 1937 by Sandstrom and Wahlgren that calcification occurs in viable, not necrotic, tissue.[124] Moseley, in 1963, stated that "at operation, I have noted that the tendon was well vascularized around the area of deposit and therefore disagree with the idea that the calcium salts were precipitated in an area of previous disease with resultant ischemia."[92]

DeSeze and Welfling in 1970 coined the term "tendinites calcifiantes," denoting an evolutionary process of calcifying tendinitis tending toward spontaneous healing.[26] This differs in many ways from degenerative tendinopathy and now appears to be the description of choice for calcifying tendinitis. Uhthoff, in 1975, elucidated the pathogenesis of calcification, correlating histologic findings with clinical symptoms.[135]

Codman, in his classic text, pointed out that disease in the supraspinatus tendon tended to appear in a specific area of the tendon about "half inch proximal to the insertion degenerative of the tendon." He called this area the "critical portion."[21] This area was later renamed by Moseley and Goldie as the "critical zone."[93] Multiple studies of vascularity of the rotator cuff tendon by microangiographic techniques have been performed. The critical zone is a watershed area within the supraspinatus tendon, with its blood supply coming from the proximal muscular portion of the supraspinatus as well as from the osseotendinous junction. This may account for a relative decrease in perfusion. This was supported by studies performed by Rothman and Parke.[120] However, Moseley and Goldie thought that this area had a rich anastomosis.[93]

Rathbun and Macnab, in 1970, performed cadaveric studies to study the vascularity of the supraspinatus tendon.[112] They found that the perfusion to the critical zone in the supraspinatus tendon was largely dependent on the arm position. The critical zone was subject to a "wring-out" phenomenon, from pressure on the tendon exerted by the humeral head with the humerus in the adducted position.

Terminology

The terminology surrounding calcifying tendinitis has also evolved along with a better understanding of the pathogenesis. In 1907, Painter first used the term "calcifying bursitis."[101] This was later used by Bergemann and Steida in 1908,[10] and again by Steida in 1908.[133] In 1937, Sandstrom coined the term "peritendinitis calcarea."[124] The first appearance of the term "calcifying tendinitis" appeared in the North American literature in 1952 by Plenk.[108] DeSeze and Welfling coined the term "tendinite calcifiante" in 1970.[26] In 1979, Dieppe referred to this condition as calcified peritendinitis.[27] The term "tendinitis" is preferred over

tendinosis because of the acute nature of symptoms and the tendency toward complete resolution. "Tendinosis" is more appropriately used for conditions of a degenerative nature in which symptoms are chronic and tend toward a gradual deterioration over time.

Classification

There are many classification schemes created for calcifying tendinitis of the rotator cuff, some based on clinical presentation and others based on radiologic findings. DePalma and Kruper divided calcifying tendinitis into three categories based on the presenting symptoms: acute, subacute, and chronic.[25] Bosworth classified calcifying tendinitis by the radiographic size of the deposits.[15]

Patte and Goutallier described two forms of calcifying tendinitis: localized and diffuse. Localized deposits were discrete, dense, and homogeneous in density, and they had a tendency to heal spontaneously. In contrast, the diffuse form tended to appear "fluffy" on radiograph, was heterogenous in density, and tended to heal more slowly. Patients with the diffuse type were believed to have a higher likelihood of clinical symptoms.[102]

Uhthoff and Sarkar proposed that calcifying tendinitis is a dynamic disease that progresses through a complete cycle in most patients. Calcifying tendinitis has two distinct pathologic phases: the formative phase and the resorptive phase. Contained within these two phases are three distinct stages of calcification: the precalcifying stage, the calcifying stage, and the postcalcifying stage[142] (Fig. 6-2).

Other causes of calcification about the shoulder are numerous, including arteriosclerosis of brachial and axillary vessels, calcified soft tissue or bony neoplasms, patients with chronic renal failure, heterotopic ossification following acromioplasty and distal clavicle resection, subdeltoid calcifying bursitis in rheumatoid arthritis, and chronic acromioclavicular separation with calcification of the coracoclavicular ligament, as well as in diabetes.[1,2,9,11,27,41,47,59,78,79,89,107,126] Other distinct causes of calcification about the shoulder include the "Milwaukee" shoulder. This is characterized by massive calcifications in association with a complete rotator cuff tear, severe glenohumeral arthritis, acromioclavicular arthritis, or some combination thereof. Dystrophic calcifications are seen at the torn ends of the rotator cuff tendon after a complete tear.

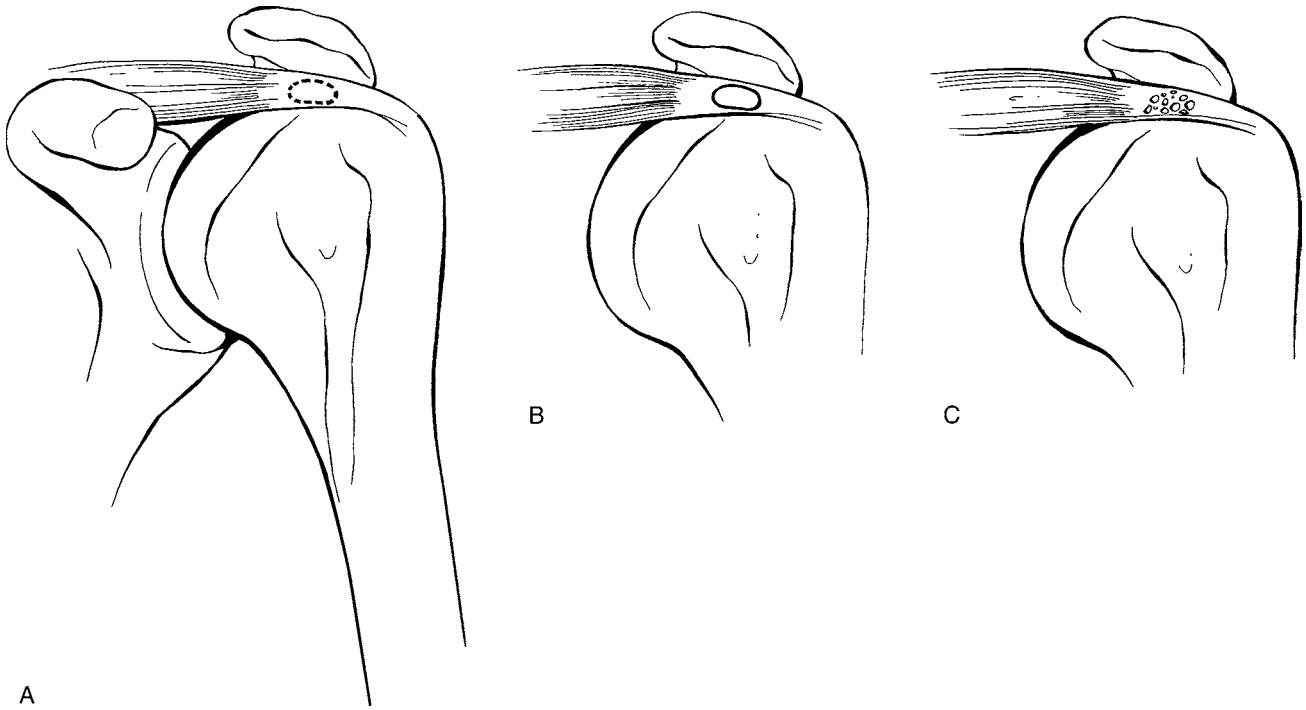

Figure 6-2 The calcification of the rotator cuff tendon progresses through three distinct stages: **(A)** The precalcific stage demonstrates a metaplasia of the tenocytes into chondrocytes. Degenerative changes of the tendon are absent. The signal for this metaplasia is unknown. **(B)** The calcifying stage begins with the formative phase, with small foci of calcium deposits that can coalesce into a large homogeneous deposit. The absence of a vascular response distinguishes this calcification process from endochondral ossification. **(C)** The postcalcific stage begins with resorption of the calcific deposit and ends with reconstitution of the normal tendon.

Pathogenesis

Considerable controversy still exists over the cause of calcifying tendinitis. Codman initially proposed that calcifying tendinitis is a degenerative process of the rotator cuff tendon. He believed that the fibers of the tendon degenerate, become necrotic, and then develop dystrophic calcification.[21] Other authors accepted this concept. In 1946, McLaughlin pointed out that the earliest lesion in rotator cuff tendon calcification is a focal hyalinization of fibers that eventually become fibrillated, detached from the tendon, and wound up into rice-like bodies that undergo calcification.[81] This theory was further supported by an experimental study performed by Macnab. He was able to show that interruption of the vascular supply to the Achilles tendon in rabbits can produce the hyalinization of the tendon fibers, followed by calcification.[72] Brewer has shown that as the supraspinatus tendon ages, its vascularity diminishes. By about the fourth or fifth decade, many fascicles in the tendon have thinned and fibrillated. The natural senescence of rotator cuff tendons with decreased vascularity is suggested as a contributing factor to the pathogenesis of calcifying tendinitis.[17]

In contrast, Uhthoff et al. have pointed out that there are various aspects of calcifying tendinitis that are not consistent with a degenerative process.[135,138] In calcifying tendinitis, the calcification occurs in apparently viable tissue. Degenerative calcification occurs in nonviable or poorly vascularized tissue. The peak incidence of calcifying tendinitis is in the fourth and fifth decades in most studies. The incidence of calcifying tendinitis is rare in the sixth decade. Calcifying tendinitis has not been reported in patients who were older than the age of 71. This would include autopsy findings on patients older than 71, including those with a previous history of calcifying tendinitis. These well-documented characteristics of calcifying tendinitis support the concept that this distinct clinical entity is a reparative process progressing through a predictable disease cycle.

Uhthoff and Sarkar have divided the typical cycle of calcifying tendinitis into three distinct stages: precalcific, calcifying, and postcalcific. The precalcific stage is marked histologically by metaplasia of the tenocytes into chondrocytes. This is accompanied by increased proteoglycan formation. The cause for the initiation of this process is unknown. Codman suggested that hypoxia may play a role in initiating this process,[21] but other catalysts may include microtrauma, disuse, hormonal factors, or dietary factors. The formative phase marks the beginning of the calcification, the hallmark of which is calcium deposition, primarily into matrix vesicles within the chondrocytes. Histologic examinations reveal that, during the formative phase, the surrounding tissue is relatively avascular and is marked by the absence of vessels. This process of calcification is distinctly different from endochondral bone formation, which is marked by the presence of blood vessels at the time of calcification. Generally, the foci of calcium deposits coalesce to form large deposits until a very dense homogeneous and typically well-delineated calcium deposit is formed. Clinical symptoms are often absent. However, when clinical symptoms accompany the formative stage, the findings are typically similar to patients with mild to moderate subacromial impingement.[76] The lack of hyperalgesia and the ability to use the shoulder for many activities may not lead to medical evaluation and confirmation of the diagnosis. Intermittent worsening of the symptoms, including diffuse pain, difficulty with overhead activities, and rest pain, defines the clinical picture of "chronic" calcifying tendinitis. At this stage, plain radiographs for the evaluation of nonspecific shoulder pain may detect a calcific deposit within the rotator cuff tendon, most typically the supraspinatus tendon.[39] During the formative phase, the calcium deposit exists primarily as a chalky deposit that is well demarcated within the tendon. The duration of the formative phase is variable, and it may last for many years.

The proliferation of blood vessels at the periphery of the calcium deposit heralds the phase of resorption. With the vascularization process, cellular infiltration occurs with macrophages, mononuclear giant cells, and fibroblasts initially seen at the periphery of the calcific deposit. These cells mediate an aggressive inflammatory process, releasing various cellular mediators and enzymes that break up the calcium deposit and phagocytose the calcium. With the vascular and cellular infiltration comes an obligate increase in the intratendinous pressure, largely as a result of the edema, which causes the classic acute hyperalgesia of calcifying tendinitis. The pressure may be so great that the deposit ruptures into the subacromial bursa, or into the area external to the subacromial bursa in the subacromial space. Roentgenographically, the calcium deposit now appears poorly delineated and fluffy. Concomitant with the inflammatory response, fibroblasts lay down collagen in the cavity left behind by the resolving calcium deposit. As the collagen matures and the resorptive process proceeds to conclusion, the fibers line themselves along the axis of the tendon, allowing return of the tendon structure, and eventual tendon function.

The postcalcific stage is marked by fibroblasts laying down collagen, primarily type III, which later remodels into type I collagen. This final stage completes the natural cycle of calcifying tendinitis. There is speculation, but no firm evidence, that the healed tendon may be more susceptible to degenerative tearing over time (Fig. 6-3).

Incidence

The incidence of calcifying tendinitis varies with diagnostic techniques, the patient population being studied, and the geographic location. At a recent meeting, North American

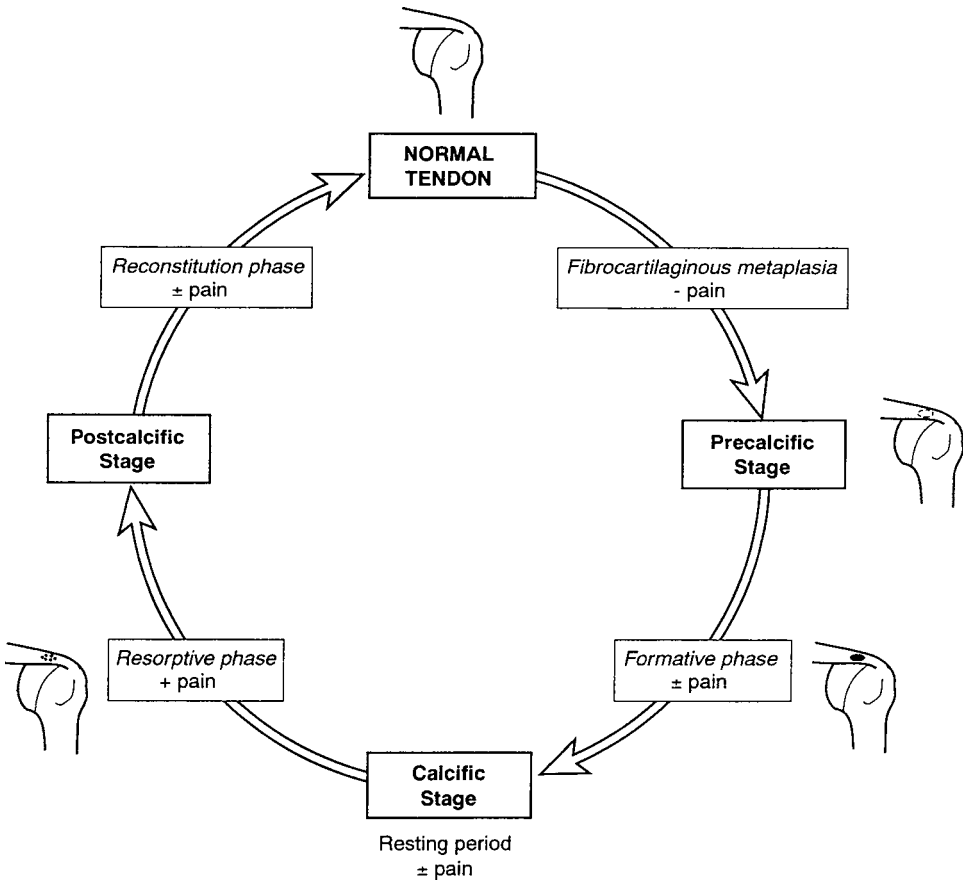

Figure 6-3 Summary of the natural cycle of calcifying tendinitis.

shoulder surgeons reported an overall decrease in incidence of calcifying tendinitis.[142] In fact, Neer commented that surgical treatment of calcifying tendinitis was one of the most common procedures performed at his teaching hospital when he was a resident.[96] However, surgical treatment and the diagnosis of this disorder has become less common owing to factors that are not understood. The reported incidence of calcifying tendinitis seen by European and Japanese surgeons has not significantly changed during the same time period.

Bosworth reported a 2.7% incidence of calcifying tendinitis among 6,061 asymptomatic office workers.[14] In a report from France, Welfling et al. reported an incidence of 7.5% of the disorder among 200 asymptomatic shoulders. However, in 925 symptomatic shoulders, there was a 6.8% incidence.[146] Ruttimann reported a 20% incidence in 100 asymptomatic shoulders.[122] Regional variation suggests that hereditary and dietary factors may play a role in the predisposition toward calcifying tendinitis.

Most authors report that the highest incidence of calcifying tendinitis occurs between the ages of 40 and 50 years. Thirty-six percent of the patients in the study by DePalma and Kruper[25] fell into this age category, as did 42% of the patients in a report by Uhthoff and Sarkar.[137] Welfling et al., on the other hand, reported the highest incidence between the ages of 31 and 40 years. No patient was older than the age of 71 years.[146] Nutton and Stothard reported a case of a 3-year-old child with acute calcific supraspinatus tendinitis.[99] Recently, Hsu et al., reporting on their patient population from Taiwan,[53] reported that 69% of the patients in their study were older than 60 years of age, the first report to suggest that calcifying tendinitis may have a distinct clinical variant in an Asian population, with its onset in an older-aged group.[53] Furthermore, the significant incidence of associated rotator cuff tears in Hsu et al.'s study is in distinct contrast with other studies of calcifying tendinitis.

The calcifying deposits are most commonly located in the supraspinatus tendon. Plenk reported an 82% incidence in the supraspinatus tendon.[108] In Bosworth's report, the supraspinatus was involved in 51% of his patients, the infraspinatus in 44.5% (Fig. 6-4), the teres minor in 23.3%, and the subscapularis in 3% (Fig. 6-5).[14] DePalma and Kruper reported that the supraspinatus was involved in 74% of their cases.[25] Hsu et al. reported a similar 70.7% involvement of the supraspinatus, with 26.8% involving the infraspinatus.[53]

There is still some controversy over the role of the subacromial bursa in calcifying tendinitis. It is commonly believed, however, that the subacromial bursa is not a

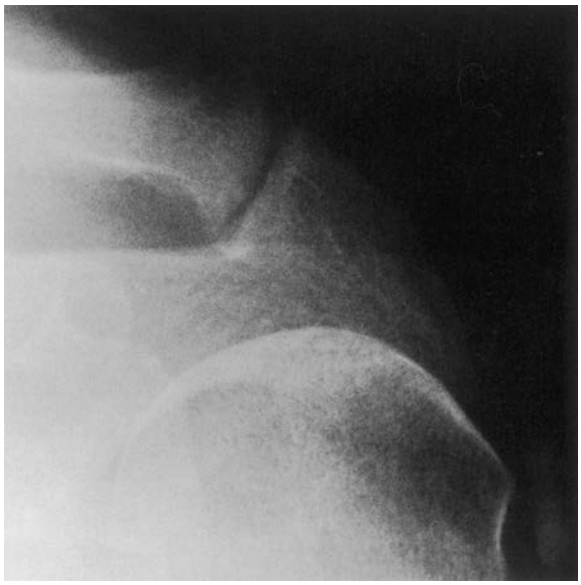

Figure 6-4 Calcifying tendinitis involving the infraspinatus tendon, which occurs in approximately one-third of patients with calcifying tendinitis. (Courtesy of Dr. Gilles Walch.)

factor in the chronic phase of this disorder. This belief is supported by Carnett, who noted that bursitis forms a minor and infrequent feature of chronic calcifying tendinitis.[19] Ishii et al. noted bursal reactions in subacromial bursal specimens removed at the time of surgery from 63 patients, 6 of whom had calcifying tendinitis. Their findings clearly showed that the strongest bursal reaction was present with a rotator cuff tendon tear, and was weakest in calcifying tendinitis.[55] Pedersen and Key looked at the pathology of calcareous tendinitis and subdeltoid bursitis, and found that at surgery the bursa was thin and translucent, light pink, and without evidence of acute inflammation. They did note some free calcium material in several instances. However, in each of these cases, there was also roentgenographic evidence of rupture of the calcium deposit into the bursa. As they stated, "unless there was free material in the wall of the bursa, this was not the site of gross inflammatory change."[104] Sarkar and Uhthoff stated that "it has been known for a long time that calcification of rotator cuff tendons occurs primarily in the tendon substance and not in the bursa."[125] Thus, it appears the bursa may become involved secondarily from either impingement, with subsequent inflammation of the bursa, or from rupture of the calcifying deposit into the bursa, with secondary inflammation.

In the gender of the patients, most authors agree that there is a higher incidence among females than males. Uhthoff and Sarkar reported a 57% incidence of female involvement,[137] while Bosworth reported a 76.7% incidence.[14] Welfling et al.[146] reported that 62% of his patients with calcifying tendinitis were female, and 64% of the patients in the study by Lippmann were female.[67] DePalma and Kruper also reported that 60.3% of their patients were female.[25] However, Hsu and his collaborators reported only a 26% incidence of female patients, again drawing attention to the distinct difference in their Asian patient population and possibly the clinical condition they have reported.[53]

Calcifying tendinitis may be related to certain occupations or activities. DePalma and Kruper reported that 41% of their patients were housewives and 27% were executives

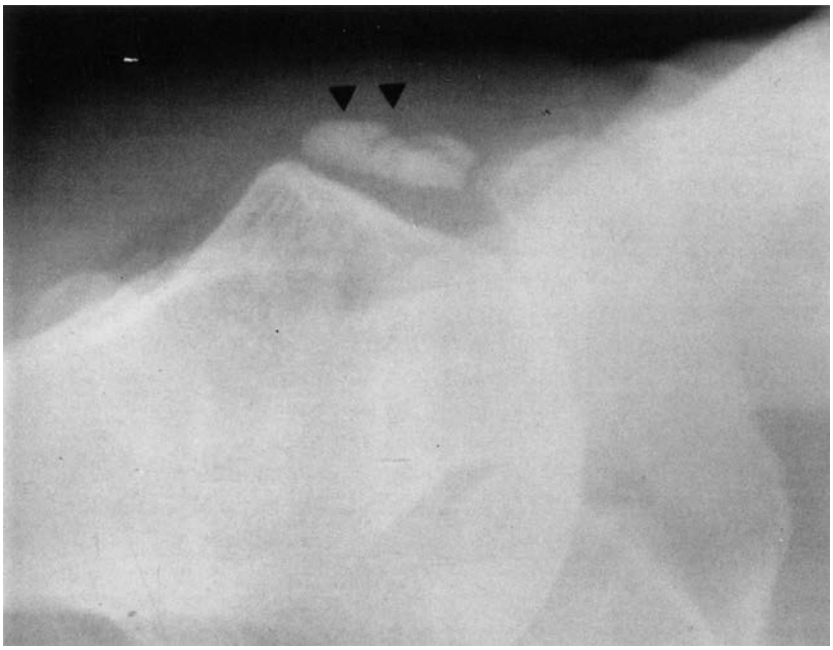

Figure 6-5 Calcifying tendinitis involving the subscapularis tendon. Because the deposit is superimposed on the humerus with an anteroposterior radiograph, this rare situation can be easily missed if an axillary lateral radiograph is not obtained. (Courtesy of Dr. Gilles Walch.)

and salespersons.[25] Uhthoff and Sarkar reported that 43% of their patients were housewives and 44% were clerical workers.[137] It appears that heavy laborers suffer less from calcifying tendinitis than do sedentary workers or workers who primarily perform nonstrenuous tasks with their elbow at their side. There does not appear to be a direct relation between trauma and calcifying tendinitis.

The incidence of bilateral involvement increases with the length of follow-up. Welfling et al. reported an incidence of 24.3% of bilateral calcifying tendinitis of the shoulder.[146] DePalma and Kruper reported a 13% incidence of bilateral calcifying tendinitis,[25] and Uhthoff and Sarkar reported a 17% incidence.[137]

In some instances, calcifying tendinitis appears to be part of a systemic disease.[107] This is supported by the incidence of calcifications occurring at other sites, most notably around the hip. No other study has suggested a similar incidence of hip involvement. Welfling et al. reported that 62.5% of his patients with calcifying tendinitis of the shoulder had simultaneous calcifications around the hip, whereas in the control group, only 4% had calcifications about the hip.[146] Gschwend et al. were unable to prove an association with diabetes or gout.[45] However, other authors feel that there is an increase in the incidence of calcifications in many areas of the body in patients with diabetes, including the shoulder region.[59,78] Abnormalities in calcium or phosphate metabolism have not been reported as part of the clinical syndrome of calcifying tendinitis, although they are clearly evident with other conditions leading to soft tissue calcifications, such as renal osteodystrophy.[2] There have also been attempts to correlate HLA-A1 presence with calcifying tendinitis, although the role for HLA testing in diagnosis or management of patients is unclear.[130]

Clinical Presentation

The clinical presentation is highly variable. An understanding of the pathogenesis of calcifying tendinitis allows the clinician to make proper diagnostic as well as management decisions. The formative phase of calcification is frequently subclinical or even asymptomatic. It is often discovered serendipitously. As Baer observed during surgery, large deposits may lead to an impingement syndrome.[5] In contrast, the resorptive phase, which occurs later in the cycle of calcifying tendinitis, is commonly characterized by acute symptoms. The pain seen at this stage is related to the increase in intratendinous pressure from the vascular proliferation, influx in inflammatory cells, edema, and swelling. As tendon volume increases, the unyielding dimensions of the subacromial space may add additional pressure to the involved tendon, leading to evidence of secondary impingement.[135]

Bursitis has been suggested as a source of pain. However, during surgery, bursal reaction is minimal and is localized to a focal area with hyperemia. It is usually not severe enough to cause a bursal thickening. The rupture of the calcific material into the bursa may cause a crystalline-type bursitis. However, DeSeze and Welfling reported 12 patients who had rupture of the calcified deposit into the bursa, only eight of whom showed symptoms.[26]

Many authors have stressed the typical subclinical nature of the formative phase. Codman noted that the "usual history is not acute pain in the beginning."[21] Gschwend et al. observed that the calcification is often asymptomatic at the beginning, but its disappearance is marked with acute pain.[45] Wilson stated that many patients know about the calcium deposit for months to years before an acute attack.[147] Rowe broke down the clinical course of patients with calcifying tendinitis into the silent phase, the impingement phase, and the acute phase.[121] Pinals and Short,[107] Booth and Marvel,[13] Simon,[131] Re and Karzel,[113] and Bosworth[15] provided similar descriptions.

Most authors discuss two primary clinical phases—acute and chronic—with some variation. The subacute or chronic phases are characterized by mild symptoms, whereas the acute phase has more severe symptoms.[33,38,146] DePalma and Kruper divided the clinical presentation of patients into three groups, based on the duration of symptoms. The acute cases had severe shoulder pain for less than 4 weeks duration, the subacute cases had more mild symptoms lasting anywhere between 1 and 6 months, and the chronic cases were those in which patients had mild symptoms present for more than 6 months.[25] Jones observed that the onset of pain in patients with calcification of the supraspinatus tendon was very sudden and severe and usually not precipitated by injury. He stated that "there is inability to rest or sleep and sometimes patients have become mentally strange after several days of such pain."[57] He was most likely describing the presentation of hyperalgesia related to the resorption phase of calcifying tendinitis. Lapidus coined the term "dormant" deposits with which patients may be without symptoms, except for occasional aches and pains. He suggested that rupture of the tendinous tissue into the floor of the bursa, with extrusion of the calcifying material, was the cause of the developing acute symptoms.[64] Moseley described calcifying tendinitis in terms of four distinct phases: a silent phase, a mechanical phase, followed by rupture of the calcified material into the subbursal space, and finally intrabursal rupture of the calcific deposit.[91] Lippmann described a phase of increment and a phase of disruption. The phase of increment correlates with very mild symptomatology. Radiographic appearance is discrete and homogeneous in density. The phase of disruption, however, is marked by severe pain and a radiographic appearance that is heterogeneous and fluffy.[67] As implied from these various clinical classification schemes, the pathologic stages of calcifying tendinitis correlate with the clinical presentation, with mild symptoms common with the formative stage, but severe symptoms being typical of the onset of the resorptive phase.

The intensity of the pain may be very severe with the acute clinical phase. Initially, rupture of the calcified deposit into the bursa was presumed to be the cause of these symptoms. However, DeSeze and Welfling documented acute symptoms in only 8 of 12 patients who had roentgenographic evidence of rupture of the calcium deposit into the bursa. They postulated that the acute symptoms appear to be related to the increase in intratendinous pressure caused by vascular proliferation, inflammatory cell accumulation, and edema. Their explanation is supported by the common intraoperative observation that the calcium deposit literally spurts from the tendon when the tendon is incised during the acute clinical phase.[26]

Calcifying tendinitis should be understood as a continuum, not two distinct subgroups of a disease. Uhthoff and Sarkar stress that "the calcifying deposit must first be formed and thereafter be removed before the tendon is reconstituted." In their experience, the process of formation is usually without symptoms. Serendipitous roentgenographic discovery is common. However, during the stage of resorption, symptoms are usually severe, corresponding to the acute phase in other classification schemes. Once the resorptive phase has resolved, the tendon reconstitutes its fibrous architecture in this self-healing condition, completing the cycle of calcifying tendinitis.[137]

The duration of symptoms correlates with their acuity. The more acute the symptoms are, the shorter the duration of symptoms is. Simon reported a definite relation between the intensity of symptoms and duration. The acute symptoms last up to 2 weeks; subacute 3 to 8 weeks; and chronic more than 3 months.[131] DePalma and Kruper stated that the acute symptoms last less than 1 month, subacute between 1 and 6 months, and chronic longer than 6 months.[25] Booth and Marvel stated that "acute calcifying tendinitis is a self-limited process with a natural history of 6 to 14 days."[13] According to Carnett, the chronic cases "ran a self-limiting course and irrespective of treatment, the patient made a complete recovery at varying periods from a few months up to 3 years."[19] Codman believed that few cases were exceedingly acute, and that most cases of calcifying tendinitis ran a subacute course over months to years.[21] In Howorth' s series, the duration of symptoms in his patients with calcifying tendinitis was 2 days to 20 years, with an average of 2 years. Only 24 patients out of a consecutive series of 100 patients had symptoms of less than 1 month's duration. Nearly all of the others came in because of intermittent exacerbations of pain.[52] Pendergrass and Hodes made the observation that acute symptoms subside in less than 2 weeks, even in the absence of treatment.[105] The importance of the relation between the acuity of symptoms and their resolution becomes paramount when discussing intervention and the "success" of treatment because it appears that most patients presenting with acute hyperalgesia will improve even without treatment.

The location of pain is typically referred to the insertion of the deltoid in over half of the patients. This referral pattern, common with supraspinatus tendinopathy, reflects the frequent involvement of the supraspinatus tendon with calcifying tendinitis. Less commonly, the pain radiates toward the neck. Most patients give a history of difficulty with overhead tasks, inability to sleep on the affected shoulder, or increasing pain during the night that often prevents sleep. Other complaints may include shoulder stiffness, less strength with the arm away from their side, or mechanical complaints, such as "snapping" or "catching" of the shoulder.

Physical Examination

Physical examination characteristics are dependent on the phase of presentation. With the subacute or chronic phase, examination findings often mimic subacromial impingement, with mild decreases in range of motion and a "positive" impingement sign.[76] Kessel and Watson described the "painful arc of motion," with symptoms of pain elicited with motion of the arm between 70 and 110 degrees of elevation. The painful arc most commonly relates to subacromial pathology.[62] Patients may have a sensation of painful catching caused by a localized impingement of the calcified mass on the coracoacromial arch. The chronic phase of calcifying tendinitis may also present with supraspinatus and infraspinatus atrophy.

During the acute phase, pain is frequently intense, with patients often stating that they have never felt more severe pain. The severe pain leads to guarding against any motion, for they fear the examination may worsen their symptoms. Even if they allow motion, the glenohumeral motion and even the scapulothoracic motion may be severely limited by muscular spasm. Strength testing is prohibited by the pain. Provocative tests, such as the impingement sign, are impossible because of the loss of motion. This "pseudoadhesive capsulitis" is distinguished from idiopathic frozen shoulder by the severity of the pain, by the acute and rapid onset of the pain, and by the radiographic findings. The severity of the pain and the shoulder examination findings may also be confused with acute brachial plexitis (Parsonage-Turner syndrome), but is distinguished by the lack of pure neurologic findings and sparing of the elbow, wrist, and hand function.

Imaging Studies for Calcifying Tendinitis

The calcium deposits are localized to an area within a tendon that is approximately 1 to 2 cm from its insertion site on the tuberosity of the humerus.[21] The pattern of the deposit may be characteristic for the stage of calcifying tendinitis; therefore, a full understanding of the pathogenesis is essential to proper radiologic interpretation.

When a diagnosis of calcifying tendinitis is suspected, radiographs of the shoulder are mandatory. Radiographs can make or confirm the diagnosis, suggest the pathologic phase, and permit radiographic follow-up. Calcifying tendinitis can be suggested by the clinical features discussed, including the age of the patient, the pattern of symptoms, and examination findings. Furthermore, in patients presenting with findings suggestive of subacromial abnormality, a careful review of the radiographs is important. Harbin pointed out that a diagnosis of bursitis was frequently made in cases of calcifying tendinitis because of the failure to recognize the calcification on roentgenograms.[48]

Although many radiographic views of the shoulder may be helpful for specific conditions, routine radiographs must include a true anteroposterior view of the shoulder and an axillary lateral one.[77] Other views are directed by the history and the findings on clinical examination. Although many cases of calcifying tendinitis will be detected with a true anteroposterior view of the glenohumeral joint, additional anteroposterior films, with the shoulder in internal as well as external rotation, are recommended to see the tuberosities and the tendon insertions in multiple profiles. Anteroposterior radiographs with the humerus in neutral rotation show the greater tuberosity in profile, thereby bringing any calcium deposits within the supraspinatus tendon into view. Calcifications within the subscapularis tendon are detected with an anteroposterior view of the humerus in external rotation. With the humerus in internal rotation, precise localization of calcium deposits in both the infraspinatus and teres minor can be made based on their relative location on the greater tuberosity. If calcification is present in the middle one-third of the greater tuberosity, the infraspinatus tendon is likely involved. If the distal one-third of the greater tuberosity is involved, the teres minor is involved. These calcifications are frequently missed on standard anteroposterior radiographs owing to superimposition of the calcification over the proximal humerus (Fig. 6-6). In addition, calcifications within the long head of the biceps are frequently seen adjacent to the upper portion of the glenoid, and the position of these deposits is unchanged by external or internal rotation of the humerus. Calcifications in the region of the lower portion of the glenoid may represent involvement of the short head of the biceps or the triceps. Rupture of the calcium into the subacromial bursa may give a "skullcap appearance."[131] A supraspinatus outlet view is helpful in determining whether a deposit is in the supraspinatus or infraspinatus and whether it is encroaching on the coracoacromial arch (Fig. 6-7).

In 1934, Codman reported the presence of a pyramidal shadow in the region of the subacromial bursa just above the tip of the greater tuberosity and stated that this represented calcification within the region of the subacromial bursa. However, on exploration during surgery, he noted no bursal involvement. The calcium deposit was completely contained within the tendon of the rotator cuff.[21]

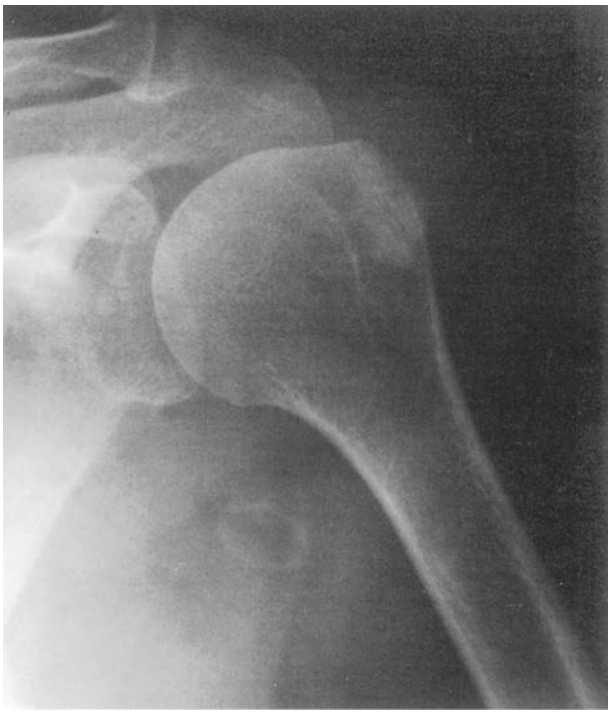

Figure 6-6 Calcifying tendinitis involving the infraspinatus or teres minor can be readily seen on an anteroposterior radiograph of the shoulder with the humerus held in internal rotation.

Bosworth believed that routine use of fluoroscopy was more accurate than routine roentgenographic examination, with concern that radiographs commonly missed calcifications that were superimposed over the humeral head or the acromion.[15] He claimed that "regardless of whether

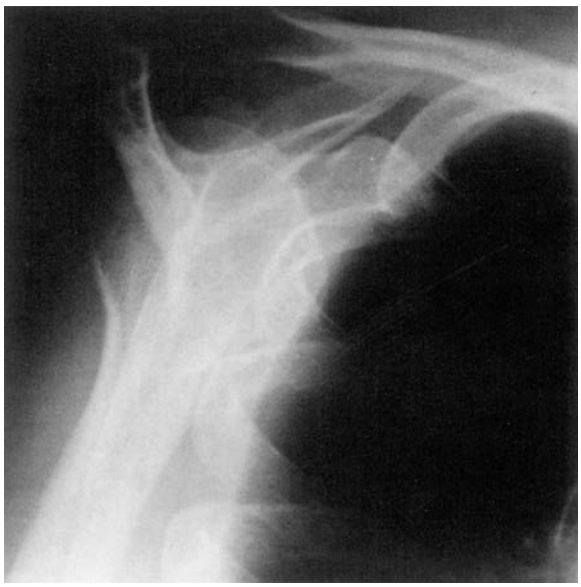

Figure 6-7 A supraspinatus outlet view confirms the involvement of the supraspinatus tendon. Localizing the calcific deposit is essential for successful needling or surgical decompression of the lesion.

or not roentgenograms are taken, fluoroscopy should never be omitted as an essential part of the exam." He also pointed out that many calcified deposits are "discovered accidentally," with nothing being done about it. Howorth also felt that fluoroscopy may be useful to localize the calcium deposition.[52] Fluoroscopy may help with visualization of the calcific deposit, but its value over routine radiographs in determining treatment is unknown. He felt that his technique was a more sensitive way to detect the calcifications than even surgical exploration.

Bosworth reported a classification scheme based on the size of the calcium deposit. Small deposits, up to 0.5 mm in diameter, were of little or no clinical significance; medium-sized deposits, 0.5 to 1.5 mm in diameter, were of moderate clinical significance; and the larger deposits, of more than 1.5 mm in diameter, were most likely to cause clinical symptoms.[15] Patte and Goutallier described a classification scheme that divided the radiographic appearance of calcifying tendinitis into a localized form and a diffuse form. The localized form was characterized by round or oval, homogeneously dense calcifications that were close to the bursal wall. These tended to heal spontaneously. The diffuse form was thought to be situated deeper than the localized form, close to the bony insertion of the tendons. The density was heterogeneous, and these were considered to cause more pain and to take longer to resolve.[102] Most authors feel that the classifications by both Bosworth and by Patte and Goutallier are not of much clinical value.

The French Society of Arthroscopy has classified rotator cuff calcifications into four categories (Fig. 6-8).[88] Type A is a homogeneous calcification, with well-defined limits, and represents approximately 20% of the patients presenting in a large multicenter review. Type B is heterogeneous calcification that is fragmented, but with well-defined limits and was seen in 45% of the patients. Type C calcification is a heterogeneous calcification, with poorly defined limits, sometimes with a punctate appearance; 30% of the patients present with this finding on radiographs. Type D calcification is a dystrophic pattern at the insertion of the rotator cuff. This pattern clearly represents a degenerative process and should not be confused with calcifying tendinitis (Fig. 6-9). With types A and B, the calcifications, at surgery, have a pasty consistency 50% of the time. Type C is diffuse, and a well-organized calcific deposit may not be present. This classification has relevance to the stage of calcifying tendinitis, the radiographic appearance, and implications for intervention.

Other authors have attempted to correlate the appearance of the calcified deposit on roentgenogram with the clinical phase of calcifying tendinitis. DePalma and Kruper described two radiologic types. Type I was amorphous, "fluffy" and "fleecy" in appearance, and heterogeneous, with a poorly defined periphery. This pattern was usually encountered in acute phases. Occasionally, they noted a contiguous overlying crescent-like streak that they believed represented rupture of the calcified deposit into the subacromial bursa. Type II had a well-defined outline and was homogeneous in density. This appearance was common in the subacute or chronic phase of calcifying tendinitis.[25] DeSeze and Welfling[26] and McKendry et al.[80] were also able to correlate clinical symptoms with the radiographic appearance of the calcium deposit. Friedman could not find a correlation between the size of the particles and the severity of symptoms.[33]

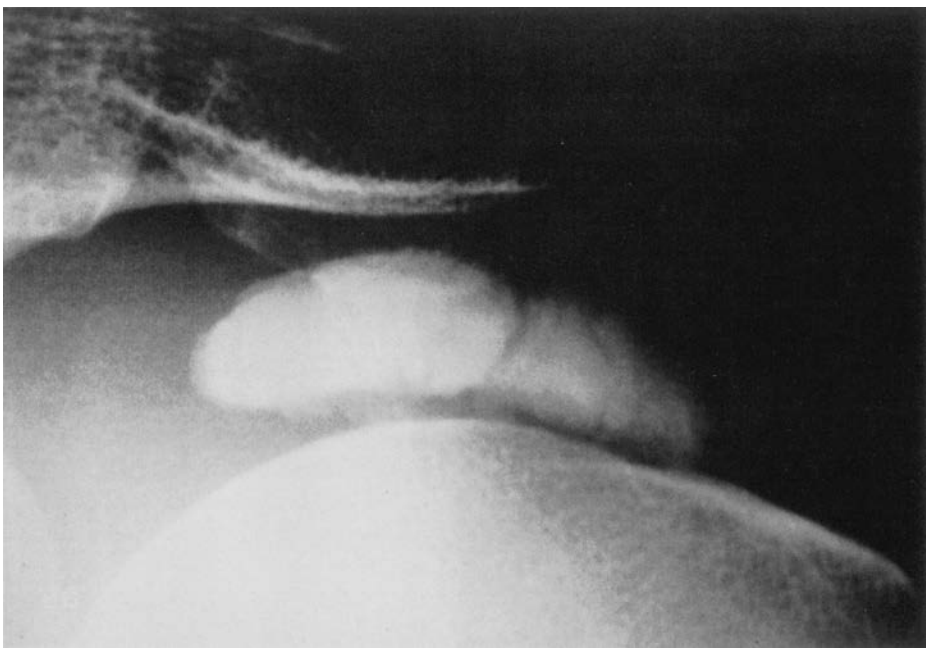

Figure 6-8 The French Society of Arthroscopy has classified rotator cuff calcifications into four categories: *type A:* homogeneous, well-defined limits; *type B:* heterogeneous, fragmented, well-defined limits; *type C:* heterogeneous, poorly defined limits; *type D:* dystrophic pattern. A type B calcification is seen in approximately 45% of the patients in their multicenter evaluation. Fifty percent of patients with this pattern will have a pasty consistency to the calcific material at the time of surgery. (Courtesy of Dr. Gilles Watch.)

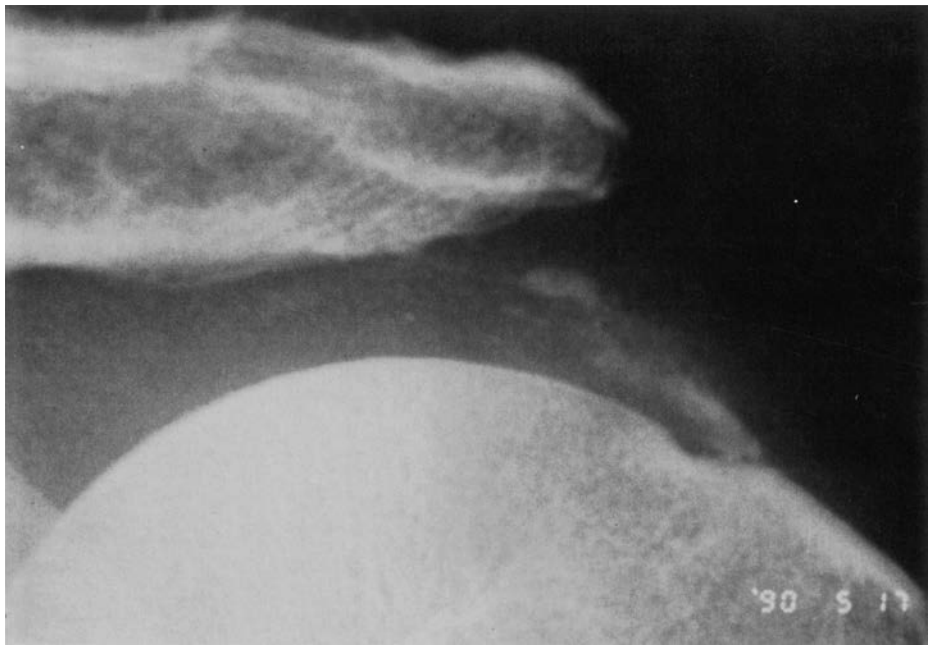

Figure 6-9 Type D calcification with a dystrophic pattern typical of degenerative process, but not calcifying tendinitis. (Courtesy of Dr. Gilles Walch.)

Pulich emphasized that tendinous calcifications differ from capsular or bursal calcifications because the calcific deposit of calcifying tendinitis is contained within the tendons of the rotator cuff and remains confined to the points of insertion of the tendon. Bursal or capsular calcifications eventually conform to the cavity within which they are contained.[111]

Uhthoff et al. do not believe that all cases end with rupture of the calcified deposit into the subacromial bursa.[138] When this did not occur in their patients, symptoms tended to be more prolonged and recurring. Furthermore, they demonstrated a strong correlation between the clinical symptoms, radiographic appearance, and intraoperative findings in 41 patients undergoing surgery for calcifying tendinitis.[80] Of the 41 patients, 31 had chronic or subacute symptoms, and 10 had acute symptoms. Twenty-four of 31 patients with chronic symptoms had roentgenograms consistent with the formative phase of calcifying tendinitis. Intraoperatively, the calcified deposit appeared chalk-like in 29 of the 31 patients. Of the 10 patients with acute symptoms, eight had x-ray films consistent with the resorptive phase; nine patients also had a toothpaste-like consistency of the calcified deposit.

Other radiographic concerns include coexisting conditions. Some authors have raised the question of concomitant presence of a rotator cuff tear with calcifying tendinitis. Although McLaughlin and Asherman stated that the presence of calcifying tendinitis is strong evidence against a rotator cuff tear,[83] other authors, including Hsu and his colleagues, believe that one should commonly consider the possibility of coexisting calcifying tendinitis and rotator cuff tear.[53] However, their study contrasts with many of the tenets concerning calcifying tendinitis that are confirmed in previous studies, and suggests that the report by Hsu et al. is discussing a subset of calcifying tendinitis, a completely different condition, or a variation of calcifying tendinitis unique to his Taiwanese patient population. Also, many patients were older than the age of 60, at which an increased incidence of rotator cuff tears would be expected in a control population without calcifying tendinitis. However, others have suggested an increased incidence of rotator cuff tears with calcifying tendinitis. Kernwein observed that, in patients older than the age of 40 years with calcifying tendinitis, there was a 90% probability that an arthrogram would reveal rotator cuff tear.[61] This report has not been supported by others.[39] Wolfgang observed that 23% of the patients that had a rotator cuff tendon repair also had calcifying tendinitis.[148]

Jim et al. looked at the coexistence of calcifying tendinitis and rotator cuff tendon tears prospectively in their Taiwanese patient population. In 81 patients with calcifying tendinitis, an arthrogram was performed to look for concomitant rotator cuff tear. Twenty-two arthrograms were positive. Small amounts of calcifications were statistically more likely to be associated with rotator cuff tear in this population. They concluded that calcifying tendinitis with rotator cuff tendon tear is not uncommon, especially in older patients.[56]

Confounding this issue, Loew et al. correlated the presence of calcifying tendinitis with subacromial impingement findings. They looked at 75 patients with calcifying tendinitis, and prospectively performed roentgenographic studies as well as magnetic resonance imaging (MRI) of these patients to identify additional findings of subacromial

impingement. They found degenerative tendon changes in 11% of the 75 patients, and a type III acromion in 16% of these patients. Their conclusion was that there was little correlation between calcifying tendinitis and additional findings of subacromial impingement.[70]

The role of computed tomography (CT) and MRI in calcifying tendinitis is still unclear. CT may help localize the calcific deposit, but is unlikely to add additional information that will influence treatment. On MRI, the T1-weighted images show calcifications as decreased signal intensity, whereas T2-weighted images may show perifocal increased uptake around the deposit, largely from edema during the resorptive phase.[51,54] A cautious interpretation of the MRI findings is important because the decreased signal intensity, combined with the relatively thin dimensions of the cuff lateral to the calcific deposit, may be interpreted as a large rotator cuff tear. This false MRI interpretation, when combined with a limited physical examination secondary to pain, may lead to a more aggressive surgical approach than is indicated by the true pathology. Re and Karzel stated that routine use of CT and MRI are probably unnecessary if adequate roentgenograms are obtained.[113] However, if evaluation of coexisting pathology is the intent, MRI is the most effective specialized radiographic study.[51,54] An arthrogram will also provide additional information on the integrity of the rotator cuff.

Maier prospectively studied 65 shoulders with chronic calcific tendinitis treated with extracorporeal shock wave therapy, and was able to correlate preoperative gadolinium-enhanced MRI findings to outcomes. Patients underwent preoperative and postoperative radiographic analysis, as well as Constant scoring. Preoperative MRI findings included the presence or absence of contrast enhancement of the synovia, the calcific deposit, and the subacromial bursa. Among the 16 patients that lacked contrast enhancement around the deposit, 15 patients had satisfactory outcome, compared to 44 patients with positive contrast enhancement, with 57% satisfactory outcome. Similar outcomes were observed among those shoulders that lacked contrast enhancement in both the synovia as well as the bursa. However, there was no significant correlation between size or morphology of the calcific deposit. The authors concluded that the absence of contrast enhancement, especially around the deposit, is a strong predictive parameter of a positive clinical outcome.[73]

Ultrasound has also been used for evaluation of calcific deposits. Hartig and Huth demonstrated that ultrasonography was more sensitive than plain radiographs for detecting calcific deposits in the rotator cuff tendons.[50] In 217 cases of calcifying tendinitis, ultrasound detected 100% of the lesions, while plain radiographs demonstrated 90%. Ultrasound has the added benefit of not exposing the patient to unnecessary radiation. Unfortunately, ultrasonography is highly dependent on technicians and radiologists with significant experience in evaluating shoulder conditions.

Natural History

Calcifying tendinitis proceeds through a cycle of calcification, resorption, and then tendon reconstitution in most cases (see Fig. 6-3). The self-healing nature of calcifying tendinitis has been emphasized. Carnett pointed out the "tendency for the hyperacute pain to ease up after a few days or within two weeks." He also stated that "in many chronic cases, the condition ran a self-limiting course and irrespective of treatment, the patient made complete recovery at varying periods from a few months up to 3 years."[19] Unfortunately, not all cases of calcifying tendinitis resolve expediently and without sequelae.

Codman questioned the ability of the involved rotator cuff tendon to completely heal and reconstitute its normal integrity. He stated that "although it is the rule that cases of calcified deposits recover with no known sequelae, I am more and more inclined to think that they must result in some atrophy of the tendon, whether they are absorbed naturally or are removed."[21]

Howorth noted that the calcareous deposits often change in size and density over a period of months or years, but may in fact change over a few days. He also noted that calcium deposits within the tendons commonly persist for many years unless there is spontaneous or surgical drainage. Rupture of the deposit into the subacromial bursa not only results in a change in the location and density of the calcified material, but also usually is followed by immediate relief of symptoms and absorption of the calcified material.[52]

McLaughlin, in 1946, reported that 90 patients who had calcified deposits discovered incidentally by roentgenograms returned at a later date with pain, presumably owing to irritation by the calcified deposit. Acutely painful lesions frequently had a portion of the calcified deposit in contact with the bursal floor and occasionally within the subacromial bursa itself. He suggested that if the lesion was left untreated it could produce constant mild pain for as long as 15 years. However, these calcified deposits were rarely found in unselected cadavers older than 66 years, which indicated that "most if not all deposits are limited by some spontaneous mechanism prior to the sixth decade."[81]

Lippmann observed that the phase of interval symptoms abides by no time schedule, and has been observed to persist for as long as 12 years. He noted that a close inverse relation between the severity of pain and its duration exists, but rarely does the painful absorption of calcium persist for longer than 3 weeks.[67] Moseley outlined a detailed natural history of calcified deposits in the rotator cuff. He described an initial "silent phase," which was characterized by minimal clinical signs and symptoms. The "mechanical phase" was characterized by impingement-type symptoms. These patients tended to have a painful arc of motion. The "subbursal rupture" phase is a result of partial evacuation of the calcified deposit. As such, symptoms

in this phase of the disease tend to recur. The final phase, which he called "intrabursal rupture," is characterized by complete drainage of the calcified deposit under the floor of the bursa and secondarily into the bursa. Clinical symptoms were most severe with this phase, corresponding to resorption of the calcific deposit. He believed that the calcified deposit had to be completely eliminated before the patient was free of symptoms.[91]

Lapidus and Guidotti reported on "a number of cases of spontaneous recovery in the patients who came under observation days following the onset of the acute symptoms." They also noted complete disappearance of the deposit radiographically in those same patients.[65] Ghormley observed that although the tendency for complete resolution was the rule, some cases would go on to "heterotopic ossification."[38] This concept has not been supported by others.

Uhthoff and Sarkar have conclusively demonstrated that calcifying tendinitis is an active, cell-mediated process that tends to progress toward spontaneous resolution, including tendon reconstitution. The roentgenographic appearance, the clinical symptomatology, and the histologic and gross appearance can be reasonably predicted by knowing any one of the aforementioned features and by understanding the cyclic nature of this disorder.

Other authors have suggested that because the success of intervention is high and rapidly resolves the clinical symptoms, patients should not have to wait for the expected resolution of their condition. Codman felt that if he personally developed calcifying tendinitis, he would rather have surgical treatment than wait for the condition to resolve on its own.

Harbin also suggested that "conservative treatment in the acute cases seems to be unwise." He recommended that "since the operative treatment is so simple, offers so little danger, and promises complete relief in practically 90% of the cases, one should have little hesitancy in urging it in all cases in which the condition has persisted over a period of months without relief by the more conservative measures."[48]

Harmon reported that the natural course of calcifying tendinitis could be favorably influenced by treatment properly applied relative to the time in the cycle of the disease. He too recommended removal of large to medium-sized deposits within the rotator cuff tendons. This was based on his observations that few quiescent or mildly symptomatic deposits disappear spontaneously. However, in the acute and hyperacute deposits, these tended to disappear in 7 to 14 days with conservative treatment.[49] DePalma and Kruper believed that the natural history of calcifying tendinitis could usually be favorably altered by the treatment chosen. They reported on the results of a long-term follow-up study of shoulder joints treated for calcifying tendinitis, surgically as well as conservatively. They observed that 84% of the patients treated conservatively had favorable short-term results. However, long-term results revealed that only 61% were favorable. In contrast, surgical management produced 96% favorable results. They noted that the convalescence period was longer when the calcifying tendinitis was treated surgically.[25]

Most recently, Kempf et al. noted that although the natural evolution of this calcifying tendinitis is favorable, the cyclic natural history described by Uhthoff may in fact be blocked at any stage of the disease.[60] Even though many studies have demonstrated that conservative treatment of these deposits can be successful in most of these patients, a study by Noel and Brantus encourages a cautious posture for prognosis because only 50% of their 124 cases treated without surgery demonstrated favorable results.[97]

TREATMENT

The treatment of calcifying tendinitis has tended to vary according to the expertise and experience of the treating physician. There have been a variety of treatment methods proposed, all of which have had some success in the treatment of calcifying tendinitis. An understanding of the pathophysiology of calcifying tendinitis is essential when determining the correct intervention. Uhthoff and Loehr's explanation of the three pathophysiologic stages of calcifying tendinitis created a framework in which to establish an appropriate treatment algorithm.[142]

Determining the correct stage of calcifying tendinitis includes a careful history, physical examination, and appropriate plain radiographs. Acute calcifying tendinitis is severely painful, with patients splinting their upper extremity against any movement. Narcotic medications may be required to control pain and allow sleep at night. Patients often demand intervention because of the severity of their symptoms and the effect on their daily activities, even if they understand that their condition may actually be resolving. Because acute symptoms are associated with the resorption phase, which is frequently measured in days or weeks, treatment at this stage is likely to be associated with a favorable result, even though the treatment may have had no direct effect on the natural history of the patient's condition. With chronic symptoms secondary to calcifying tendinitis, intermittent worsening of symptoms may lead to patient dissatisfaction. The goal of intervention should be to establish a treatment plan that can accelerate the natural history of this condition or encourage the resolution of symptoms without significant risk to the patient.

Nonoperative Treatment

Virtually all patients with calcifying tendinitis are successfully treated with nonoperative management. Gschwend and colleagues estimated that more than 90% of patients are adequately treated with conservative management.[45]

Litchman et al. reported that they were able to treat 99 of 100 patients with calcifying tendinitis without surgical intervention.[68] Treatment options include analgesics, nonsteroidal antiinflammatory medications, physical therapy, infiltrations, needling or barbotage with or without aspiration, extracorporeal shock wave treatment, and antiinflammatory radiotherapy.[6,16,41,45,49,69,74,94,103,105,106,118]

On initial presentation, patient complaints are most commonly related to pain. Nonsteroidal antiinflammatory medications are often prescribed. Their efficacy for the treatment of chronic or acute calcifying tendinitis and their effect on the natural history is unknown. During the acute hyperalgesia of the resorption phase, narcotic medication may be indicated for the relief of severe pain that interferes with sleep and daily activities.

Many authors recommend physical therapy for the treatment of patients with calcifying tendinitis, especially those patients presenting with mild pain and chronic symptoms.[14,32,38,45,67,81,92] The primary focus of physiotherapy is to reestablish normal shoulder motion. Some authors have suggested that therapy is necessary to prevent an idiopathic frozen shoulder. However, there is no evidence to confirm that calcifying tendinitis leads to a generalized contracture of the glenohumeral joint capsule, which is the hallmark of a frozen shoulder.[77] Restriction in motion is more likely to be secondary to pain, muscular spasm, and an increased volume of the rotator cuff within the confines of the coracoacromial space. Because the coracoacromial arch is congruent with the normal rotator cuff tendon and proximal humerus, a change in the volume of the rotator cuff tendon from a calcific deposit or the combined effects of the deposit and factors involved in the resorptive phase are likely to lead to mechanical irritation. This mechanical irritation will lead to decreased volitional movement of the shoulder to avoid discomfort. The primary focus of therapy is mobilization, avoiding the stiffness that accompanies inactivity and pain so that once the calcifying tendinitis has improved, there is no long-term restriction on shoulder motion. Exercises initially include pendulum exercise and gentle passive range of motion. As the symptoms improve, stretching of the shoulder can be accomplished with passive to active motion, including forward elevation, external rotation, internal rotation, and horizontal adduction.

Physical modalities, such as ultrasonography, infrared heat, and iontophoresis, have been used to treat calcifying tendinitis.[44] However, there is no evidence that any of these physical modalities have a beneficial effect on the resolution of the calcifying tendinitis, either in the acute or chronic clinical phase. Griffin and Karselis have observed that ultrasonography was unable to mobilize the calcium crystals or to stimulate the resorption of the calcium deposit.[44] However, ultrasonography did provide short-term pain relief.

Needling or Puncture Aspiration

Some form of needling of the calcific deposit has been commonly recommended for the treatment of calcifying tendinitis. The methods of needling or puncture aspiration have been varied and include the placement of a needle within the calcific deposit to decompress the pressure at the site of the calcifying tendinitis; an injection of a local anesthetic alone; an injection of a local anesthetic with needling of the deposit, followed by a cortisone injection; an injection of cortisone alone in the subacromial space; and a needle aspiration-irrigation to decompress and remove the calcific deposit. There are some data to support each of these techniques. Much of the confusion arises from the treatment of calcifying tendinitis during the resorption stage. Because the acute symptoms are commonly associated with an expedient resolution of the condition, any treatment at this time is likely to be "successful." In fact, the treatment goal during the acute hyperalgesic clinical phase, corresponding to the stage of resorption, should be primarily directed toward improving patient comfort, with a secondary goal of avoiding any intervention that may inhibit the tendon restoration. Because each method may be effective, the single best treatment must also take into account the cost to the patient both in terms of potential risks as well as the costs related to the expense of the procedure.

Several authors have recommended needling or barbotage of the calcific deposit (Fig. 6-10). In Codman's treatise on rotator cuff disorders,[21] he refers to Flint's 1913 publication describing aspiration of the calcific deposit. After reading Flint's publication, Codman tried the aspiration and felt at the time of the aspiration that he actually punctured the deposit, which allowed the contents to leak into the bursa and relieve the patient's pain. He stated this was not Flint's exact plan, but was his achievement nonetheless. Barbotage can be accomplished either with a blind approach, as described by DePalma and Kruper,[25] or may be done with a more sophisticated measure of localizing the lesion using either fluoroscopic control or ultrasound.

Patterson and Darrach recommended needling of the calcification in conjunction with an injection of local anesthetic in the subacromial space.[103] Friedman demonstrated the ability to achieve pain relief in 85% of patients treated with this method.[33] However, in 11 out of 70 shoulders, the method failed to relieve the acute pain. Furthermore, Friedman noted that the amount of actual calcium removed did not appear to be important to the outcome of the needling of the calcified deposit. He concluded that piercing the calcium deposit was essential, whether or not the calcium was removed by aspiration or irrigation.

In DePalma and Kruper's report, a 1% solution of a local anesthetic was used to anesthetize the area of the subacromial space.[25] After needling the deposit and attempting to remove as much of the calcific mass as possible,

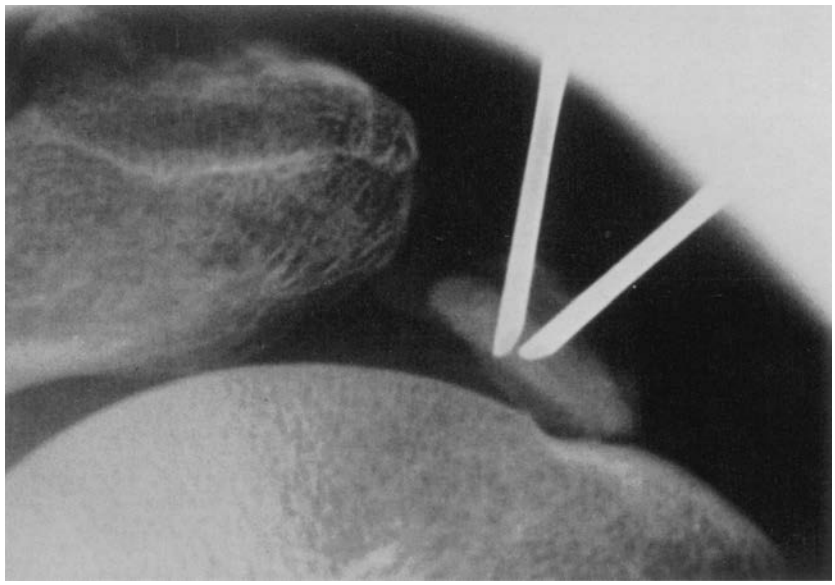

Figure 6-10 Needling of the calcific deposit decompresses the lesion, decreases the pressure in the tendon, and subsequently alleviates the symptoms of calcifying tendinitis. Needling can be performed "blind" in the office, or by using specialized techniques, such as fluoroscopy, to accurately localize the deposit. A second needle can be inserted into the area of the deposit for additional decompression or lavage. (Courtesy of Dr. Gilles Walch.)

multiple punctures were then placed with the 18-gauge needle into the rotator cuff tendon. It was suggested that this would cause an active hyperemia, enhancing dissolution and absorption of remaining calcific material. After completion of the needling, 10 mL of hydrocortisone was injected at the site of needling. One hundred fifty-four shoulders were evaluated for calcifying tendinitis, 86 of these treated by conservative methods. Ninety-four of the shoulders were reevaluated at follow-up, with 41 of the shoulders being treated by multiple puncture of the deposit. Only 23 of the 41 shoulders treated conservatively were reexamined, with 13 having a good result, five having a fair result, and five having a poor result. In other patients contacted by questionnaire, 18 shoulders in total, 12 were rated as good, four fair, and two poor. It was their opinion that conservative management provides satisfactory short-term results in most cases.

Key recommended needling in acute cases when a single large deposit is seen on radiographs.[63] After needling and aspirating the deposit, multiple punctures were performed. He suggested needling under local anesthesia for patients who would best be treated by operative removal but did not want to undergo an operation. Key felt that it was not a reliable form of treatment, and he recommended surgery for those patients who did not respond to physical therapy and physical modalities.

Lapidus presented 16 cases of patients presenting with acute symptoms.[64] He found that treatment by infiltration with a local anesthetic and injection of saline into the calcified deposit caused relief of symptoms and disappearance of calcification soon afterward and, therefore, injection therapy was beneficial. It was clear that his treatment occurred at the time of resorption and it was likely that resolution of the calcium would occur without the injection. Moutounet and his colleagues discussed the radiologic localization of the calcific deposit with subsequent needling.[94] After needling the deposit, corticosteroid was injected into the area of the deposit. They concluded that direct puncture of the calcification shortens the natural history of both acute and chronic calcifying tendinitis and accelerates resorption in 50% of the cases. With the addition of a small amount of cortisone, pain was relieved, and 80% good and very good results were achieved.

Clement also described a method of needling of the calcific deposit.[20] After spraying the skin with ethyl chloride, a no. 22 needle containing lidocaine and methylprednisolone was used to inject the solution into the subacromial space. Once the local anesthetic had become effective, the calcific deposit was needled repeatedly. Clement recommended 15 to 20 times. This was done without any type of radiologic localization of the calcific deposit. Clement mentioned that the acute pain subsided within 24 hours. Within a few days, the patients were referred to the therapist for ultrasonic treatment. He concluded that the pain was relieved with needling and injection and that the absorption of calcium was improved with ultrasonic treatments. There was no control group to evaluate whether the ultrasonic treatment had any effect in addition to the needling. In his series, there were no recurrences.

Farin et al. described a more sophisticated technique using ultrasonic-guided needling and aspiration, as well as lavage of the calcific deposits.[31] The technique of ultrasonographic localization was described, and two case reports were provided. The goal was direct needling of the deposit, followed by aspiration and lavage to remove as much of the calcific material as possible (Fig. 6-11). Within

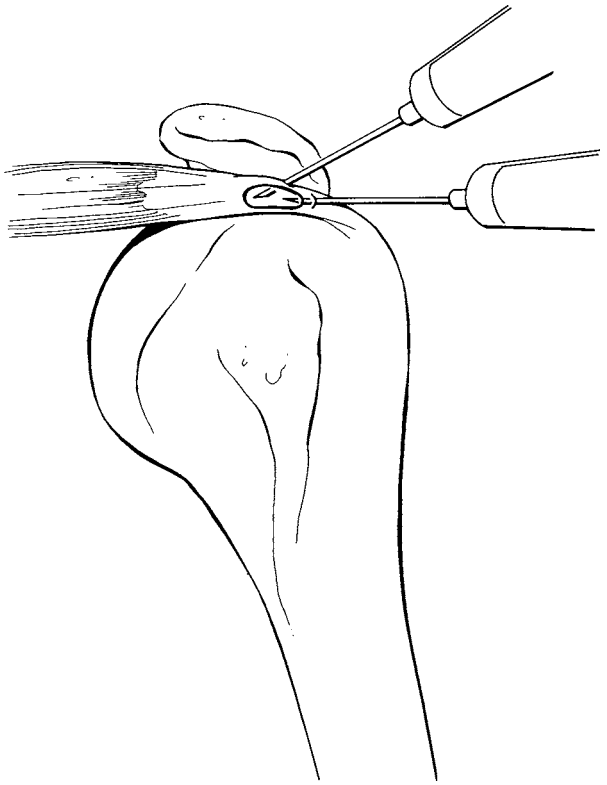

Figure 6-11 Needling and lavage can be effective if the deposit is localized. Sophisticated imaging techniques, such as fluoroscopy or ultrasound, permit accurate localization and direct penetration of the calcific deposit.

a few days of the treatment, both patients had a substantial reduction in their discomfort and were started on physical therapy programs. Bradley et al. reported 11 cases with radiographically evident calcific deposits. By using an ultrasound guidance technique, the deposit was needled and aspirated. Six of 11 patients had "relief" on the same day, four additional patients were pain-free after 2 weeks, and the remaining patient, who had no change in symptoms, had a supraspinatus tear seen on the ultrasonographic examination.[16]

Although needling of the deposit with adjunctive use of a local anesthetic is widely accepted, the addition of corticosteroid to the local treatment has been controversial. Harmon, discussing the treatment for calcific rotator cuff tendinitis in 609 shoulders, performed 263 needlings after local injection of hydrocortisone Meticorten anesthetic drugs.[49] A total quantity of 20 to 40 mL suspension with one-half 1% lidocaine and one-half hydrocortisone, 25 mg (or prednisolone 25 or 50 mg/10 mL) was used. This was blindly injected into multiple sites just proximal to the greater tuberosity, which was determined by the patient examination and radiographs. The results were considered excellent by this method alone, with the disappearance in 14 days or less after the last injection. However, approximately 54% of this group required two or more injections to alleviate the symptoms. Furthermore, there was no difference in those shoulders that were injected and needled with lidocaine alone or with the lidocaine and corticosteroid suspension. Subjectively, there did appear to be decreased muscle spasm and periarticular stiffening with the corticosteroid suspension.

Friedman noted that patients who had corticosteroid injected at the time of the calcium needling appeared to have less pain after the anesthetic effect of procaine (Novocaine) had worn off.[33] However, he emphasized that the decompression of the calcific deposit was far more important than the local effect of hydrocortisone. Those deposits that had been definitely pierced or aspirated had results that were similar whether or not corticosteroid had been used. It did not appear that corticosteroids had any negative effect, nor did they appear to have any effect on the reduction in the size or the disappearance of the calcium deposit.

Lapidus recommended anesthetizing the shoulder with a local anesthetic followed by multiple punctures through the calcified deposit.[64] At the completion of the multiple puncture, an injection of corticosteroid is performed. In his series of 248 cases, 83 patients received a corticosteroid injection. It is unclear as to what criteria were used to decide on which patient received a corticosteroid injection. Of the 83 patients who did receive the injection, 19 (23%) actually had aspiration of the calcific deposit. Lapidus stated that "practically all" of the patients were promptly relieved, and it was unclear whether the cortisone affected the final result. Murnaghan and McIntosh treated 27 patients with local anesthetic and 24 patients with corticosteroid and was unable to demonstrate a difference in the results.[95]

Gschwend found that corticosteroid injections were short acting, and their effect was on symptoms alone.[45] Moseley felt that steroids injected into tense deposits may cause extensive necrosis and, therefore, recommended that if steroids and a local anesthetic are to be injected, it must be only into the subacromial space and bursa.[92]

Neer believed that cortisone injections do have a role in the treatment of patients with calcifying tendinitis.[96] When patients had pain and stiffness that could not be effectively treated with physiotherapy, he recommended a limited number of local steroid injections to reduce inflammation and resume exercises. When the resorptive phase had begun and pain was severe, he recommended an injection of lidocaine followed by needling, and then completed with an injection of corticosteroids. In those deposits that had occurred during the formative stage, again, injections, including cortisone injections, were recommended. Neer noted that when he was a resident, the most frequent shoulder operation in his hospital was excision of calcium. However, he believed that treatment using parenteral nonsteroidal antiinflammatory medications and corticosteroids reduced the surgical excision of calcium to a rare procedure.

Other authors have expressed concern over the injection of corticosteroid and the disruption of the normal tendon-

healing process, or tendon reconstitution. Lippman believed that corticosteroids could inhibit the resorptive phase and possibly return the calcifying tendinitis back to its static or resting phase.[67] Uhthoff and Sarkar have also stated that there is no indication for an injection of cortisone when treating the acute clinical or resorptive phase of calcifying tendinitis.[141]

In conclusion, an injection of corticosteroids is primarily effective as a long-acting pain reliever, possibly because of the resolution of the associated inflammatory process of the resorptive phase. Cortisone does not appear to have any benefit on the resolution of the calcific deposit. However, despite theoretical concerns, there is no evidence that an injection of corticosteroids into the subacromial space following needling of the calcific deposit has any adverse effects.

Radiotherapy

Radiation or x-ray therapy has been advocated for the treatment of calcifying tendinitis of the shoulder.[6,100,105,108] Treatments have varied from a single large dose of radiation to small doses on a repetitive basis. Improvement in symptoms has been reported in 70% to 100% of patients. Radiotherapy was initially proposed in Europe and later used in the United States. Plenk evaluated the effect of radiation therapy in 21 patients, with an additional 17 patients as a control group.[108] In the control group, the patients were set up exactly as the study group, only a lead shutter was placed in front of the x-ray tube housing to prevent any radiation of the shoulder. Remarkably, a definite improvement or complete relief of pain within 6 weeks after therapy was discovered in 15 of the treated patients and 15 of the control patients. It was Plenk's opinion that "calcifying tendinitis of the shoulder is essentially a self-limiting disease." He argued that there was no benefit from radiation therapy, and stressed the importance of understanding the natural history of calcifying tendinitis.

Recently, Ollagnier and coworkers reported on a group of 44 patients receiving what they termed "antiinflammatory radiotherapy."[100] All patients were treated with a dose of radiotherapy ranging from 600 to 1,200 R, divided between five and nine sessions. With a mean follow-up of longer than 2 years, 68% of the patients were satisfied or very satisfied with the treatment. However, when the treatment was broken down into the various stages of calcifying tendinitis, it was clear that patients presenting with the formative stage of calcifying tendinitis had equivocal results, with 6 out of the 14 patients not a part of the satisfied group. In those patients in the resorption phase or demonstrating heterogeneous pattern to the calcific deposit, six out of the eight were in the satisfied group. The author speculated that antiinflammatory radiation therapy was effective and that it is "at least as efficient as needle aspiration or lithotripsy."

Baird reported on 18 cases of calcifying tendinitis:[6] Nine patients were in the acute phase, five patients in the subacute phase, and four patients in the chronic phase. He suggested that only one exposure was required to relieve the pain completely and restore normal function for patients in the acute phase and some patients in the subacute phase. No serious adverse effects were noted. Given the cost, potential risks to the patient, and lack of evidence demonstrating its effectiveness, it would appear that radiation therapy for calcifying tendinitis is primarily of historical importance.

Extracorporeal Shock Wave Treatment

Dahmen initially reported on the efficacy of extracorporeal shock wave therapy using low-pulse energy.[24] There was no investigation into the changes of the calcific deposit after the shock wave application, but clinically there was a significant improvement in shoulder pain.

Loew studied 20 patients who were treated with a lithotripter in two sessions of 2,000 pulses each.[69] Twelve weeks following the treatment, 15 of the 20 patients had a marked reduction of symptoms, with a 30% improvement overall. Fourteen patients had a transient subcutaneous hematoma. MRI failed to demonstrate any damage to the tendon or the bone. These patients demonstrated chronic calcifying tendinitis, with a history of symptoms for longer than 12 months and radiologically proven calcification of the rotator cuff measuring greater than 10 mm in the anteroposterior roentgenogram, consistent with the formative stage. The calcific deposits were of homogeneous consistency and would correspond to the type A classification by the French Society of Arthroscopy. No patients had rotator cuff tears. Radiographic studies 6 weeks following the extracorporeal shock wave therapy demonstrated that 11 of the 20 patients had a change in their radiographic architecture. At 12 weeks, 12 patients demonstrated radiographic changes, with seven showing a complete resolution of the calcium deposit.

Rompe et al. evaluated the use of extracorporeal shock wave therapy in the treatment of 46 patients with calcific deposits of the supraspinatus tendon.[118] Inclusion criteria included persistent shoulder pain, in combination with the calcific deposit of the supraspinatus that had been present for more than 12 months and had failed conservative therapy for at least 6 months. The deposits were sharply outlined and densely structured, with a minimum diameter of 10 mm. This technique uses an electromagnetic shock wave generator in a mobile fluoroscopy unit. A regional anesthetic agent was necessary to complete the procedure. At 6 weeks, partial resorption of the calcium deposit was noticed in 43% of the patients, with complete resorption in 10%. By 24 weeks there was a 48% partial resorption and a 15% complete disintegration. Again, based on the results, Rompe noted that extracorporeal

shock wave therapy was more effective than injections or needle irrigation for the treatment of calcifying tendinitis in the formative stage.

Loew reported results of shock wave therapy in 195 patients with chronic calcifying tendonitis, including subjective, functional, and radiologic findings at 6 months after treatment. The treatment groups were split into two groups, the first of which was divided into low-energy or high-energy shock waves. The second group was treated with either one or two high-energy sessions. Pain relief correlated with the energy level applied to the calcific deposit, with 5% in the control group and 58% in the two high-energy session group. Constant scores increased only 3 points on average in the control group at 3 months, but increased 15 points on average in the two high-energy group. In addition, there was radiologic disappearance of disintegration of the calcific deposits in only two patients in the control group, and 12 patients in the two high-energy session group. The authors concluded that improvements seen with the use of shock wave therapy is dose-dependent. It should be noted that only 79% of the patients in the high-energy group were available for follow-up, with 11 patients electing other treatment (injection or surgery) and 13 refusing further examination.[71]

Haake et al. compared the results of extracorporeal shock wave therapy directed at the origin of the supraspinatus tendon versus the calcified area in 50 patients, and found that at 12 months, the group that had exact focusing at the calcific deposit had zero failures, while the group without exact focusing of the shock wave therapy had 14 failures. When the radiographic appearances of the calcific deposits were compared, there was no statistical difference between the two groups. There were no significant side effects in either group. The authors concluded that exact fluoroscopic focusing of extracorporeal shock wave therapy at the calcific deposit is recommended.[46]

Wang reported results of shock wave therapy in 31 shoulders treated with 1,000 impulses of shock waves at 14 kV. At 12 weeks, 21 shoulders were available for follow-up, with 62% showing significant improvement or having no complaints. Radiographic evaluation showed complete elimination of the calcific deposit in only six patients, and partial dissolution in another five patients. They were able to show a correlation between functional improvement and elimination of the calcific deposits.[144]

Rompe et al. compared the results of conventional surgery to shock wave therapy in 79 patients. Twenty-nine patients had surgical excision of the calcific deposit, and 50 patients were treated with high-energy shock wave therapy (3m000 impulses at 0.6 mJ/mm^2). The authors concluded that for patients with homogenous deposits, surgery was superior to shock wave therapy. For patients with inhomogenous deposits, high-energy extracorporeal shock wave therapy was equivalent to surgery, and should be given priority because of its noninvasiveness.[119]

Daecke et al. studied the long-term effects of extracorporeal shock wave therapy in 115 patients, 56 of whom received one session of shock wave therapy (group A), and 59 of whom received two sessions (group B). The authors noted that improvements in pain relief, Constant score, and radiographic improvement were energy-dependent. Additionally, there were significant differences between the two groups. At 4 years, 20% of the patients had undergone surgery on the affected shoulder. The authors concluded that while the failure rate was high, extracorporeal shock wave therapy was successful for 70% of the patients in this study, and no long-term complications were seen.[23]

Wang reported a 2-year follow-up study on 39 shoulders treated with shock wave therapy. The results showed that 91% of the patients obtained complete or nearly complete resolution of their symptoms. In addition, 57% of the shoulders showed complete dissolution of calcium deposits, with no recurrence at 2 years follow-up. It should be noted that 2 years postoperatively, six shoulders were eliminated from the study because of poor compliance or inadequate data. Additionally, there was no mention of how they were doing at their latest follow-up.[145]

Durst et al. reported one case of osteonecrosis of the humeral head 3 years and 4 months after undergoing extracorporeal shock wave therapy for chronic calcific tendonitis in a female patient. She had received three sessions of shock wave therapy, with 1,600 to 1,700 impulses at 12 to 13 kV over a period of 1 month. There were noted improvements in pain, as well as a reduction in the size of the calcific deposit, following this treatment. Unfortunately, the patient presented with stage IV osteonecrosis of the humeral head. The patient had no other predisposing factors for developing osteonecrosis. The authors have postulated that this may have been caused by injury to the anterior humeral circumflex artery. The arcuate artery was only 10 mm from the site of the calcific deposit, and the patient's MRI showed osteonecrosis in the portion of the humeral head that was supplied by the anterior humeral circumflex artery.[28]

In the past decade, there has been a growing enthusiasm for using extracorporeal shock wave therapy for the treatment of chronic calcific tendonitis. The majority of studies seem to indicate that while there seems to be an improvement in patients' symptoms short term, the best long-term results correlate with radiographic disappearance of the calcific deposit. Additionally, dissolution of the calcific deposit occurs more reliably when higher amounts of energy are directed at the deposit. While the complication rate of shock wave therapy seems low, and is most commonly an intramuscular hematoma, there have been reports of transient bone marrow edema and one case of osteonecrosis of the humeral head.[28] At this time, the results of shock wave therapy remain inferior to surgical débridement.

Surgical Treatment

Codman wrote: "My personal opinion is that surgical removal of the deposit is practically free from danger, sure to relieve the severe symptoms at once, and, in fact, that it generally will relieve all really troublesome symptoms within a few weeks."[21] He further opined that "it would be rational to thrust a large aspirating needle into the deposit under guidance of the fluoroscope, but I would prefer to be operated upon were I the patient." He suggested that the operation could be performed with a local anesthetic despite an open approach to the supraspinatus tendon. Bosworth concurred, stating that the most dependable way of eliminating the symptoms and the disease of calcifying tendinitis is by open surgery.[14]

Lippmann reviewed his results of 100 consecutive cases treated with surgical decompression of the calcific deposit.[67] His conclusion was that patients with calcifying tendinitis treated surgically can expect return of normal function to the shoulder. McLaughlin discussed the operative decompression and removal in over 200 cases.[82] Surgical treatment was believed to be necessary in less than 10% of all cases of calcifying tendinitis. In most of the surgical cases, he believed that the severity of the patients' symptoms and their request for treatment of their pain led to surgical management. He concluded that surgical treatment produced the most certain and permanent results.

Gschwend et al. recommended surgical treatment when there was a progression of symptoms, interference with activities of daily living, and a failure of conservative management.[45] Such treatment was correlated with a good or an excellent result in more than 90% of patients. Neer stated that surgical indications should include the failure of conservative treatment, as well as multiple hard, gritty deposits with long-standing symptoms.[96] Despite a practice dedicated entirely to the treatment of shoulder problems, he was performing the procedure only once or twice a year. He also was keenly aware that the recovery period for long-standing lesions is longer than one might expect from relatively minimal open surgery and removal of the calcifying deposit, suggesting residual tendinopathy despite removal of the calcific lesion.

McKendry et al. reported on the surgical treatment of 57 patients with calcifying tendinitis.[80] Surgery was performed after an average follow-up of 2.8 years after the onset of symptoms. Sixty percent of patients were pain-free postoperatively at 6 weeks, although 30% had continuing pain beyond 12 weeks. Preoperative factors predicting a longer convalescence could not be identified. They emphasized that a fluffy appearance to the calcium deposit is associated with the resorptive phase; therefore, a trial of conservative treatment was recommended.

In summary, many investigators have shown that surgical management is effective for the treatment of calcifying tendinitis. Surgery is primarily indicated for the treatment of symptomatic patients with calcifying tendinitis in the formative stage, which is confirmed on radiographs demonstrating a relatively homogeneous calcific deposit. Patients who have acute severe symptoms or patients with evidence of heterogeneous calcification are likely to be in the resorptive phase and, therefore, continued conservative management would be recommended. Patients treated surgically during the resorptive phase are likely to have favorable results because the natural history is that most of these patients will go on to resolve their calcifying tendinitis, no matter what treatment is performed.

Surgical Technique

Open Approach

Traditionally, an open surgical technique through a split in the deltoid muscle has been demonstrated to be effective in visualizing and removing the calcific deposit. Codman recommended a local anesthetic, exposure through a split in the deltoid muscle, and removal of the calcific deposit.[21] McLaughlin felt that local anesthetic and treatment in the office was an "invitation to catastrophe." Others have suggested a general anesthetic, with the patient in a sitting position and support for the head.[63] The incision begins over the margin of the acromion and extends downward for approximately 4 cm. The deltoid fibers are split and the calcium deposit is readily apparent if it is in the supraspinatus, the most common location (Fig. 6-12). The site of the deltoid split may need to be modified, depending on the exact location of the deposit. The deposit is incised in line with the fibers of the rotator cuff tendon, and the contents are allowed to escape or are removed with a curette. Postoperative immobilization is unnecessary.

Howorth reported on the surgical treatment of 23 shoulders and 22 patients over a 6-year period.[52] Relief of pain and improved motion was noted in all cases. Eighteen patients were reevaluated at an average of 19 months after their surgery. Only 5 of 18 patients demonstrated excellent results, with no pain, weakness, disability, or limitation of motion; 10 patients demonstrated good results, and three patients demonstrated fair results. Analysis of his study group suggests that four out of the five patients with excellent results were in the resorptive phase, whereas the 10 patients with good results were in the formative phase. Two of the three patients with fair results were also in the formative stage. The other patient had been initially treated for severe shoulder stiffness. Howorth concluded that operative removal of the calcific deposit was the surest and most rapid method of relief.

Friedman treated 20 shoulders in 15 patients with surgical removal.[33] Most of his patients had acute pain a few days to 2 weeks before the surgery. Twelve of the shoulders had deposits measuring over 2 cm, with five of the shoulders having deposits measuring more than 3 cm. Eleven of

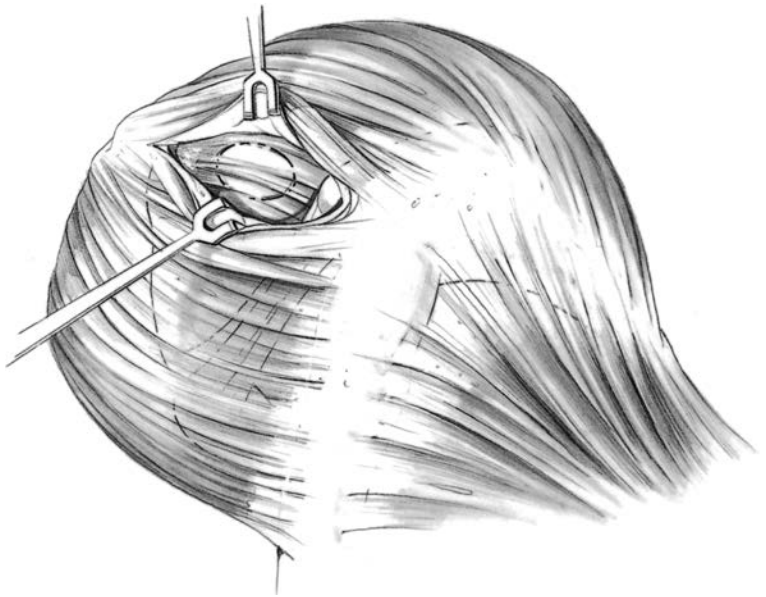

Figure 6-12 The approach to the calcific deposit is dependent on its location. For deposits in the supraspinatus tendon, the most common location, a small skin incision in Langer's lines is followed by a split of the deltoid at the anterolateral deltoid raphe. After incising the underlying bursa, the deposit is seen within the substance of the tendon. Incision of the tendon is performed in line with the fibers of the tendon, and then the calcific material is completely removed. At the completion of the procedure, all layers are closed "side to side," permitting an accelerated rehabilitation program.

the 20 shoulders had been treated initially with infiltration and needling that had failed to relieve the acute symptoms. A deltoid split was used to approach the deposit. The deposit was either decompressed by incising the center and allowing the material to escape under pressure, or curetted if the deposit was dry and gritty. In most patients, acute pain was relieved within 1 or 2 days after surgery. Friedman noted that there was a select group of patients who seemed to take an extended period of time to recover from a limited surgical approach. In one patient, severe stiffness developed, with range of motion returning after 4 months.

Harmon performed a direct surgical excision of the calcific deposit in 104 shoulders, which represented 17% of his patient population with calcifying tendinitis.[49] His indications for surgery included two different classes of patients: (a) those who had failed repeated needlings and injections and (b) those with fulminating hyperacute symptoms. He considered the latter group a surgical emergency and, after surgical decompression, relief was instantaneous. He recommended nonsurgical treatment in any case of calcific deposits in the subscapularis tendon, for none of his patients presented with acute symptoms, and all were recovering following a simple needling procedure. The outpatient procedure was performed using a local anesthetic and a split through the upper deltoid. Full active motion was attained in 80% of his patients within 3 to 5 days. It was unclear how many patients represented the second class or those with "fulminating hyperacute symptoms." Because these patients are in the resorptive phase of the calcifying tendinitis, it is likely that nonsurgical treatment would also have demonstrated good results.

Moseley recommended operative treatment for patients with mechanical symptoms in addition to pain, or when conservative treatment fails and the patient continues to demonstrate a "stiff and painful" shoulder.[92] He reported 304 cases treated with an open operative approach and direct exploration and removal of the deposit. In his opinion, the "few recurrences" are due to remaining calcific deposit after the procedure. Ghormley also stated that it was essential that all of the calcified material be removed and the tendon left intact. He reported three case reports, all with successful results.[38]

Litchman et al. analyzed 100 consecutive cases of calcific tendinitis treated with an open surgical approach over a 10-year period.[68] They stated that these 100 cases represented less than 1% of the patients seen for calcifying tendinitis, which suggests that 1,000 patients a year presented to their practice with evidence of calcifying tendinitis. The patients were divided into four clinical categories: acute (12%), acute–recurrent (13%), chronic (45%), and chronic with acute exacerbation (30%). These authors concluded that the patients presenting with chronic or chronic with acute exacerbation symptoms benefited the most from the surgical approach. The surgical procedure is performed under a general anesthesia with the arm draped free for rotation during the procedure. The deltoid muscle is split and the deposit identified. Serial vertical incisions in the rotator cuff tendon may be necessary to identify the deposit in chronic cases. The calcareous material is removed and an elliptical portion of the tendon is excised, followed by curettage. The tendon defect is then closed with an absorbable suture. In the review of the 100 cases, patients generally regained normal function of the shoulder. The duration of convalescence following the surgery to full recovery was directly related to the degree of chronic disability before the surgery. The average patient returned to full activities after 4 weeks, but in patients who demonstrated severe restrictions of glenohumeral motion, the time to full recovery was "often doubled or tripled."

Vebostad reported on the surgical treatment of 43 shoulders with calcifying tendinitis treated over a 13-year period.[143] He divided the groups into three different types of operations: Group A was simple excision of the calcific deposit; group B was excision of the deposit combined with partial resection of the acromion; and group C was a partial resection of the acromion alone. There were no specific indications for deciding whether the partial acromion resection should be performed when an identifiable calcific deposit was present; when the deposit could not be easily identified, patients were treated with a partial resection of the acromion alone (group C). Of 43 operations, 34 were considered to be "successful." On average, at 2.4 months following the surgery the pain had been relieved. Patients in the group that had undergone resection of the calcium alone had a much shorter period of recovery. There was no difference in the final results in all three groups. Vebostad recommended a simple removal of the deposit as the preferred procedure. However, in patients who have substantial increase in pain in the middle range of abduction or when the deposit could not be localized, an acromioplasty was beneficial.

Uhthoff et al. should be credited with much of our current understanding of the cell-mediated, nondegenerative characteristics of calcifying tendinitis.[135,138] Armed with an understanding of the basic pathophysiology underlying this disorder, they sought a better understanding of the surgical indications and pathologic findings for this disorder. In a report authored by McKendry et al., 57 patients with calcifying tendinitis were treated with an open procedure by 11 different orthopedic surgeons.[80] All patients were treated with a simple curettage of the calcified material, with removal of a small segment of the adjacent tendon. Surgery was performed an average of 2.8 years after the onset of symptoms. By 6 weeks, 61% of patients were pain-free, but 30% continued to have pain beyond 12 weeks. They concluded that patients with a radiographic fluffy appearance of the deposit associated with acute pain are in a self-healing stage of the disease, and nonoperative treatment is recommended. Patients with long-standing symptoms unresponsive to conservative measures with a radiographic homogeneous appearance of the deposit are appropriately treated with curettage of the calcified material.[141]

Despite the relative success of open decompression of the calcific deposit, some patients failed surgical treatment, with continued symptoms of pain and stiffness. Clinical findings suggested pathology localized to the relation between the affected tendon and the coracoacromial arch; therefore, some authors have investigated the efficacy of an open anterior acromioplasty in the treatment of calcifying tendinitis. Postel reviewed a group of 31 shoulders, with the diagnosis of calcifying tendinitis, that were treated by an acromioplasty alone, without excision of the calcification.[110] The mean duration of symptoms before the operation was 4.6 years. In 14 cases, lavage and needling was attempted, followed by an injection of cortisone, but relief of pain was incomplete. Normal mobility was noted in 15 patients, but the remainder of the patients had significant limitations in arm elevation. Preoperatively, the calcification in 13 cases was heterogeneous, corresponding to the resorptive phase. Eight cases demonstrated a dense homogeneous calcification, and six cases had multiple, small, and dense calcifications. Four cases had a history of calcifying tendinitis, but the calcification had resorbed despite their persistent symptoms. Sixteen cases demonstrated a type II acromion.[12] Acromioplasty was performed using an open technique in 24 cases, with an arthroscopic technique used in seven cases. Adding to the complexity of the evaluation, the acromioclavicular joint was also resected in 13 cases. The results were better in the group for whom the acromioclavicular joint was resected, with no failures and 70% excellent and very good results. Only 40% excellent and good results were achieved in the group with a simple acromioplasty. Improvements in range of motion and decrease in pain were noted in most patients. The authors proposed that when a superficial homogeneous deposit is noted, incision and curettage is effective. However, when there is intratendinous heterogeneous calcification, an acromioplasty should be strongly considered. The authors suggested that the acromioplasty permits reduction in the inflammation of the rotator cuff tendon. It is possible that there are other effects from the acromioplasty in addition to alleviating the mechanical aspects, such as decreasing the pressure within the tendon, thereby facilitating the resorptive process, and the natural cycle of calcifying tendinitis by improving blood flow to the rotator cuff tendon.

Gazielly et al. evaluated the results of open acromioplasty combined with excision of the calcium deposit in 39 patients who had at least a 1-year history of shoulder calcification.[36] All patients underwent an anterior acromioplasty, as well as an excision of the calcification and suturing of the curetted tendon. Follow-up averaged over 2 years. Seventy-four percent of the patients had calcification in the supraspinatus, 20% in the infraspinatus, and 6% in the subscapularis. Intraoperative findings of the calcification demonstrated 74% of patients with a solid or chalk-like consistency, 18% with a fluid consistency, and the remaining 8% with mixed consistency. The functional rating for the operative shoulder corresponded to 98% of the contralateral shoulder function compared with 63% preoperatively. Radiographic follow-up demonstrated that the main calcium deposit had been fully eliminated in 98% of the shoulders. Eighty-two percent of patients felt that they had recovered from their condition, whereas the remaining 18% judged that their condition was much improved or improved. It was the opinion of the authors that the clinical results were dependent on the total excision of the calcification, as well as the suturing of the edges of the tendon, which could only be done through an open

approach. The authors then went on to look at their own personal series of having treated more than 100 calcifications by arthroscopic surgery over a 6-year period. From a comparison of their personal series, it was their opinion that arthroscopic excision is difficult, the learning curve for the surgical skills is steep, and the postoperative morbidity from an open procedure is not significantly different from the arthroscopic technique.

Arthroscopic Approach

Arthroscopic treatment of calcifying tendinitis has several potential advantages. The deltoid split to approach the calcific deposit can now be performed through a small arthroscopic portal. Furthermore, as some authors have suggested, an acromioplasty can also be performed arthroscopically, minimizing the iatrogenic risk of deltoid detachment following open acromioplasty. Ark et al. reported on the results of the arthroscopic treatment of chronic calcific tendinitis in 23 patients at an average of 26 months after the procedure.[4] Indications for surgery were persistent shoulder pain in the presence of calcific tendinitis despite at least 1 year of conservative treatment. Conservative treatment included physical therapy (eight patients), nonsteroidal antiinflammatory medications (16 patients), and steroid injections (14 patients). All patients continued to have pain at night. The calcium deposit was localized to the supraspinatus tendon in 20 patients, the infraspinatus tendon in two patients, and the subscapularis in one patient. Nineteen patients demonstrated a dense calcific deposit consistent with formative phase, whereas four patients demonstrated a fluffy deposit. The arthroscopic procedure was performed in the beach-chair or the sitting position. The authors noted that hypertrophic bursitis was often noted on arthroscopic examination of the subacromial space, and a partial bursectomy was necessary. Other authors have demonstrated that the bursal reaction is primarily a localized reaction to the specific area of pathology.[55] Ark et al. reported the release of the coracoacromial ligament in nine of their patients for hypertrophy or localized inflammation. In three patients that demonstrated a prominence to the anterior acromion, an arthroscopic acromioplasty was also performed. A needle was used to identify and, if possible, release the calcific deposit (Fig. 6-13). Needling was followed by a small longitudinal incision within the cuff in line with its fibers (Fig. 6-14). If necessary, up to three incisions were made to completely expose the deposit. Finally, a small curette was used to further liberate the calcific deposit (Fig. 6-15). Any contents from the deposit and "inflamed tissue" were débrided. Bupivacaine without cortisone was injected at the completion of the procedure.

At follow-up at an average of 26 months after the procedure, 11 patients (50%) had full relief of pain, and an additional nine patients (41%) were satisfied with the results, although they had occasional episodes of discomfort. Two patients had persistent pain and after a second arthroscopic procedure were relieved following 5 months of recuperation. The authors felt that these patients had undergone an inadequate removal of the calcium deposit during the primary procedure.[4]

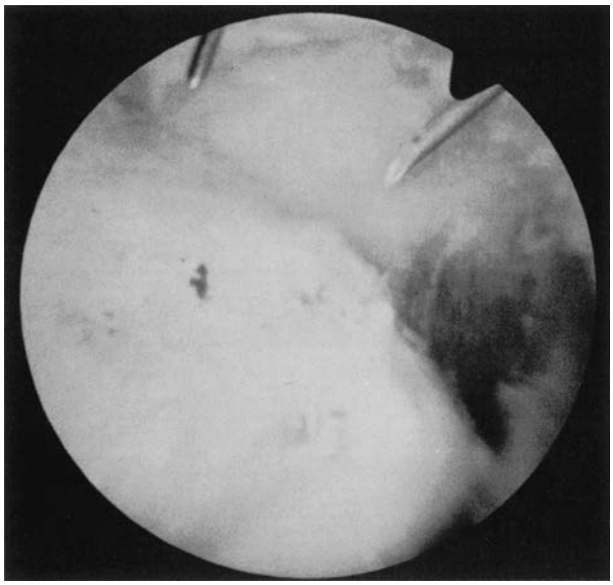

Figure 6-13 Arthroscopic treatment of calcifying tendinitis offers the ability to localize and decompress the deposit with minimal injury to the surrounding healthy tissues. Once the deposit is localized, puncturing the deposit will release the calcific contents. (Courtesy of Dr. Gilles Walch.)

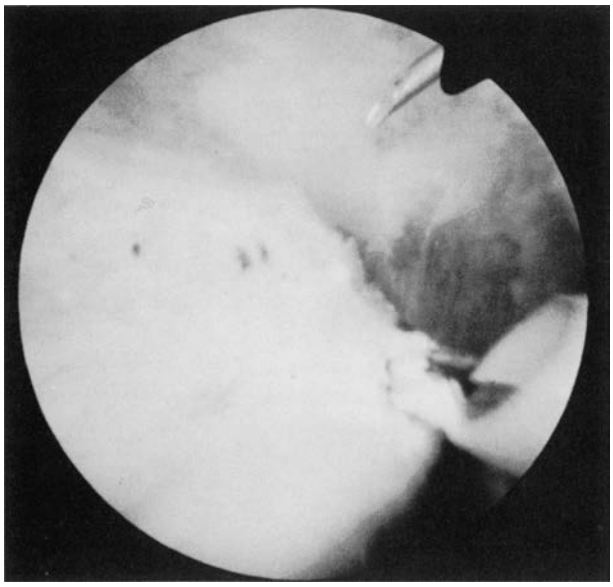

Figure 6-14 A small longitudinal incision in line with the fibers of the tendon can be performed with an 18-gauge needle or a knife. The release of the calcific material is dramatic when the contents are under pressure. (Courtesy of Dr. Gilles Walch.)

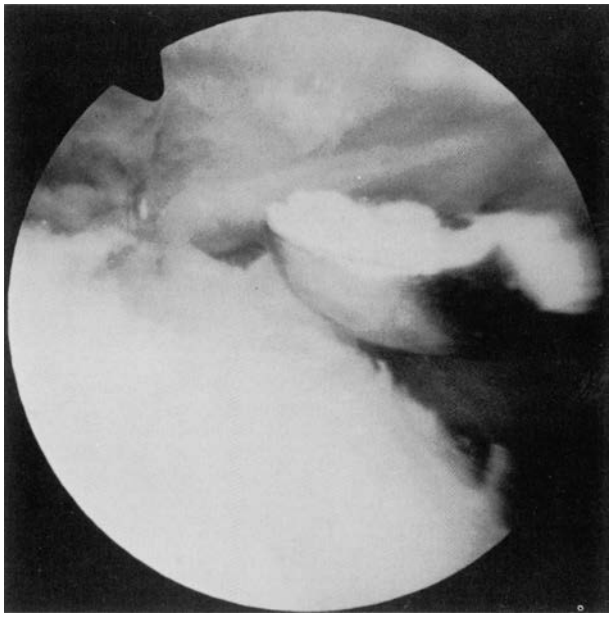

Figure 6-15 A curette can be used to remove any remaining calcific material from the deposit site. (Courtesy of Dr. Gilles Walch.)

Follow-up radiographic studies performed in this patient group demonstrated one patient with an intact large calcium deposit, 12 patients with partial removal of the deposit, and nine patients with complete removal of the calcium. Twelve of 14 patients in whom there was persistent calcium still had significant relief of their pain. The authors, therefore, concluded that complete excision of the calcific deposit during the arthroscopic procedure was not essential for a good outcome.

In fact, a report by Barchilon and Gazielly specifically looked at the amount of calcium deposit removed at the time of shoulder arthroscopy and its relation to overall results.[7] Over a 4-year period, 78 patients with chronic calcifying tendinitis were treated with arthroscopic evaluation and removal of the calcific deposit. From preoperative plain radiographs, the average area of the deposit was 2.1 cm^2. Overall, an average of 74% of the calcific deposit was removed. Removal of deposits from the infraspinatus was less effective than removing deposits from the more common location in the supraspinatus (86%). They also found no correlation between the amount of calcium remaining after surgery and the final result. Kempf et al. also favored isolated excision of the calcific deposit, emphasizing that calcifying tendinitis occurs in an otherwise globally healthy tendon.[60]

However, as noted before, not all patients respond to decompression of the deposit alone, whether it is performed with needling, an open procedure, or an arthroscopic procedure. The poor results have led some surgeons to consider the role of an acromioplasty in the treatment of calcific tendinitis. Ellman reported satisfactory results in 15 of 16 patients who were treated with a débridement or removal of the calcific deposit followed by an arthroscopic subacromial decompression.[30]

Synder and Eppley also described satisfactory results using arthroscopic techniques for decompression of the deposit in 13 of 13 patients, but also suggested that arthroscopic evidence of impingement or hypertrophy of the coracoacromial ligament indicates the need to include an arthroscopic acromioplasty.[132] Re and Karzel supported this recommendation, suggesting that arthroscopic treatment include decompression of the deposit, with an arthroscopic acromioplasty indicated only in those patients with clinical or arthroscopic evidence of impingement.[113]

Mole et al., in the French Society of Arthroscopy, organized a multicenter study to evaluate the results of arthroscopic treatment of chronic calcifying tendinitis.[88] This retrospective review did not randomize patient treatment, but instead looked at the results of treatment by the individual members of the society. One hundred twelve patients were included in the analysis. The surgeons used a variety of treatment options, including excision of the calcific deposit, excision of the deposit with acromioplasty, and acromioplasty without treatment of the calcific deposit. There was no statistically significant difference in the final results using these different treatments. Functional results were evaluated using the Constant score.[37] Excellent results were achieved in 61% of patients, very good results in 24%, good results in 7%, and fair results in 5%. Eight patients had poor results. Acromioplasty did not provide any additional improvement in the final results. However, the authors recommended that an acromioplasty be performed when the calcification could not be found, which occurred in 12% of their patients. The most important factor for a successful result appeared to be the ability to remove the deposit. Poor prognostic factors included the preoperative limitation of passive mobility, the existence of calcific deposits of the lesser tuberosity, and the existence of a rare complete or significant intertendinous tear of the rotator cuff tendon that was found in 4% of the cases.

Gleyze and colleagues also concluded that isolated excision of the calcification is the appropriate treatment, with little evidence for the routine performance of an acromioplasty.[39] However, using the French Society of Arthroscopy classification of radiographic findings in calcifying tendinitis, an acromioplasty is recommended with or without excision of the remaining calcific deposit in patients with type C calcification (heterogeneous) and a lack of clinical evidence of acute pain. This is true for supraspinatus calcific deposits alone. This pattern, radiographically, may be part of the resorptive process; however, the lack of hyperalgesic pain and the presence of chronic symptoms suggest that the resorptive process has paused or has failed. In this study, symptoms were present for more than 1 year. It is suggested by Kempf et al. that these tendons have been unable to complete the typical calcifying tendinitis cycle

and, therefore, the patient is left with a pathologic supraspinatus tendon.[60] Consequently, an acromioplasty may remove the mechanical irritation from the overlying acromion or decrease the intratendinous pressure, allowing increased blood flow and possibly stimulating the completion of the resorption cycle with improved tendon healing. The physiologic effects of an acromioplasty for chronic calcifying tendinitis are unknown.

AUTHORS' RECOMMENDED TREATMENT

The treatment of calcifying tendinitis is based on a thorough understanding of its cycle, the clinical presentation, and the radiographic findings. All three aspects are crucial to avoid treatment that is not only less successful but possibly harmful to the rotator cuff. Because calcifying tendinitis is generally a transient phenomenon, any treatment that is potentially harmful or may disrupt normal tissues around the shoulder must be clearly indicated before it can be recommended.

With these principles in mind, patients presenting with acute pain representing the resorptive phase of calcifying tendinitis are most appropriately treated with nonsurgical measures. Patients with calcifying tendinitis in the formative phase are best treated initially with nonoperative measures. If the condition fails to resolve, surgical treatment is effective in alleviating the patient's symptoms and allowing a return of shoulder function. The most difficult decision is with those patients for whom there appears to have been the initiation of the resorptive process, and yet symptoms and radiographic findings persist. Despite the same epidemiologic attributes, the various manifestations of calcifying tendinitis must be managed with unique strategies (Fig. 6-16).

Conservative Treatment

Virtually all patients with calcifying tendinitis are best treated nonoperatively. When patients present with the acute symptoms of the resorptive phase of this disorder, intervention is dictated by the patient's severity of symptoms. In this phase, patients often present with severe pain and an inability to move their shoulder. Many patients are desperate for relief from the pain that interferes with every aspect of their life, including sleep. Explaining to the patient the transient nature of their severe pain is not reassuring at this time. Although nonsteroidal antiinflammatory medications and even narcotics are helpful, their benefit is minimal, and usually it does not allow patients to sleep comfortably. Physical therapy and gentle motion exercises are likely to aggravate the symptoms; therefore, they are not initially indicated.

The primary goal of treatment is pain relief, and the most effective method to accomplish this task is needling of the deposit. The deposit is carefully evaluated using plain radiography. Bilobular or multilobular involvement should be noted. The anteroposterior views of the shoulder, including rotation views, will allow the deposit to be localized. Fortunately, most deposits are in the supraspinatus, which is easily approached without concern for injuring neurovascular structures. Deposits in the infraspinatus will often demonstrate an area of localized soft tissue prominence and tenderness on the lateral side of the shoulder. With the arm at the patient's side, lesions in the supraspinatus are referenced from the anterolateral corner of the acromion, which is usually in close proximity to the deposit. Furthermore, palpation of the bicipital groove provides an additional reference point on physical examination. Palpation directly over the deposit is accompanied by a severe worsening of the pain. If necessary, the arm can

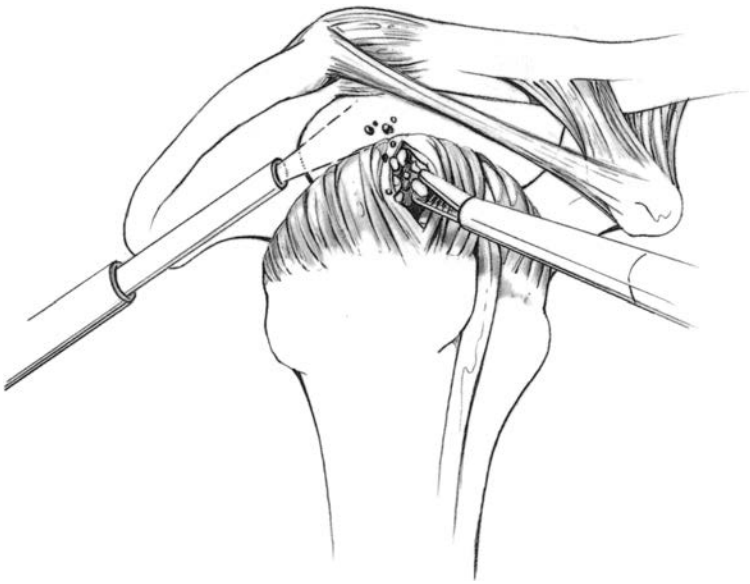

Figure 6-16 Arthroscopic approach to a calcific deposit within the tendon of the supraspinatus. The arthroscope is in the posterior portal, with the working instruments in the lateral portal. An anterior portal is created for outflow. A basket forceps or small curette placed in the lateral portal can be used to remove the calcific material.

be placed into a slight amount of extension to move the site of the deposit from under the acromion. Once this site is localized, then 20 to 30 mL of lidocaine is used to anesthetize the skin at a site that will allow directed needle penetration. Most commonly, this is at the anterolateral edge of the acromion. A local anesthetic is used to anesthetize the skin. The needle is then directed toward the deposit in the rotator cuff tendon. A small amount of local anesthetic is injected as the needle progresses toward the deposit, anesthetizing the entire path. Slowly progressing the tip of the needle may allow the physician to sense the resistance or roughness of the calcific material. Aspiration is attempted at this time, although this is difficult and often unsuccessful. A repeated needling or puncturing of the area containing the deposit is performed approximately five to 10 times. At the completion of the procedure, a corticosteroid injection accompanied with a long-acting anesthetic is placed into the subacromial space, not the tendon. The use of cortisone has not been associated with any adverse effects specific to the treatment of calcifying tendinitis. Patients are instructed to rest their shoulder for 1 to 2 days and then to gradually work on regaining their range of motion. Furthermore, they are provided with a prescription for either a nonsteroidal antiinflammatory medication or mild narcotic. Application of ice to the site of pain may also help alleviate their symptoms. Frequently, pain relief is dramatic, and the symptoms quickly dissipate. Although physical therapy may be necessary for patients who demonstrate shoulder stiffness, this is frequently not prescribed, for patients will return with close to a full range of motion and decreased pain within a few weeks, sometimes as quickly as a few days.

If the first attempt has been unsuccessful in alleviating symptoms, a second needling is performed. If a partial response was achieved, a second "blind" needling using the technique described is attempted in the office. However, if there was no response to the first needling, the second needling procedure is performed in the radiology suite with the assistance of fluoroscopy, or with the use of ultrasound if available, to accurately locate the deposit. After anesthetizing the skin and soft tissue along the path to the deposit, the deposit is directly needled, followed by an injection of a solution of anesthetic and cortisone into the subacromial space. In our experience, this treatment plan has been successful in resolving pain rapidly with generally a full return of shoulder function. Surgical intervention has been unnecessary for the treatment of acute symptoms associated with the normal resorptive phase of calcifying tendinitis.

Treatment during the formative stage, or resting stage, is based on the duration and severity of the symptoms, as well as the examination findings. Initial conservative measures include nonsteroidal antiinflammatory medications for pain relief. There is no evidence that these medications accelerate the disappearance of the calcium or positively affect the completion of the calcifying tendinitis cycle. When pain is under control, a daily program of stretching exercises will help maintain the mobility of the shoulder. Physical modalities, such as ice or heat, may be beneficial. Other physical modalities provided under the supervision of a therapist, such as ultrasound or diathermy, may have some short-term benefit by relieving pain, but again, these have not been proved effective in the resolution of this condition. The cost of these physical modalities is substantial and, therefore, the authors cannot recommend their use until some benefit is proved other than short-term pain relief.

Corticosteroid injections are recommended if the symptoms persist despite oral medications and therapy. Corticosteroids may be used initially in those patients with an intolerance or allergy to nonsteroidal antiinflammatory medications. There is no evidence that a corticosteroid injection will facilitate the resolution of the calcifying tendinitis cycle. Uhthoff and Sarkar have recommended against their routine use owing to the theoretical concern that the effect of corticosteroids may actually interrupt or delay the resorptive phase.[141] However, this concern has not been validated in a well-controlled study, and we have noted marked improvement in symptoms with one or two injections in those patients who have had symptomatic calcifying tendinitis for less than 6 months.

"Blind" needling of the calcific deposit during the formative stage is not recommended. These dense homogeneous deposits do not permit an easy release of the calcific material. Dual needling and lavage may be beneficial. To be successful, this technique requires fluoroscopic isolation and may also involve a regional or general anesthetic agent to relax the patient and permit the dual needling of the calcific deposit, followed by lavage. The goal of needling and lavage should be removal of as much of the deposit as possible. Following the procedure, a brief period of rest is recommended, along with nonsteroidal antiinflammatory medications or a mild narcotic for pain relief. Physical therapy is directed toward reestablishing normal shoulder motion, then progressing to recovery of shoulder function. Patients should be forewarned that even successful recovery from this procedure is extended, often lasting 3 to 6 months.

Patients who have had chronic symptoms for more than 6 months with radiographic evidence of calcifying tendinitis meet the appropriate criteria for surgical intervention. Imaging of the rotator cuff tendon is also recommended if surgical intervention is contemplated. Although we have rarely seen evidence of true tendinopathy, such as a significant partial-thickness or full-thickness tearing of the rotator cuff, others have reported an incidence of approximately 4% in this patient population.[88] Furthermore, two studies from the same institution in Taiwan suggested a significantly higher incidence of rotator cuff tearing in older patients and in patients with Asian heritage presenting with long-standing calcifying tendinitis in the formative

phase.[53,56] Knowledge of a significant tendinopathy will enable better preoperative planning for the patient and the surgeon.

We prefer an arthroscopic approach to the surgical treatment of calcifying tendinitis. Arthroscopy permits localization of the deposit and removal of the contents of the deposit, while minimizing injury to the unaffected tissues. We have also incorporated a side-to-side repair of the rotator cuff defect when the removed calcific deposit left a substantial defect in the tendon. In our opinion, an acromioplasty is rarely necessary. However, an acromioplasty may be indicated when significant clinical or arthroscopic findings of impingement are present. Furthermore, an acromioplasty appears to be beneficial when the calcific deposit cannot be localized owing to a poorly circumscribed lesion, usually seen as a type C calcification pattern on preoperative radiographs. Although arthroscopic techniques are preferred, open procedures performed through a split in the deltoid tendon without detaching the anterior acromion attachment of the deltoid origin are equally effective for localization of the deposit and removal of the contents. Both surgical procedures are performed on an outpatient basis, and patients are started on an early range-of-motion program. Postoperative corticosteroid injections have not been indicated.

Open Surgical Technique

Exposure of the involved rotator cuff tendon depends on its location. Most rotator cuff calcifications will be within the substance of the supraspinatus tendon. This tendon is readily approachable through a split in the deltoid muscle centered at the anterolateral edge of the acromion. Regional anesthesia, general anesthesia, or a combination of the two techniques can be used for the surgical procedure. A long-acting local anesthetic is injected at the surgical site. The use of local anesthesia alone has been described, but is not recommended. An incision is made in Langer's lines from the lateral corner of the acromion toward the coracoid process. The deltoid raphe is identified perpendicular to this skin incision. Dissection is carried through the anterolateral deltoid raphe, beginning at the acromion and progressing inferiorly. The fibers are split and separated by self-retaining retractors. Three centimeters of exposure provides a generous approach to the supraspinatus tendon without risk of injuring the innervation of the anterior deltoid. The patient is positioned in the beach-chair position for the surgical procedure.

Once the split in the deltoid tendon is made, the overlying bursa is incised to expose the rotator cuff tendon. A partial bursectomy is performed to have adequate visualization of the rotator cuff tendon. When locating the deposit, the most significant intraoperative landmark is the biceps tendon. In general, a supraspinatus calcific deposit will be approximately 1 cm posterior to the biceps tendon and approximately 1.5 cm from the attachment site of the supraspinatus tendon on the greater tuberosity. Once the site of the calcific deposit is identified, either by direct visualization, palpation, or estimation from the preoperative radiographs and intraarticular landmarks, an incision is made at the site of the calcific deposit in line with the fibers of the rotator cuff tendon. The calcium is exposed, irrigated, and then débrided with a curette to remove as much of the calcific deposit as possible. Although it is not necessary to completely remove all of the calcium, it does appear to be beneficial to remove as much of the contents as possible to decrease the intratendinous pressure, the thickness of the tendon, and the risk for an acute inflammatory reaction to remaining calcific debris. If a significant amount of the tendon appears to be involved, side-to-side repair of the tendon edges with absorbable sutures can be performed, but the benefit is unclear. At the completion of the removal of the calcific deposit, the subacromial space is irrigated thoroughly. The deltoid is closed with a side-to-side repair using absorbable sutures. The skin is closed with a running subcuticular closure supported with small adhesive strips. Patients are allowed range of motion as tolerated. Sutures are removed within 5 to 7 days. If the patient has difficulty regaining range of motion, a supervised therapy program may be initiated. Recurrence of the calcific deposit does not occur, although some of the deposit may persist after surgery. Patients may exhibit a prolonged course of recovery, taking 3 to 6 months to regain their motion, strength, and function.

Arthroscopic Technique

Preoperative assessment with radiographs including an anteroposterior view of the humerus in neutral rotation, external rotation, and internal rotation is essential to predict the site of the calcific deposit. Anesthesia can be delivered through a regional interscalene block, a general anesthetic agent, or a combination of the two. We prefer a beach-chair position, but a lateral decubitus position with a gentle amount of traction on the arm is also effective. In the lateral position, the arm is placed at approximately 20 degrees of abduction with slight forward flexion. Approximately 10 lb (4.5 kg) of traction are used for smaller patients, 15 lb (6.8 kg) for larger patients.

The glenohumeral joint inspection is carried out in a systematic fashion. Partial-thickness and full-thickness rotator cuff tears have been seen with calcifying tendinitis and can be evaluated with standard arthroscopic techniques. Marking the deposit is possible if the area of involvement can be identified from the glenohumeral joint. An 18-gauge spinal needle is passed through the skin, into the area of the deposit, then into the glenohumeral joint while the arthroscope is maintained in the glenohumeral joint. A stiff synthetic suture is then passed through the needle into the glenohumeral joint and a grasper is used to pull the suture

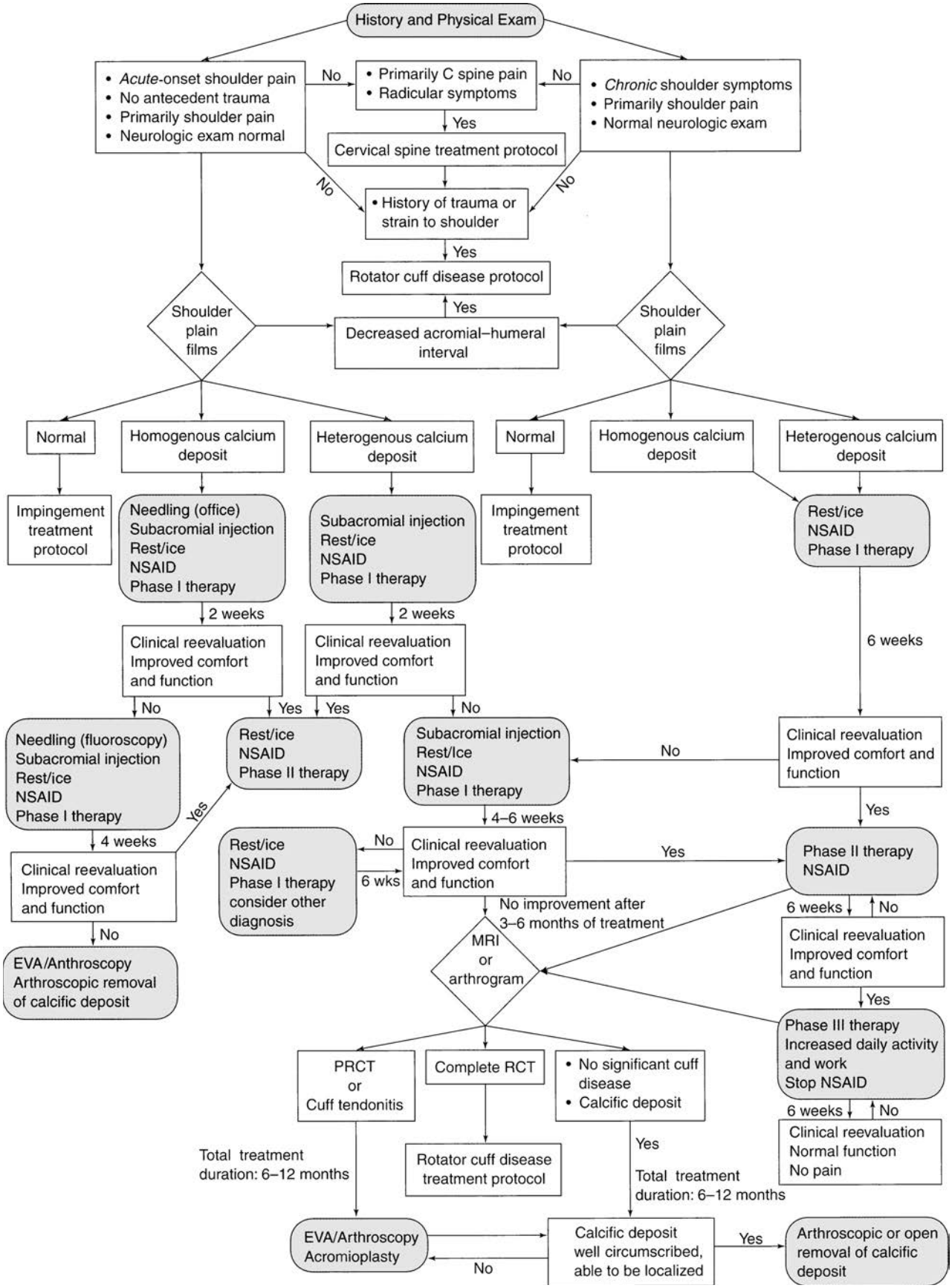

Figure 6-17 Algorithm for evaluation and treatment of calcifying tendinitis. EVA, ; MRI, magnetic resonance imaging; NSAID, nonsteroidal antiinflammatory drug; PRCT, partial rotator cuff tear; RCT, rotator cuff tear.

out of the anterior portal. This technique is also used to mark partial-thickness rotator cuff tears for later inspection from the subacromial space.

Once the subacromial space has been established and is acceptable, which often includes removing some of the bursa, the examination of the subacromial space also proceeds in a systematic fashion. In addition to evaluating the coracoacromial ligament and undersurface of the acromion, careful inspection of the rotator cuff tendon is necessary. Once the site of the calcific deposit is located, it is generally necessary to establish a third lateral portal for a direct approach to the calcific deposit. Once the calcific deposit has been clearly visualized, a no. 11 blade on a long-handled knife, an arthroscopic blade, or an 18-gauge needle can be advanced through the lateral portal. The supraspinatus tendon is incised in line with its fibers to expose the calcific material. Visualization is maintained using an arthroscopic pump. Once the calcific deposit has been incised and some of the material removed, a curette can be advanced through the lateral portal to débride the remaining contents of the deposit (Fig. 6-17). An arthroscopic power shaver is not recommended because inadvertent removal of some of the normal tendon is likely. A hooded shaver may be beneficial without risking injury to the remaining intact tendon. The shoulder should be irrigated thoroughly and exchange of fluid should be rapid to maximize removal of the calcific material. We have also incorporated a side-to-side repair of the rotator cuff defect when the removed calcific deposit left a substantial defect in the tendon. The skin is closed with an interrupted suture closure of the portals. Patients are allowed range of motion as tolerated. Sutures are removed within 5 to 7 days. If the patient has difficulty regaining range of motion, a supervised therapy program may be initiated. Recurrence of the calcific deposit does not occur, although some of the deposit may persist after surgery.

If the calcific deposit is indistinct or cannot be localized, an arthroscopic acromioplasty is advised. The benefit of the acromioplasty appears to be related to the decompression or widening of the subacromial space, decreasing the mechanical irritation of the rotator cuff tendon. There may also be underlying physiologic changes, such as decreased tendon pressure and improved blood flow, that may allow tendon healing or simply the resolution of pain. The need for an acromioplasty may be predicted from the preoperative radiographs. An inhomogeneous, poorly circumscribed lesion associated with chronic symptoms suggests a stop in the normal cycle of calcifying tendinitis. The calcific material is within the tendon fibers, but not readily accessible; therefore, it cannot be directly addressed with surgical removal. In our opinion, a well-performed arthroscopic acromioplasty in this setting has distinct clinical advantages over a traditional open acromioplasty.

SUMMARY

The key to successful treatment of calcifying tendinitis is a complete understanding of the pathophysiology of this disease. Calcifying tendinitis tends to follow a characteristic disease cycle in most patients. The cycle includes the formation of the calcific deposit, the resorption of the deposit, and then tendon restoration. Some patients demonstrate findings that suggest an aborted cycle, with chronic symptoms and radiographic evidence of persistent calcific material or incomplete resorption. However, clinical characteristics and cadaveric studies suggest that resorption of the deposit always occurs, even though the resorption may occur over many years.

Calcifying tendinitis is a condition of unknown cause, although it is understood that the calcium deposition is a cell-mediated process that occurs in nondegenerative rotator cuff tendons. The epidemiology of this disease is the same despite various physiologic, clinical, and radiographic stages. Understanding the pathology and then combining this understanding with the presenting clinical and radiographic features enable one to formulate successful treatment strategies.

Treatment options are numerous, but the most successful strategies have included decompression of the calcific deposit. During the acute hyperalgesic stage, consistent with the resorptive phase, pain relief is the primary goal. This can be rapidly accomplished by needling the calcific deposit without interrupting the natural resolution of the resorptive phase. Chronic symptoms are more difficult to treat, and they may require direct surgical intervention. The primary goal of surgery is the decompression and removal of the calcific material without causing undue harm to tissues not involved in the disease process. Arthroscopic decompression and removal of the deposit offers an accurate and effective approach for this condition. When symptoms persist despite the absence of a homogeneous, well-circumscribed lesion, an acromioplasty is beneficial in the treatment of the persistent tendinopathy, although the postoperative recovery is longer than expected. In the future, a better understanding of the cause and the cellular signals that trigger the resorptive process may permit better medical management of this condition.

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7 Disorders of the Biceps Tendon

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INTRODUCTION

The clinical significance of the long head of the biceps tendon to shoulder function has been a subject of controversy for some time. Opinions on the contribution of the intraarticular biceps tendon have varied the entire spectrum, with proponents suggesting a vestigial function analogous to an "appendix of the shoulder" to those who believe it can play a critical role in shoulder stability. Historically, there have been wide shifts in surgical treatment of the long head of the biceps tendon. In the 1940s, the biceps tendon was seen as a major source of shoulder pain and tenodesis favored as a primary procedure.[33,50,90] As the focus shifted to the rotator cuff, tenodesis of the long head of the biceps became less popular. Later, concerns regarding a possible secondary role of the long head of the biceps tendon led to recommendations for preservation whenever possible.[81,95,115] More recently, previously accepted theories on the functional role of the biceps in head depression have undergone reexamination in the context of an increased awareness about the symptomatic significance of a retained, chronically inflamed tendon.[12,97,110,137]

With the accumulation of research and clinical experience specific to the long head of the biceps tendon, it is becoming increasingly accepted that pathology in this structure can have significant symptomatic consequences that will require directed treatment plans. As with most controversial treatment issues, the proper strategy will ultimately reflect a balance of previously recognized concerns about functional deficits and persistent symptoms. The purpose of this chapter is to review the anatomic, functional, and clinical information about the long head of the biceps tendon required to formulate logical treatment plans.

ANATOMY

The long head of the biceps tendon arises from the posterosuperior labrum and the supraglenoid tubercle. The site of origin of the long head has been shown to be variable. Habermeyer et al. described the long head origin in the majority of cases (50%) as being completely labral. Attachment to the supraglenoid tubercle alone was less common (20%); the tendon more commonly (30%) arose from both origins, sometimes appearing bifurcated.[54] Another study of the origin of the tendon of the long head of the biceps in 105 cadaveric shoulders showed that 40% to 60% arose from the supraglenoid tubercle and surrounding labrum.[130] The rest of the origins were from labrum alone. The labral attachment has been further described as being in four types: type I, the labral attachment being entirely posterior (22%); type II, most of the labral contribution being posterior with some anterior component (33%); type III, equal contributions from both the anterior and posterior labra (37%); and type IV, with most of the contribution being anterior (8%)[130] (Fig. 7-1). Additionally, the orientation of the tendon attachment to the superior glenoid tubercle was dependent on the type of labral attachment. In those biceps tendons with a mostly posterior labral attachment, the superior glenoid tubercle origin was more likely to be posterior at a 1 o'clock position for a left glenoid. In those situations with an anterior labral attachment, the glenoid tubercle origin was more likely to be slightly anterior at an 11 o'clock orientation for a left glenoid.

The relative biomechanical contributions of the supraglenoid and labral attachments of the biceps origin have been investigated by Healey et al.[63] This cadaveric study focused on the effect of selective release of the long head of biceps origins on the linear stiffness and displacement of the biceps tendon. In all specimens, release of the bony anchor resulted in significantly greater displacement of the biceps tendon in comparison to release of the labral attachments. Isolated release of the bony anchor resulted in a 52% reduction of the stiffness of the biceps tendon in comparison to a 15% decline in stiffness observed with isolated release of the superior labrum.

The tendon is encased within the synovial sheath of the glenohumeral joint (Fig. 7-2). It traverses obliquely within the shoulder joint arching anteriorly over the head of the humerus and exits the shoulder joint beneath the transverse humeral ligament along the intertubercular sulcus, also referred to as the bicipital groove (Fig. 7-3). Open only at the glenohumeral cavity medially, the synovial sheath ends as a blind pouch at the end of the bicipital groove (see Fig. 7-2). In 50 cadaveric dissections, Habermeyer and coauthors found the mean length of the tendon to be 9.2 cm.[54,56] The tendon was widest at its origin (mean 8.5×2.8 mm) and progressively narrowed down to form the muscle belly (mean 4.5×2.8 mm). At the level of the insertion of the deltoid, the tendon evolves into an elongated muscle belly joined medially by the short head. The two bellies lie closely approximated but are separable until about 7 cm from the elbow joint.

The ascending branch of the anterior humeral circumflex artery runs adjacent to the tendon and is the main arterial supply to the long head in the bicipital groove. Labral branches of the suprascapular artery may also contribute to the blood supply of the long head of the biceps tendon at its origin.[30,113] The musculocutaneous nerve (C5–7) constitutes the motor innervation to the muscle.

RESTRAINTS OF THE LONG HEAD OF THE BICEPS TENDON

While the greater and lesser tuberosities help to contain the tendon within the bicipital groove, retention of the long head of the biceps is mainly provided for by the surrounding soft tissues.

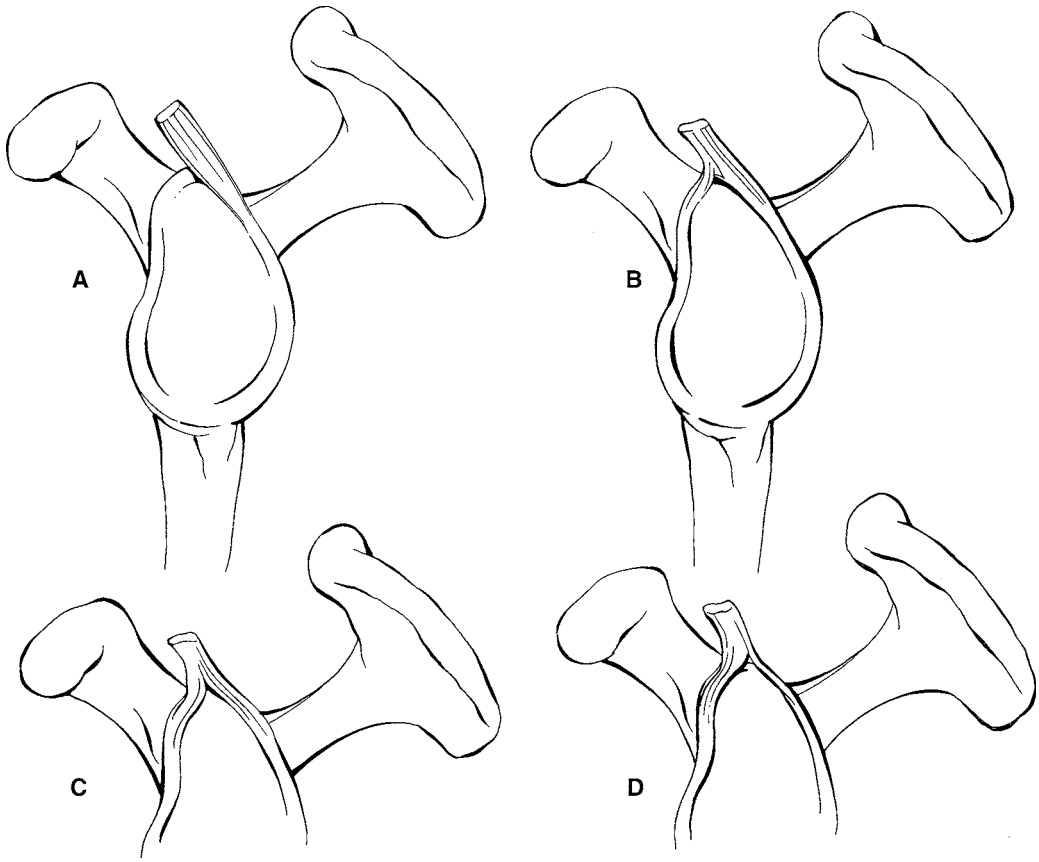


Figure 7-1 Schematic drawings of the variations in the biceps origin. **A.** Type I. The labral attachment is almost entirely posterior. This is seen 22% of the time. **B.** Type II. Most of the labral contribution is posterior with a small amount of anterior contribution. This is seen 33% of the time. **C.** Type III. There are equal contributions from both the anterior and posterior labra, and this is seen 37% of the time. **D.** Type IV. Most of the labral contribution is anterior with only a small posterior contribution, and this is seen 8% of the time.

The bicipital groove is hourglass shaped, being narrowest and deepest in its midportion. With a mean width of 9.6 mm and depth of 2.2 mm, it is wide and shallow at its entrance. The mean measurements at the midportion are 6.2 mm wide and 5.0 mm deep. The tunnel flattens out at its exit, 7.3 mm wide and 2.4 mm deep.[54,56] The medial wall angle varies widely among individuals, with reported mean values from 56 degrees to 64 degrees.[65] Although the humeral head size is larger in men, there is no significant difference between the two sexes in the relative size of the groove.[5] In some cases, a "supratubercular ridge" extends forward and downward from the region of the articular cartilage to the upper and dorsal portion of the lesser tuberosity. First described by Meyer, it is estimated to occur in 17.5% of humeri.[88,89] The significance of the supratubercular ridge is unclear; but according to Meyer, it diminishes the efficacy of the tuberosity as a trochlea, and when present can predispose to dislocation of the long head of the biceps tendon out of the groove.

Pfahler and coauthors investigated the bony anatomy of the biceps tendon radiographically, comparing differences in anatomy between patients diagnosed with anterior shoulder pain versus normal controls.[109] Radiographs of the bicipital groove were obtained to define the dimensions of the groove and to assess for local degenerative changes. A significant variation in the medial opening angle of the biceps groove was noted in all subjects, average 44 degrees, with a range of 9 to 74 degrees. The presence of degenerative radiographic changes in the biceps groove was noted in 44% of patients with ultrasound-proven biceps pathology. The authors were able to correlate specific anatomic variations in the bony anatomy of the biceps groove that correlated with pathologic changes of the biceps tendon on ultrasound. Compared with the control group, a flat biceps groove and a small medial opening angle were associated with ultrasonic abnormalities of the biceps tendon.

Laterally, the long head of the biceps tendon runs in the rotator interval. This triangular space between the supraspinatus and the subscapularis tendons is devoid of rotator cuff and bridged by fibrous tissue. The transverse humeral ligament bridging the bicipital groove forms the

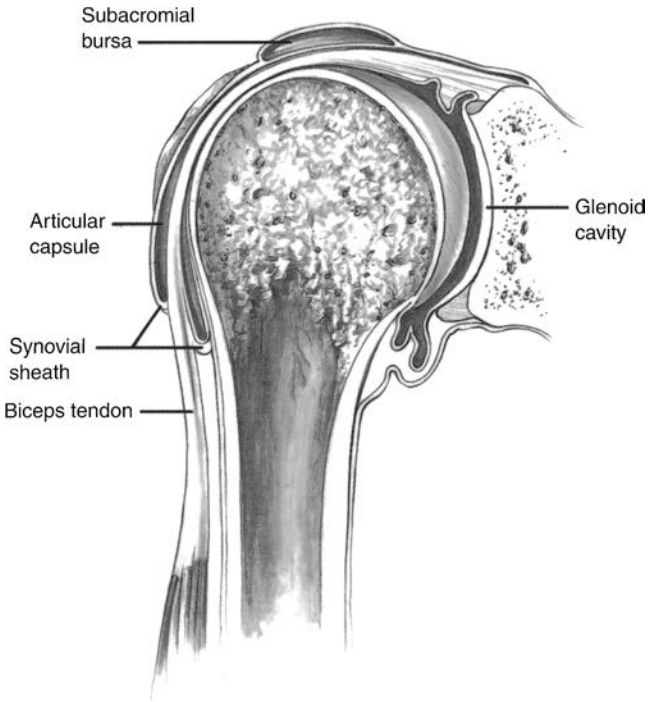


Figure 7-2 A schematic of a sagittal section taken through the glenohumeral joint. The biceps tendon is covered by reflection of the synovial sheath, which maintains a tendon as an extra synovial structure despite its intraarticular location. The synovial sheath ends as a blind path distally outside of the bicipital groove.

Figure 7-3 A schematic of an axial view of the glenohumeral joint showing the relative position of the biceps tendon from internal to external rotation. In internal or neutral rotation, the biceps maintains an oblique course across the joint starting from an anterior location. It is only in external rotation that the biceps tendon courses over the apex of the humeral head.

apex of the triangle laterally. The coracoid process with the origin of the coracohumeral ligament medially constitutes the base. The coracohumeral ligament has a broad origin extending from the base of the coracoid process along the lateral border of the coracoid for about 18 mm[61] and forms the roof of the space. Located in the floor are the long head of the biceps and the superior glenohumeral ligaments (Fig. 7-4).[26,27,49,56]

Most authors agree that the transverse humeral ligament bridging the bicipital groove does not play an important role in retaining the biceps tendon.[1,56,88,89,107] The ligament is often weak or absent and is present lower down in the bicipital groove. Rather, the coracohumeral ligament is believed to be more important. It consists of two bands, the superior band blending into the adjacent tendinous edge of the supraspinatus and inserting into the greater tuberosity.[26,27,38,56] The inferior band blends with the superior border of the subscapularis and inserts into the lesser tuberosity and the transverse humeral ligament. The superior glenohumeral ligament is also important for biceps tendon retention within the groove. It has two narrow osseous attachments medially. One attachment is from the apex of the labrum, shared with the biceps tendon, and the other is from the base of the coracoid process.[129] While distinct from the coracohumeral ligament at its medial attachment to the superior labrum and the neck of the scapula, the superior glenohumeral ligament blends imperceptibly with the coracohumeral ligament laterally at its insertion into the superior surface of the lesser tuberosity.[27] Thus, in the lateral part of the rotator interval, at the proximal portion of the bicipital groove, the roof and the floor blend to form a sling for the biceps tendon as it enters the bicipital groove.

Werner et al. studied the macroscopic and microscopic anatomy of the rotator interval in reference to the constituents of the biceps sling.[135] Cadaveric sections of the lateral portion of the rotator interval showed the superior glenohumeral ligament to form a U-shaped fold crossing under the biceps tendon before inserting into the proximal aspect of the intertubercular groove. The superior glenohumeral ligament appeared to form a semicircular anterior support for the lateral part of the biceps tendon. Microscopic analysis demonstrated that fibers of the superior glenohumeral ligament covered the inferior, anterior, and superior aspects of the biceps tendon with a collagen fiber orientation perpendicular toward the tendon. These authors emphasized the importance of the superior glenohumeral ligament in stability of the long head of the biceps within the rotator interval.

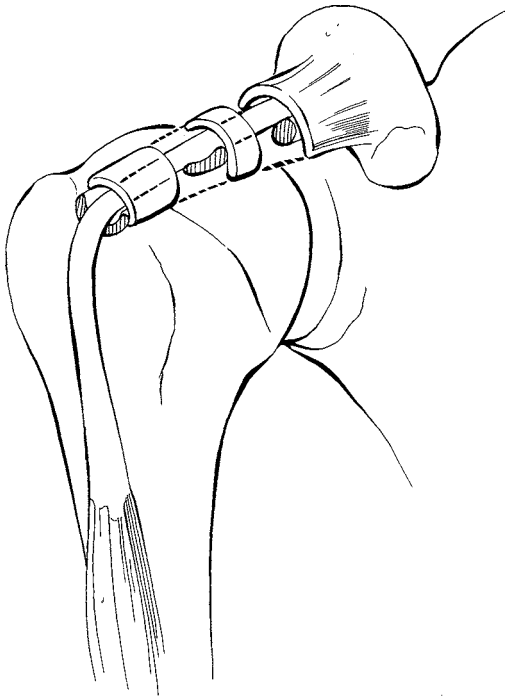


Figure 7-4 A schematic representation of the relationship of the coracohumeral ligament and superior glenohumeral ligament to the long head of the biceps tendon. The long head of the biceps tendon is stabilized in the bicipital groove by the coracohumeral ligament (*black*), which forms the roof, and the superior glenohumeral ligament (*hatched*), which forms the floor.

Distal to the summits of the greater and lesser tuberosities, it is the insertion of the pectoralis major tendon that appears to be important in retention of the long head of the biceps.[1] The sternocostal portion of the pectoralis major tendon, constituting the deeper lamina, inserts more proximally in the humerus than the clavicular portion. A fibrous expansion arises from this head, forming a falciform margin at the deep edge of the tendon, is attached to both sides of the groove, and blends proximally with the capsule of the shoulder joint.

In the absence of pathologic changes in the restraining structures, there are certain anatomic features that may predispose dislocation of the tendon of the long head of the biceps. These include (a) that the intraarticular portion of the tendon may lie more eccentrically over the anterior half of the sloping, smooth, rounded surface of the humeral head, favoring its medial dislocation; (b) a more widened and flattened tendon origin at the superior glenoid; (c) a shallow medial wall of the groove, which would not provide as significant a restraint to medial subluxation of the tendon as the tendon of the long head of the biceps makes an abrupt turn from a horizontal direction to a vertical course at the intertubercular sulcus (the lesser tuberosity acts as the "trochlea" for the tendon at this point); and (d) that the supracondylar ridge, when present, diminishes the prominence of the lesser tuberosity, decreasing its efficacy as a trochlea.[50,88]

FUNCTIONAL ANATOMY OF THE LONG HEAD

Perhaps no aspect of the long head of the biceps tendon has raised as much controversy as its function at the shoulder. The biceps extends from the scapula to the bones of the forearm and thus has potential function at both the shoulder and elbow. Elbow function has been well established to include both flexion and supination. The actions of the proximal tendon around the shoulder have been incompletely defined. Although the long head of the biceps has been postulated to have several roles or functions about the shoulder, definitive experimental proof has been sparse and contradictory, leaving its exact role as controversial.

More complete characterization of the functional role of the long head of the biceps can be clinically important in determining indications for tenodesis. Some reports have suggested a weak humeral head depressor role that increases in relative importance in the presence of rotator cuff tears. Other studies have suggested the long head of the biceps to work solely in relation to the elbow with no significant shoulder-related activity. Neer warned against thoughtless tenodesis of the long head of the biceps because it "destroys its function as a head depressor and may precipitate or escalate an impingement problem."[95] Leffert and Rowe offered the observation that increased size of the tendon with chronic rotator cuff rupture represented increased function compensating for the loss of the rotator cuff and strongly discouraged tenodesis.[81] However, this phenomenon has been seen only with large rotator cuff tears where the tendon was exposed to constant impingement and more plausibly reflected a chronic inflammatory response. Additionally, the more physiologic response of increased muscle mass as opposed to increased tendon size has not been noted. Aside from clinical observations, there have been several approaches employed to study the potential function of the biceps tendon. These include comparative anatomy observations, cadaveric biomechanical studies, and dynamic electromyographic analysis.

Comparative Anatomy

The asymmetrical size of the greater and lesser tuberosities and the angular orientation of the bicipital groove are exclusive to the human shoulder and represent an evolutionary adaptation to the vertical posture. The presence of two heads is unique to primates. In certain quadrupeds such as the horse, which does not have a deltoid muscle, this head is large and functions to elevate the extremity in conjunction with a comparatively large supraspinatus.[67] In quadrupeds the biceps passes over the center of the spherical humeral head in the bicipital groove, which is located between two symmetrical tuberosities and oriented perpendicular to the glenoid. As the scapula and

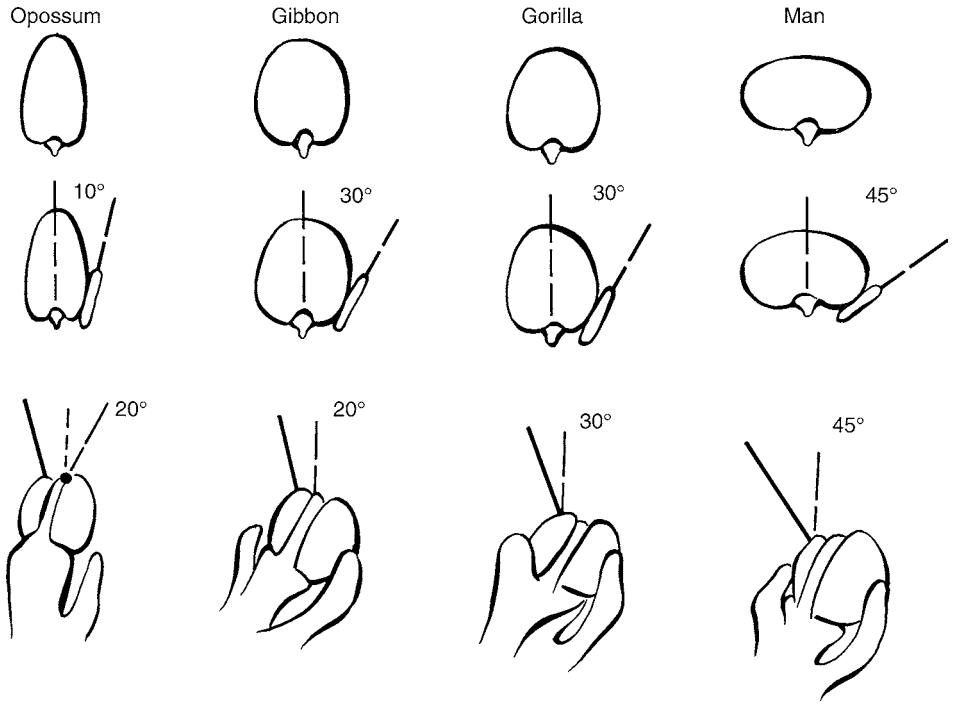


Figure 7-5 A schematic showing progressive change in positioning of the scapula and bicipital groove from the quadruped to the biped. As an adaptation upright posture, the anterior–posterior dimension of the chest wall has decreased progressively. This has resulted in secondary humeral torsion and a displacement of the bicipital groove medially and ventrally in relation to the scapula. Whereas the bicipital groove follows a straight course over the top of the humeral head in the quadruped (opossum), in man it now resides against the lesser tuberosity over the anterior portion of the head. This has resulted in a significant decrease in effectiveness of the biceps to act as an arm elevator.

glenoid face is oriented toward the forward plane, the biceps tendon in quadruped animals contributes to flexion of the shoulder.

In adaptation to an upright posture, the anteroposterior diameter of the chest wall has decreased progressively, and the scapula has rotated dorsally over the flattened chest wall.[65] To allow the articular surface of the humerus, which previously faced dorsally, to continue facing the scapula, the humeral shaft has undergone torsion. This has resulted in a displacement of the bicipital groove medially and ventrally to form an angle of 30 to 40 degrees with the plane of the scapula (Fig. 7-5). This developmental change in the tendon orientation to the glenoid has resulted in a significant decrease in effectiveness of the biceps as an arm elevator, which can only be partially restored with full external rotation of the arm. There has been a compensatory developmental adaptation by the deltoid muscle, which becomes a much more effective elevator in primates secondary to its more distal insertion, increased muscle mass, and adaptations of the acromion to increase the leverage of the muscle.[66]

Hitchcock and Bechtol studied the anatomy of the bicipital tuberosity in 100 dry human specimens and compared them to primate humeri from the Chicago Natural History Museum.[65] The human specimens demonstrated a wide variation in the depth of the bicipital groove. This was expressed as the angle of the medial wall, a smaller angle representing a shallow wide groove. While the majority of human specimens demonstrated a medial wall angle of 60 to 75 degrees, values of 45 degrees or less were observed in 20% of specimens. Except the orangutan (a primate with greater predisposition to tree climbing), who presented values similar to humans, other apes demonstrated deep bicipital grooves with consistent medial wall angles of 90 degrees in all specimens. The shallow bicipital groove in humans was postulated to be important for the development of instability and subsequent inflammation of the long head of the biceps.[65] The considerable variability of the biceps groove was considered to reflect a developmental loss of function in elevation for the biceps muscle. These authors also noted the relative ventral orientation of the long head of the biceps in relation to other mammals. Additionally, they emphasized that human beings will habitually hold their humerus in the forward plane and internal rotation, increasing the poor anatomic arrangement for the biceps to act in abduction. They concluded that an operation to fix the tendon in the bicipital groove, while removing a portion of the tendon above the groove, can relieve the symptoms caused by biceps tendon pathology and does not materially weaken the shoulder.[65]

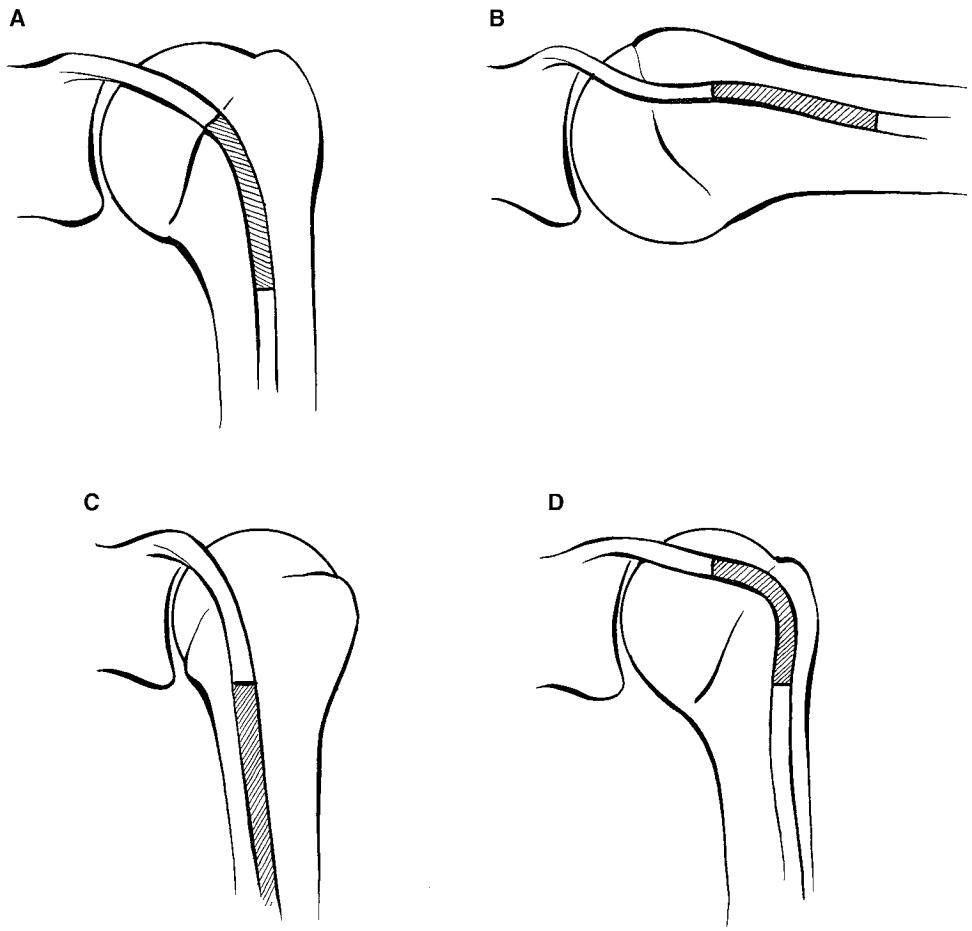


Figure 7-6 Schematic of the relationship of the biceps tendon to the humerus with glenohumeral motion. As described by Lippmann, with direct surgical observation, the biceps tendon played a passive role during humeral motion. The biceps tendon was found to slide freely between the sheath and the joint proper as the shoulder was moved. Varying amounts of the tendon were found to be intraarticular, dependent on shoulder position. A maximum of intraarticular tendon was seen with external rotation and adduction. A minimal amount of intraarticular tendon was present during elevation. **A.** Neutral rotation. **B.** Elevation. **C.** Internal rotation. **D.** External rotation.

Anatomic and Biomechanical Studies

Lippmann surgically observed in patients under local anesthesia that the long head of the biceps is a passive structure, sliding in its groove with movements of the shoulder[84] (Fig. 7-6). No motion was seen to be transmitted to the humeral head with long head of the biceps contractions during active elbow flexion. He believed the biceps played a minimal active role in producing motion at the shoulder joint. Rather, the humeral head was believed to passively move on the biceps tendon. This passive motion of the biceps tendon within the intertubercular groove has been found to be necessary for normal motion.[59] Biceps tendon adhesion within the bicipital groove either after injury, immobilization, inflammation, or incorporation of the tendon during tuberosity repairs was found to potentially limit the maximal recovery of motion. The shorter the intraarticular tendon length during adhesion is, the greater the motion restriction is. This was particularly evident if the adhesion occurred in internal rotation and adduction.

In contrast, by electrically stimulating the biceps muscle during shoulder arthroscopy in five cases, Andrews et al. observed that the tendon became taut and raised its origin at the superior labrum off the glenoid.[6] They also observed compression of the humeral head into the glenoid. The authors suggested that these compressive forces afford "stress protection" to the humerus during the act of throwing. They also suggested that sudden forceful transmission of contraction of the biceps on the superior labrum may be one mechanism for the development of superior labral anterior and posterior (SLAP) lesions.

Studies using simulated muscle contractions in freely hanging cadaveric specimens have been difficult to interpret because of difficulties in reproducing physiologic tension of the muscles around the shoulder. Extrapolation of the findings of these studies to the clinical situation has

been difficult. Kumar et al., in a cadaveric study of 15 dependent shoulder specimens, radiographically measured changes to the acromiohumeral interval when applying tension to the short and long heads of the biceps before and after division of the long head of the biceps.[79] There was no attempt to recreate contributions from the rotator cuff. An average decrease in the acromiohumeral interval by 15.5 mm was observed when the short head was tensed in the absence of an intact long head of the biceps. The authors suggested that a function of the long head of the biceps was to stabilize the humeral head on the glenoid during powerful elbow flexion and forearm supination by the biceps brachii. In this study, all recordings were performed with the arm by the side and the contribution of the biceps tendon in motion at the shoulder was not examined.

Flatow et al. studied possible restraints against superior humeral translation in a cadaveric model in which the long head of the biceps and rotator cuff muscle activity was simulated with a cable system.[42] Superior migration of the humeral head with abduction of the shoulder was then studied in the cuff-intact situation and with simulated tears of various sizes. Force along the biceps tendon restrained superior migration of the humeral head most significantly in the presence of a large rotator cuff defect.

Itoi et al., in two separate cadaveric studies, looked at the stabilizing function of the long head of the biceps.[67,68] In the first study, nine cadaveric specimens were tested in a hanging arm position in which the biceps was spring-loaded to simulate contractions.[68] In this model, with the biceps in the hanging arm position, anterior and posterior displacement was significantly decreased by long head of the biceps loading. Additionally, inferior displacement and external rotation was significantly decreased by long head loading. Interestingly, long head loading in this model produced small amounts of superior displacement of the shoulder. Similar to the study by Kumar et al., no attempt was made to recreate the actions of the rotator cuff. In the second study, biceps function was studied in simulated stable and unstable shoulders.[67] Again, anterior displacement of the head was significantly decreased by both the long head and short head loading when the arm was in 60 to 90 degrees of external rotation. It was concluded that the long head of the biceps and short head of the biceps have similar functions as anterior stabilizers of the glenohumeral joint when the arm is in abduction and external rotation, and that the stabilizing increases as shoulder stability decreases. Kuhn and coauthors, in studying the dynamic and ligamentous restraints to external rotation of the glenohumeral joint, also demonstrated that tension within the long head of the biceps contributed significant torsional resistance to external rotation at lower and midranges of glenohumeral abduction.[76]

Whereas previous authors have reported the long head of the biceps tendon to resist superior displacement of the humeral head, Soslowsky and coauthors found the biceps tendon to be an important stabilizer against inferior translation.[123] A cadaveric model was created looking at both the dynamic and static restraints to inferior translation of the humeral head in neutral abduction and the neutral or externally rotated positions. The influence of the magnitude of inferior translation on the stabilizing effects of the rotator cuff and biceps muscle and the glenohumeral ligaments was also studied. The long head of the biceps was found to be an important restraint against inferior translation of the humeral head independent of humeral rotation. The biceps tendon afforded more superior stability than the anterior and posterior rotator cuff and nearly equal resistance to inferior translation as the supraspinatus muscle.

A functional role for the long head of the biceps in anterior stability was also shown in a study by Rodosky et al.[116] In this study, performed in seven fresh frozen cadaveric shoulders, the biceps was tested in the context of simulated rotator cuff contractions by use of pneumonic cylinders. In this dynamic model, simulated contractions of the biceps muscle were shown to contribute to anterior stability of the glenohumeral joint by increasing the shoulder's resistance to torsional forces. This was particularly apparent in the vulnerable abducted and externally rotated position.

Pagnani, in a study of 10 cadaveric shoulders, also tested the effect of simulated contraction of the long head of the biceps on glenohumeral translation.[104] In the presence of a joint-reactive force of 22 N, a 50 N anterior, posterior, superior, and inferior force was resisted in part by contraction of the long head of the biceps. This was more pronounced at middle and lower elevation angles. The reduction in anterior translation was by 10.4 mm. There was only a 1.2-mm reduction in superior translation.

Interpretation of these cadaveric studies has been difficult because of an inability to reproduce the dynamic interplay and bulk effect of surrounding musculature. The dynamic forces implemented in most study designs have not been validated against actual in vivo muscles forces, which remain difficult to quantify. However, one in vivo analysis of the role of the long head of the biceps has been performed by Warner and McMahon.[133] In this study, performed in seven patients with isolated loss of the proximal tendon of the long head of the biceps, superior migration of the instant center of rotation of the humeral head was measured at 0, 45, 90 and 120 degrees of humeral abduction in the scapular plane. A significant increase in the superior translation was measured in comparison to the contralateral uninvolved side. The amount of the superior translation, however, was small, and the authors concluded that they may be more important in conditions where the acromial arch was more narrow, such as in patients with a type II or type III acromion.

Electromyographic Analysis Studies

Interpretation of cadaveric studies of the long head of the biceps muscle requires relevant electromyographic analysis

to predict the quality of applied loads. A consistent but potentially inaccurate assumption with all of these studies has been the inclusion of significant biceps muscle activity associated with shoulder motion. Multiple studies have attempted to evaluate biceps muscle function during shoulder motion through the use of electromyographic analysis. Electromyographic analysis has a significant advantage over cadaveric studies by allowing for an in vivo observation of potential long head of the biceps function.

With the use of electromyographic data gathering, some studies have suggested a biceps role in shoulder stabilization. Ting et al., in a study of five patients with unilateral rotator cuff tears, showed increased biceps activity on the ipsilateral side in comparison to contralateral controls.[128] Additionally, Jobe et al., in a study of baseball pitchers with symptoms consistent with instability, showed increased biceps activity in comparison to stable controls.[69] In a similar study, Basmajian and Latif noted activity in the long head of the biceps upon shoulder abduction only with an externally rotated and supinated forearm.[7] They concluded that the primary action of the biceps was at the elbow for flexion of the supinated forearm, the activity in the tendon diminishing with forearm pronation. The biceps tendon was also active with forearm supination with the elbow flexed or supination against resistance. The actions of the long and short heads of the biceps were similar, but the long head was generally more active.

Some electromyographic (EMG) studies of healthy subjects have shown significant activity of the biceps muscle with various shoulder tasks. Habermeyer et al. observed EMG activity in the biceps with shoulder abduction from 90 to 166 degrees, peaking at 132 degrees.[54] Upon flexion of the shoulder, EMG activity was recorded in the biceps from 0 to 164 degrees, peaking at 84 degrees. Lesser activity was recorded with other shoulder movements, with no activity observed in internal rotation. Goro and coauthors studied the EMG activity of the long and short heads of the biceps during various ranges of shoulder flexion and abduction combined with humeral rotation.[52] The forearm was braced in neutral rotation but the elbow was left free. Twenty-four different arm positions were analyzed while the healthy subjects generated 30% maximal voluntary contractions. Both heads of the biceps were found to be active during flexion and abduction tasks, with relatively greater activity at higher ranges of elevation (45 vs. 135 degrees reached statistical significance). In addition, increased biceps activity was seen when external rotation was combined with elevation in either plane.

In contrast, several EMG studies have shown the biceps to be active only during elbow motion. Furlani, in 30 healthy shoulders, showed the biceps to be completely inactive with abduction and elbow extension.[44] Only one subject showed slight activity in abduction with elbow flexion. When the shoulder was abducted against a significant load, only 3 of the 30 subjects showed some activity, and two of these subjects were considered "tense." Pauly et al., in a study of 18 volunteers, showed no significant biceps activity with shoulder abduction or rotation in any subject.[105] Gowan et al., in an EMG study of the shoulder during pitching, showed the biceps acting primarily in late cocking.[53] Moderate activity in the biceps was noted with elbow flexion in the cocking phase, reaching an intensity of about 34% of a maximal manual muscle test. With the elbow position relatively stable during the acceleration phase, the intensity of biceps action diminished to less than 25%. Peak activity in the biceps tendon was recorded during the follow-through phase, the muscle contracting to decelerate the rapidly extending elbow. Based on their findings, they concluded that the biceps acts in concert with the brachialis and triceps to control the elbow with no significant effect at the shoulder during the throwing action.

As the biceps muscle traverses two joints, interpretation of previous EMG studies has been difficult because no attempt was made to control for elbow-related actions. Yamaguchi et al., in a study of 44 shoulders, including 14 with rotator cuff tears, showed no significant shoulder-related activity of the biceps muscle when elbow function was controlled for with the use of a brace[137] (Fig. 7-7). Biceps levels remained relatively flat throughout various shoulder motions and were insignificant compared to supraspinatus levels, which increased in a motion-specific fashion. Brachioradialis controls were similarly minimal and mirrored biceps activity patterns, suggesting that any biceps activity was either background or elbow-related. No significant increase was seen in patients with rotator cuff tears. In contrast, supraspinatus activity levels showed significant increase in the presence of rotator cuff tears. The results of this study were further substantiated by Levy et al. in a study of 10 shoulders controlled for elbow motion.[82] In this study, fast and slow motions as well as loaded and unloaded shoulder motions were tested for the presence or absence of biceps activity. As previously reported by Yamaguchi et al., no significant electro-activity was identified in the long head of the biceps in response to isolated shoulder motion when elbow and forearm position were controlled. The authors of both of these studies concluded that any function attributed to the long head of the biceps was not likely to be active. Rather, the long head of the biceps had either a passive role or an active role dependent on an association with elbow and forearm activity.[68]

Summary of Functional Anatomy

To date, the exact function of the long head of the biceps tendon in shoulder function remains incompletely characterized. Whereas the comparative anatomy analysis and electromyographic observations would suggest a minimal role for the biceps tendon, the biomechanical data to date would suggest otherwise. However, electromyographic data with controlled elbow motion would suggest that very little

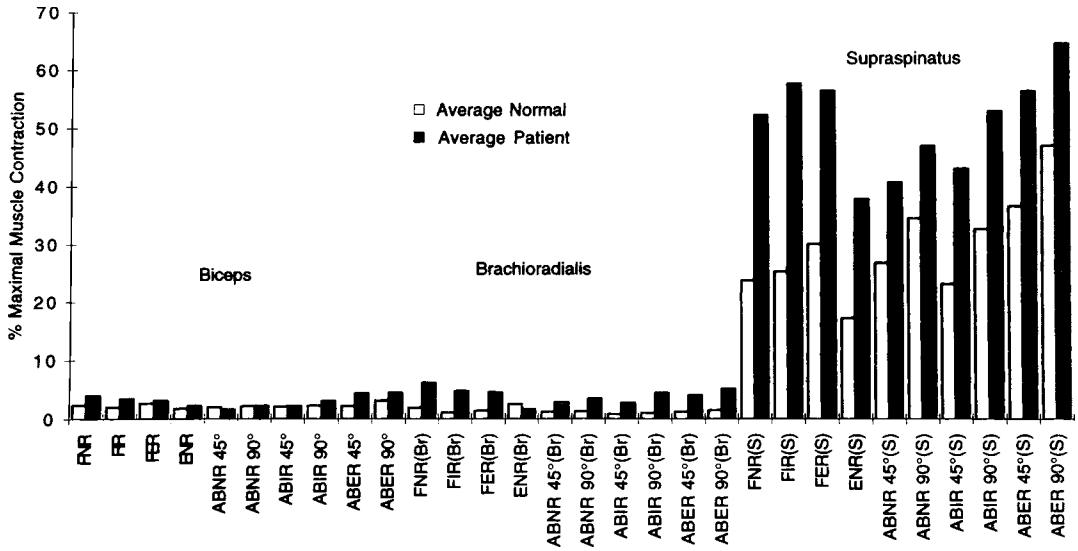


Figure 7-7 Column graph showing the combined electromyographic activity from the biceps, brachioradialis, and supraspinatus in normal control subjects and patients with rotator cuff tears. When elbow activity was controlled, minimal biceps and brachioradialis activity was achieved, which was shown for both normal and rotator cuff tear patients. In contrast, significant amounts of supraspinatus activity were seen and the amounts increased when a rotator cuff tear was present. The results suggest a lack of biceps activity coordinated with shoulder motion. y axis = electromyographic activity as a percent of maximal muscle contraction. x axis = various types of active shoulder motion. FNR, flexion, neutralrotation; FIR, flexion, internal rotation; FER, flexion, external rotation; ENR, extension, neutral rotation, ABNR, abduction, neutral rotation; ABER, abduction, external rotation; ABIR, abduction, internal rotation.

coordinated biceps activity occurs specific to the shoulder joint.[82,137] Previous studies using biceps tendon loads in cadaveric biomechanical analysis represent estimations at best. Even in complex activities such as pitching, the axial muscle contractions noted in the biceps muscle have been about 30% to 40% of those attainable.[53] Given vector analysis of the pull of the biceps, a head depression role would be unlikely to occur in most ranges of motion short of full external rotation. This does not preclude a role in anterior instability, which appears to be better supported by both the electromyographic and biomechanical analysis. In the context of anterior instability, the biceps has been shown to play a significant role in dynamic cadaveric models and in vivo where an increased EMG response was seen in unstable pitchers versus those without stability symptoms.

The absence of supportive evidence on a coordinated active role of the biceps does not preclude a significant passive role or active role secondary to elbow activity. It is possible that resting tension on the biceps may be helpful in both anterior and superior instability. Additionally, proprioceptive roles for the biceps tendon remain to be studied.

PATHOPHYSIOLOGY

Shoulder pain arising solely from the long head of the biceps can be quite severe, causing marked loss of shoulder motion. Disorders of the biceps tendon may arise from inflammatory changes in and around the tendon or may develop as a consequence of a significant injury or repeated microtrauma. These two major groups of pathologic processes (i.e., inflammatory and traumatic) are a result of the location of the tendon and its unique anatomic structure.[22,95–97,101,114] While the eventual clinical presentation of these afflictions of the long head of the biceps tendon is shoulder pain, they affect different patient populations with dissimilar pathogeneses. In this section, we will discuss the various disorders of the long head of the biceps tendon by classifying them as "inflammatory," "instability," or "traumatic" on the basis of the original initiating event. It must be stressed that the distinction is not always clear; the degenerate inflamed tendon is more prone to trauma and, conversely, repeated trauma may result in changes in the tendon indistinguishable from those of inflammation. Nevertheless, this classification can help with the organization of the pathogenesis of these disorders and formulation of protocols for appropriate management.

Biceps Tendinitis Concurrent with Rotator Cuff Disease

Tendinitis of the long head of the biceps was described by Neer to be secondary to impingement syndrome for the majority of cases encountered in clinical practice.[95] As the sheath of the biceps tendon is an extension of the synovial

lining of the glenohumeral joint and intimately related to the rotator cuff, any inflammatory process affecting one of the structures can eventually affect the others as well.[21,56,94,96–98,100,101] The terms *subacromial bursitis, rotator cuff tendinitis,* and *impingement syndrome* have been used to describe the inflammatory changes occurring in the subacromial space as a result of the impingement syndrome. This and other forms of bursitis such as rheumatoid arthritis, gout, and other crystal arthropathies and infections usually involve intraarticular synovitis.

In addition to a secondary involvement of the surrounding synovium, the long head of the biceps tendon is susceptible to the same mechanical abutment seen with impingement of the rotator cuff tendons.[22,95,101] Whether primary or secondary causes of impingement exist, the long head of the biceps occupies an anterior location within the impingement zone, which predisposes its involvement with rotator cuff disease. Besides these compressive forces, the biceps tendon is further subjected to a medial displacing force across the lesser tuberosity as it moves in the groove.[107,119]

The mechanical effects on the rotator cuff and long head of the biceps tendon appear to be age-related and tend to occur together.

Petersson performed 151 shoulder dissections in 76 cadavers.[106] No degenerative changes were observed before the age of 60. The long head of the biceps demonstrated fraying and flattening in 12 shoulders and was found ruptured in six. Four shoulders demonstrated dislocation of the tendon out of its groove. The number of shoulders with degenerative changes in the biceps tendon seemed to increase with increasing age, involving five of six cases over the age of 90.

Murthi et al. performed a prospective arthroscopic evaluation of the biceps tendon in 200 patients requiring surgery for chronic rotator cuff disease.[94] Soft tissue tenodesis was performed in 40% of the surgeries secondary to macroscopic abnormalities of the biceps tendon. The remaining 120 patients underwent open tenosynovectomy of the biceps sheath and rotator cuff repair when indicated. Synovial biopsies were collected and direct examination of the intertubercular groove portion of the biceps tendon was performed in all cases. Sixty-three percent of the specimens revealed histologic evidence of chronic inflammation, while only 18% were free of disease. Normal biceps tendons were found in only 25% of cases with no rotator cuff tear, 16% of cases with partial tear, and 11% of those with full-thickness rotator cuff tears. Thirteen percent had fibrosis of the biceps tendon. Thus, there appeared to be a strong association of coexistent biceps tendon disease with rotator cuff disease. This association increased with increasing severity of the rotator cuff disease. In the tenodesis group, only 49% of cases with gross evidence of degeneration were seen arthroscopically, even when the tendon was pulled into the joint with a probe. The remaining cases demonstrated abnormalities in the biceps tendon distal to the transverse humeral ligament.

Synovitis of the biceps tendon, likened to deQuervain's stenosing tenosynovitis, is generally found in the segment within the bicipital groove, under the transverse humeral ligament[97] (Fig. 7-8). Surgical exploration can reveal the synovial sheath of the tendon as hemorrhagic and bulging slightly above and below the transverse humeral ligament. Upon incision of the outer layer of the synovial sheath, an effusion can escape into the wound. Not present normally between the two layers of the synovial sheath around the tendon, this fluid represents the inflammatory exudate. The contained segment of the tendon will appear dull, swollen, and discolored but is still mobile in the groove. These findings correspond to the acute painful stage of the disease.[84] In later cases, the sheath appears thickened, fibrotic, and less vascular. The tendon is roughened and lies in a bed of hemorrhagic adhesions. The inflamed tendon can appear reddened but initially normal in size. In later stages, the tendon may appear atrophic or hypertrophic. The atrophic tendon is thin and frayed and represents a prerupture stage. In the "hypertrophic" type, inflammatory changes within the tendon result in an enlarged appearance. Boileau et al. described intraarticular entrapment of a hypertrophic long head of biceps tendon coined "the hourglass biceps."[17] A series of 21 cases were reported, all but one associated with a rotator cuff tear. Each patient reported anterior shoulder pain and loss of active and passive terminal elevation motion. Each subject was noted at the time of surgery to have a hypertrophic tendon that became mechanically blocked from entrance into the biceps groove with flexion of the shoulder.

Microscopically, the inflammatory changes in the tendon have been well documented. There is a pronounced round-cell infiltration of the tendon, degeneration of the tendon fibers, and edema.[84,94] The hypertrophied tendon seen in relation to large cuff tears most plausibly represents a chronic inflammatory response from continuing impingement of the tendon.[137] Eventually, the tendon can become firmly bound down in the groove with adhesions or spontaneously rupture. This can be associated with resolution of the symptoms of tendinitis. Rupture of the intraarticular portion of the tendon has been reported in 3% of anatomic dissections.[84] These changes appear age-related, with more pronounced changes occurring in patients past middle life.[33] When spontaneous rupture is seen, it is often accompanied with an instant and gratifying relief of the long-standing shoulder pain. Perhaps no other clinical observation is more supportive of the significant role biceps tendinitis can play in shoulder pain.

Primary Bicipital Tendinitis

The term *primary bicipital tendinitis* has been reserved for isolated inflammation of the long head of the biceps tendon in the intertubercular groove without any evidence of associated shoulder pathology. Most authors believe that

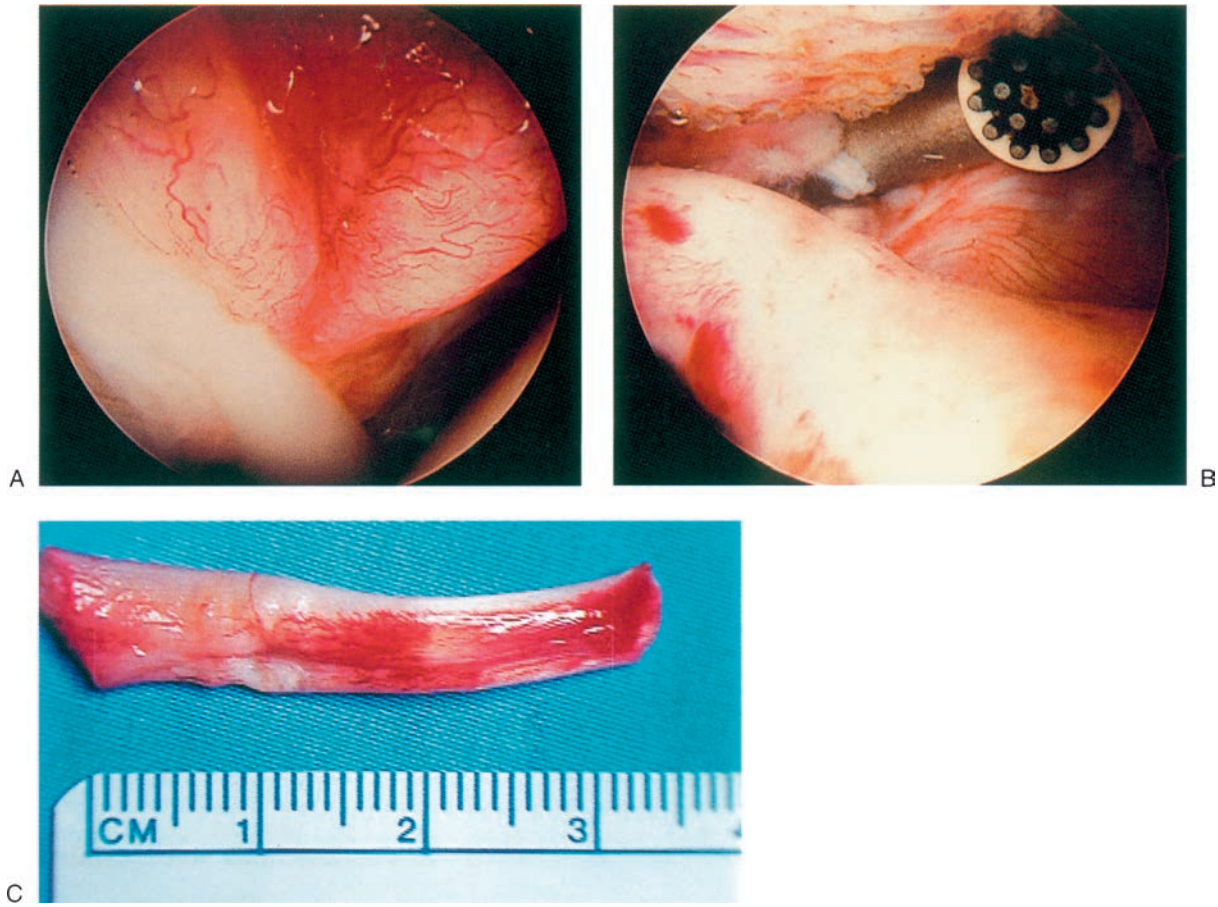


Figure 7-8 Intraoperative photos showing the marked synovitis that could be seen on the undersurface of the rotator cuff with chronic rotator cuff tendinitis. **A.** Synovitis on the undersurface of the rotator cuff is encircling the biceps tendon during its intraarticular path. **B.** Involvement of synovitis at the biceps origin. **C.** Removed tendon obtained from a patient who had failed a previous decompression for chronic rotator cuff tendinitis. Following this revision procedure, in which this tendon was removed, the patient had a resolution of symptoms.

primary tendinitis of the biceps tendon is uncommon and must be diagnosed only after exclusion of rotator cuff pathology or subacromial impingement.[34,95]

Tendinitis may be a result of direct or indirect trauma after increased activity or an underlying inflammatory disease, as a result of trauma, or secondary to instability of the tendon.[110] Often no specific factor is identifiable, constituting the idiopathic group, which in some series accounts for 43% of cases.[31] Whatever the inciting factor, the pathologic changes are restricted to the intertubercular groove and are indistinguishable from secondary tendinitis as mentioned above. However, primary tendinitis almost surely exists as a separate entity also. This bicipital tenosynovitis has been likened to that seen in deQuervain's tenosynovitis.[31,33,84,90,110] The thickening of synovitis of the tendon occurs under the transverse humeral ligament in the bicipital groove. The intraarticular portion of the tendon has been reported to always be normal.

The causes for biceps tendonitis can be multifactorial. DePalma described anomalies of the bicipital groove, together with repeated trauma, as a major factor in younger people.[33] Degenerative changes were described to be the more common factors in older people. Rathbun and Macnab, in a microvascular injection study of the shoulder, showed a critical zone in the long head of the biceps tendon similar to that seen in the supraspinatus.[113] However, this critical zone of avascularity was primarily in the intracapsular portion of the tendon.

Long Head of the Biceps Tendon Instability

The spectrum of instability of the biceps tendon varies from subluxation, which is excessive mobility of the tendon within the groove, to dislocation or complete displacement of the tendon out of its groove in the humerus.

We have used the term *primary instability* to include the spectrum of biceps instabilities from subluxation to frank

dislocation. The biceps tendon passes at an angle of 30 to 40 degrees from its origin to the groove in the humerus where it descends vertically. The tendon is relatively fixed from the point of its entrance into the bicipital groove and swings from one angle to another as the arm is rotated. When the arm is cocked to abduction and external rotation, the forces tend to displace the tendon medially, compressing it against the medial edge of the bicipital groove. In internal rotation, the tendon is thrust against the lateral margin of the groove, moving through an arc of more than 90 degrees in the process.[102] If the groove is shallow, the tendon may force its way over the greater or lesser tuberosity. If the groove is narrow and tight, the constant pressure on the tendon may lead to tendinitis.

Luxation of the long head of the biceps tendon is most commonly secondary to loss of the soft tissue restraints with degenerative rotator cuff tears.[21,56,107,131,132] It is generally agreed that the main restraint to medial dislocation of the tendon of the long head of the biceps is the integrity of the rotator interval, which can be disrupted with rotator cuff tears that involve either the superior aspect of the subscapularis or the anterior portion of the supraspinatus tendon.[21,26,27,56,61,131,132] Displacement of the biceps tendon out of its groove, in association with tears of the rotator cuff, has been reported with incidences as high as 20% of all cuff tears.[119] The tendon is found to lie medial to the lesser tuberosity, over the subscapularis, covered by a sling of the subscapularis tendon. In a cadaveric study of 153 shoulders, Petersson found two patterns of medial dislocation of the long head of the biceps tendon: the dislocated tendon sliding over the subscapularis and a dislocated tendon sliding beneath a deep tear of the subscapularis.[107] The latter pattern occurred in four of the five cases in Petersson's series. In cases with full-thickness tears of the supraspinatus tendon with rupture of the coracohumeral ligament, the tendon can displace medially over the subscapularis tendon. In the presence of a partial tear of the subscapularis tendon involving its deep surface, however, the tendon can sublux medially deep to the subscapularis muscle. Upon external inspection, the subscapularis tendon can appear intact and a dislocated tendon may be missed if the region is not carefully examined at surgery. Walch et al. described the presence of "hidden" lesions of the rotator interval in 19 of 116 cases with rotator cuff tears.[132] In these cases, exploration of the rotator interval showed associated tearing of the superior glenohumeral ligament, coracohumeral ligament, and superior portion of subscapularis. The biceps tendon was ruptured in two cases, subluxated in 14, and normal in five.

Habermeyer and Walch showed that 50% of all biceps subluxations were associated with degenerative changes in the anterosuperior aspect of the labrum, suggesting a correlation between these structural changes.[56] Gerber and Sebesta introduced a pathologic process termed anterosuperior impingement (ASI) as a distinct entity producing lesions of the biceps reflection pulley.[48] Impingement of the reflection pulley, biceps tendon, and uppermost portion of the subscapularis tendon against the anterosuperior labrum and glenoid was observed in a series of 16 patients with chronic anterior shoulder pain (the majority of which were engaged in manual labor) when the shoulder was flexed to 120 degrees in combination with horizontal adduction and internal rotation. Three cases were noted to have isolated lesions of the common insertion of the superior glenohumeral and coracohumeral ligaments (pulley lesion), 10 cases had pulley lesions and articular-side partial disruption of the subscapularis, and three cases had an intact pulley system. Pulley lesions alone or in combination with subscapularis rupture were usually associated with biceps tendon pathology manifested as degeneration, instability, or the presence of SLAP tears. Habermeyer and coauthors further supported the concept of anterosuperior impingement as a cause of biceps tendon degeneration and instability.[55] Eighty-nine patients with arthroscopically proven pulley lesions were studied, excluding all cases with full-thickness tears of the rotator cuff. Four patterns of injury to the superior glenohumeral ligament (pulley system) and subscapularis and supraspinatus tendons were identified and correlated with the intraoperative presence of ASI. Overall, abnormalities within the biceps tendon were seen in 90% of patients ranging from synovitis to frank dislocation. The presence of a pulley lesion combined with an articular-side subscapularis injury was correlated with the presence of ASI (seen in 59% of cases). The presence of a partial articular-side supraspinatus lesion and a partial subscapularis tear increased the risk of ASI to 75% compared to 26% of patients with a pulley lesion alone.

Contrary to common perceptions about subluxation, the biceps tendon does not intermittently reduce with arm motion. Rather, these luxations appear to be fixed. Subluxation has been classified by Habermeyer and Walch into three types[56] (Fig. 7-9).

Type I, a superior subluxation, comes as a consequence of a loss of the coracohumeral ligament and rotator interval sling. The subscapularis tendon is intact, preventing an otherwise true dislocation. There is often a partial lesion of the supraspinatus lesion. In type II subluxation, the biceps tendon is unstable at the entrance to the bony groove. In this situation, the tendon slips over the medial rim of the lesser tuberosity short of complete dislocation. The pathologic lesion here is a detachment of the superiormost fibers of the subscapularis tendon. Type III subluxations follow a malunion or nonunion lesser tuberosity. In this situation, a fracture of the lesser tuberosity without healing or any malunited fashion compromises the medial bony restraint to the long head of the biceps tendon allowing subluxation. Symptoms are more prominent in internal rotation. Each of these types of subluxation is associated with tendinitis and capsular synovitis in the area of the rotator interval. Chronic cases of this tendonitis can lead to attrition and later rupture of the long head of the biceps tendon.

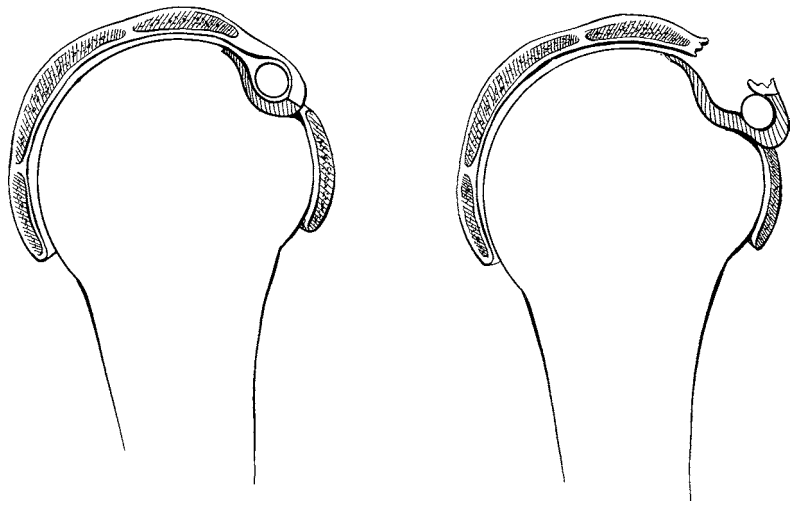


Figure 7-9 Schematic showing the anatomic basis of biceps subluxations. Subluxation can occur as a consequence of the loss of the coracohumeral ligament (*hatched*) and superior glenohumeral ligament sling (*black*). An intact subscapularis acts to prevent an otherwise complete dislocation. In type I lesions there is only a superior luxation from a loss of the interval sling. In type II subluxations, the biceps tendon is more unstable distally secondary to a tear of the superiormost fibers of the subscapularis tendon.

Frank dislocation of the long head of the biceps is nearly always associated with a tear of the subscapularis tendon. Thus, observation of a completely dislocated biceps tendon either by preoperative imaging such as magnetic resonance imaging or ultrasonography or during direct observation arthroscopically should alert the treating physician to the possibility of a concurrent full-thickness rupture of the subscapularis tendon. Habermeyer and Walch have classified these types of dislocations into type I and type II lesions.[56] Type I lesions are extraarticular dislocations combined with a partial tear of the subscapularis tendon. In this situation, the long head of the biceps tendon is completely dislocated over the lesser tuberosity. There is a rupture of the common attachment of the superior glenohumeral ligament and coracohumeral ligament. Superficial and lateral fibers of the subscapularis tendon are also torn, releasing the soft tissue restraints to the tendon. However, deep fibers of the subscapularis tendon remain intact, preventing intraarticular displacement. The lesion essentially corresponds to a more advanced evolution of a type II subluxation. In type II dislocations of the biceps tendon, there is an intraarticular dislocation of the long head of the biceps tendon. This occurs in conjunction with a complete full-thickness tear of the subscapularis. This type of dislocation is associated with an extensive tearing of the tendinous portion of the subscapularis tendon and approximately half of these have a traumatic cause.

The majority of cases of biceps tendon instability are associated with tears of both the subscapularis and supraspinatus tendons with the pattern of instability dictated by the severity of the subscapularis and biceps pulley injuries. Walch et al. reported a series of 71 cases of subluxation or dislocation of the biceps tendon describing the detailed anatomy of associated rotator cuff tears.[131] Of 445 rotator cuff tears treated over a 7-year period, biceps instability was seen in 16% of patients. All cases of biceps subluxation were associated with partial injury of the upper portion of the subscapularis tendon with concomitant abnormalities of the ligamentous pulley. An associated lesion of the supraspinatus tendon was noted in 70% of these cases. Frank dislocation of the biceps tendon was demonstrated in 46 patients (65% of cases) and was seen in two patterns: extraarticular and intraarticular. With extraarticular dislocations, deep fibers of the subscapularis and middle glenohumeral ligament remained partially intact, separating the tendon from the glenohumeral joint. Intraarticular dislocations of the biceps tendon were seen with complete detachment of the subscapularis tendon and incompetence of the biceps pulley. Seventy percent of cases of a dislocated biceps tendon were seen in the context of full-thickness tears of the supraspinatus and infraspinatus tendons.

Traumatic Rupture of the Long Head of the Biceps Tendon

Traumatic rupture of a normal long head of the biceps tendon is extremely uncommon. When long head of the biceps tendon ruptures are seen in the context of trauma, they are generally in the context of a previously degenerated tendon. Often the trauma can be relatively minor in these circumstances and is usually preceded by history consistent with rotator cuff tendinitis. However, isolated ruptures of the long head of the biceps can occur in the absence of previous history of subacromial impingement.[33,88,90,110,118] These can be seen in the context of significant trauma involving either a powerful supination force, powerful deceleration of the forearm during pitching, or fall on an outstretched arm as in SLAP lesions.[6,120–122] Type IV SLAP lesions, which include an extension of the labral tear into the intratendinous substance of the biceps, are an example of primary partial ruptures of the biceps tendon from trauma.

When partial tearing of the biceps tendon occurs, often significant pain and dysfunction are associated with the lesion. A higher suspicion of a partial-thickness traumatic tear of the biceps tendon again should be considered in the context of previous tendonitis or bicipital-like pain. In contrast, full-thickness traumatic ruptures of the biceps tendon are generally less consequential from a symptomatic point of view.[23,56,134] In these cases, the patient may have experienced significant pain in the upper and anterior brachium. This is often associated with bruising down the biceps muscle. After a period of discomfort, the pain generally subsides and the patients are generally without significant consequence to the shoulder function.

Tears of the Superior Labrum at the Origin of the Biceps Tendon

While tendinitis of the biceps tendon has been long recognized as a cause of shoulder pain, symptomatic tears of the superior labrum at the origin of the biceps tendon has only recently been established as a pathologic entity. First described in athletes by Andrews et al., the lesion has been further characterized and classified by Snyder et al., who are also responsible for attaching the acronym SLAP to these findings.[6,120–122]

While both reports included patients with tears of the superior labrum at the origin of the biceps tendon diagnosed arthroscopically, the patient populations seem to differ. Andrews et al. retrospectively identified 73 throwing athletes (51 baseball players) with no history of a single episode of significant trauma and a mean age of 23 years.[6] In Snyder et al.'s series, the 27 patients with superior labral lesions represented about 4% of the total arthroscopic procedures performed over a 4-year period.[120] With a mean age of 37.5 years, the commonest mechanism of injury (48%) was compression injury resulting from a fall on the outstretched hand. The remainder seemed to arise from traction injuries either as a sudden pull on the arm (22%) or repetitive traction with overhead throwing activities (8%). The mechanism was unclear in the remaining 22% with no history of the above.

Both of these populations, although different in presentation, most likely represent a spectrum of the same pathology, one arising from the repetitive trauma of overhead activities, the latter from a single traumatic episode.

It is postulated that a fall on the outstretched hand with the shoulder abducted and in slight forward flexion provides a proximal subluxating force resulting in direct compression at the superior labrum and biceps origin. An additional traction force may be provided by the reflex contraction of the biceps during the fall. Clavert and coauthors simulated forward and backward falls in a cadaver model to examine the likelihood of creating a SLAP lesion.[28] The humeral head was impacted against the glenoid with 1500 N in two of the test positions after preloading the rotator cuff and biceps tendons. A type II SLAP tear was reproduced in all five specimens simulating a forward fall and in two of the five specimens simulating a backward fall. The authors concluded that the compressive and shearing forces recreated during a fall on the outstretched hand is a plausible cause for type II SLAP tears.

Repeated forces transmitted through the origin of the long head of the biceps may account for the detachment of the labrum in throwing athletes. Several cadaveric studies have shown both the late cocking and deceleration phases of throwing to place high strains on superior labrum and biceps tendon. Kuhn et al. compared the late cocking and early deceleration phases of throwing in 20 cadaveric shoulders while the long head of biceps tendon was loaded to failure.[77] The early cocking test position was 60 degrees of scapular plane abduction combined with more than 125 degrees of external rotation, whereas the early deceleration position was set at 80 degrees of external rotation, 60 degrees of abduction, and 16 degrees of horizontal adduction. Baseline forces of 22 N were recreated within the rotator cuff tendons prior to biceps loading. Failure of the biceps anchor occurred at the biceps superior labral complex in 9 of 10 specimens in the late cocking position compared to 2 of 10 specimens in the early deceleration position. Five type II SLAP tears were created, four of which occurred in the late cocking position. In addition, the load to failure of the late cocking position (289 $\pm$ 39 N) was significantly less than the early deceleration position (346 $\pm$ 40 N). Pradham and coauthors investigated the strains on the anterior and posterior aspects of the superior labrum during various stages of throwing.[111] Predetermined loads based on the percent of maximum voluntary contraction of the biceps tendon during each phase of throwing from a prior EMG study were recreated. A 22-N force was simulated in the subscapularis and infraspinatus tendons during testing. The strain on the posterior and anterior portions of the superior labrum increased significantly in the late cocking position compared to the early cocking, acceleration, deceleration, and follow-through positions.

Yeh and coauthors used a finite element model to analyze the forces on the superior labrum–glenoid interface in four phases of throwing: early cocking, late cocking, acceleration, and deceleration.[138] Three types of biceps origin geometries were investigated to determine their influence in the stress distribution on the glenoid. Force transmission through the biceps tendon was determined based on the estimated biceps activity in the various stages of throwing. Rotator cuff forces were not simulated in this model. The stress magnitudes at the labrum–glenoid interface were highest in the deceleration phase of throwing for all types of biceps origins. In addition, an anterior biceps origin produced 50% higher strains on the labrum–glenoid

interface compared to a posterior biceps origin in the early deceleration phase of throwing.

Traumatic disruption of the superior labrum can occur to varying extents and has been classified into four types based on the labral injury and the stability of the labrum–biceps complex found at arthroscopy[122] (Fig. 7-10):

Type I: This lesion represents the mildest of the spectrum. The superior labrum appears degenerate and frayed (Fig. 7-10E). The attachment of the labrum and long head of the biceps tendon are preserved and stable upon probing. The long head of the biceps has a normal appearance. In an older-aged population this finding may be asymptomatic and associated with the normal age-related degenerative process.

Type II: The frayed superior labrum and the attached tendon of the long head of the biceps are stripped off the underlying glenoid in association with avulsion of the superior glenohumeral ligament complex. The superior labrum arches away from the glenoid and can be lifted off with a probe (Fig. 7-10F). The peripheral detachment of the labrum and the degenerative tissues seen at the attachment of the labrum to the superior glenoid seen in type II lesions differentiate these from the normal loose attachment of the superior labrum in its central portion.

Type III: In type III lesions, the superior labrum develops a tear with a stable labrum–biceps complex, similar to the "bucket-handle" tear of the knee meniscus (Fig. 7-10G). The origin of the long head of the biceps and the superior glenohumeral ligament complex retain their attachment to the glenoid, differentiating a type III from a type II lesion. The torn labrum devoid of any peripheral attachment can be displaced into the glenohumeral joint.

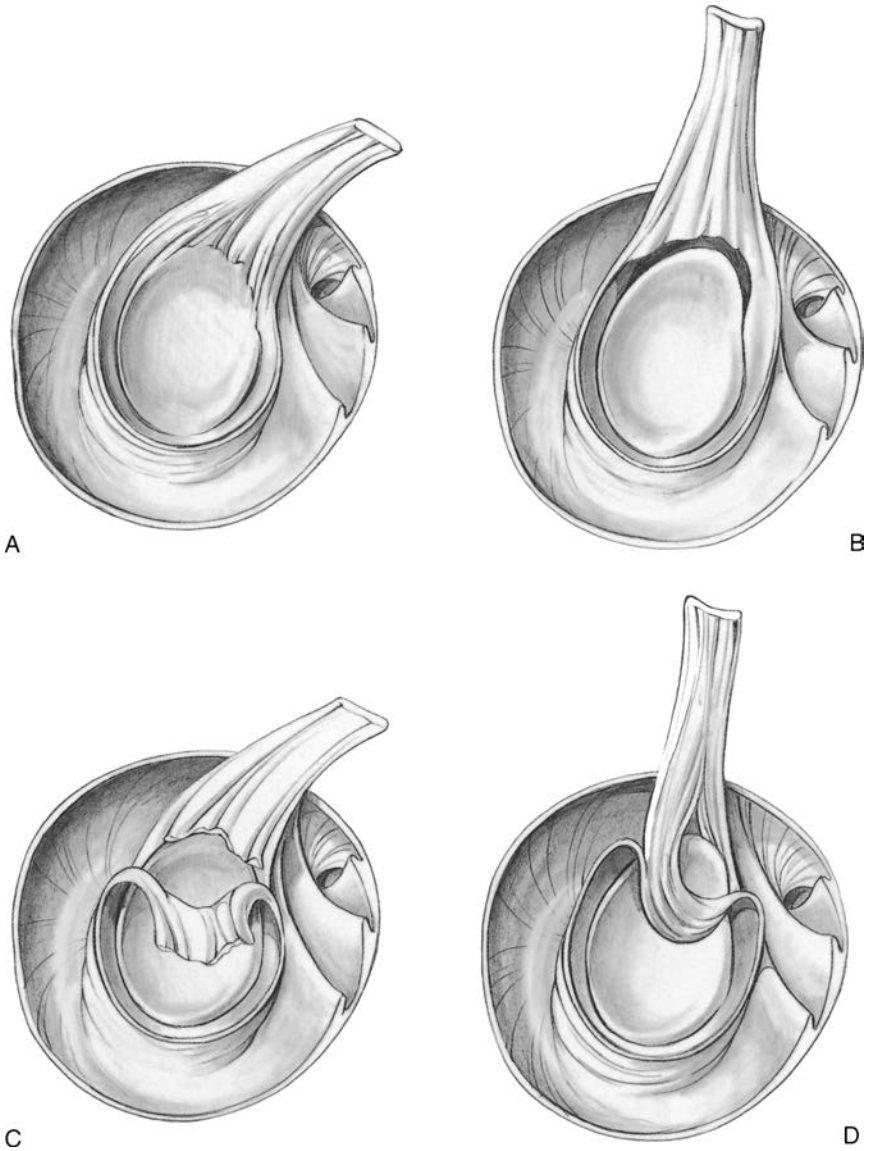
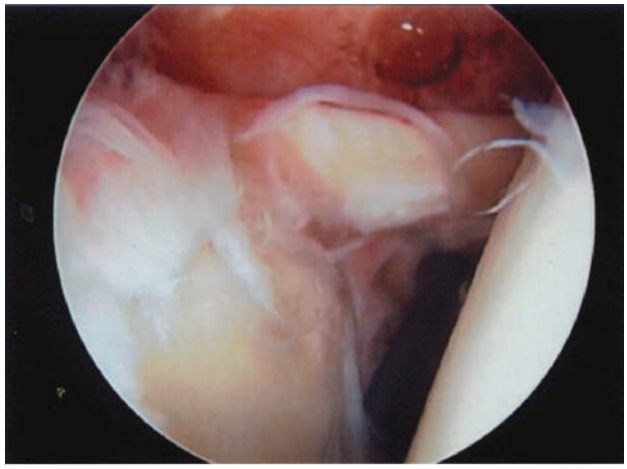
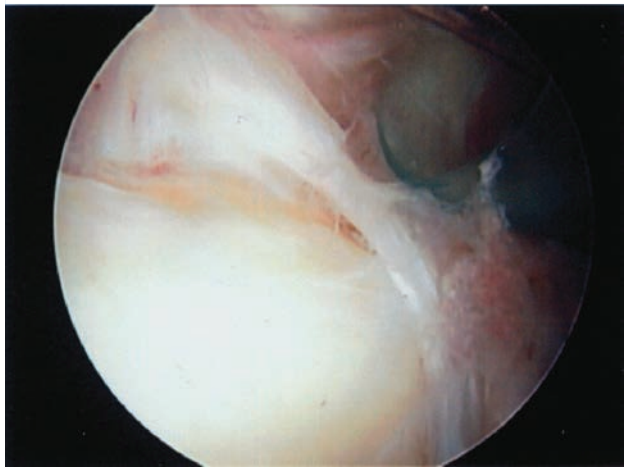


Figure 7-10 Schematic of the four types of superior labral anterior and posterior (SLAP) lesions. **A.** Type I SLAP lesions are characterized by a significant fraying or degeneration of the superior labrum. **B.** Type II SLAP lesions are characterized by detachment of the superior labrum and biceps tendon from the glenoid rim. **C.** Type III SLAP lesions are seen as a bucket-handle tearing of the superior labrum. The remaining labral tissue maintains the biceps as anchored to the glenoid rim. **D.** Type IV SLAP lesions consist of an extension of the bucket-handle labral tear into the substance of the biceps tendon. Arthroscopic views: **E.** SLAP I lesion; **F.** SLAP II lesion; **G.** SLAP III lesion; **H.** SLAP IV lesion.





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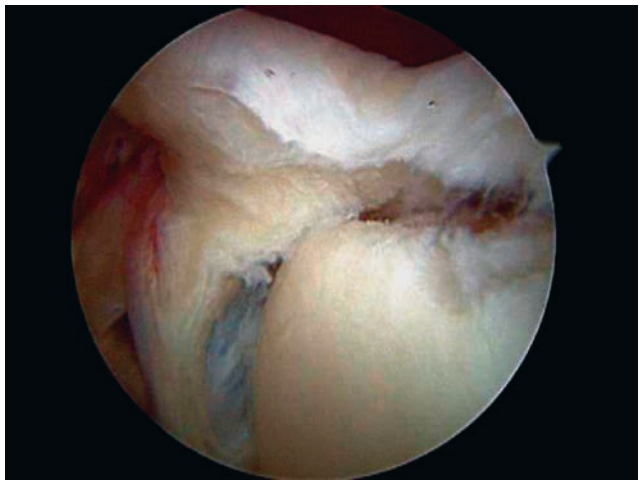

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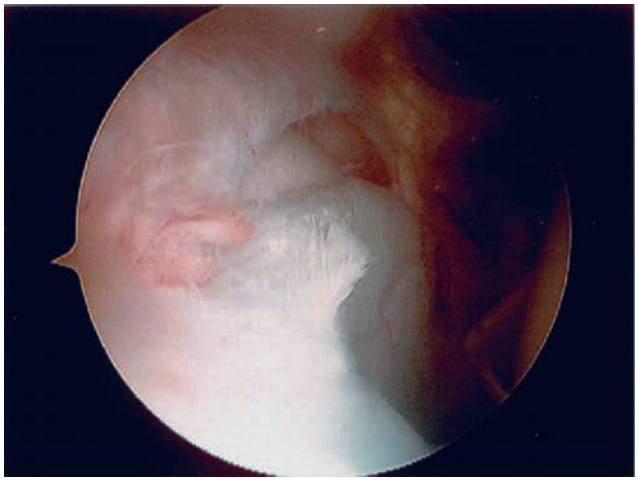

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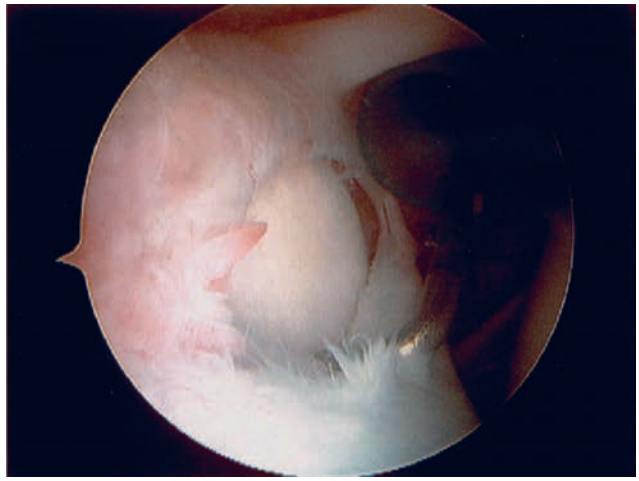
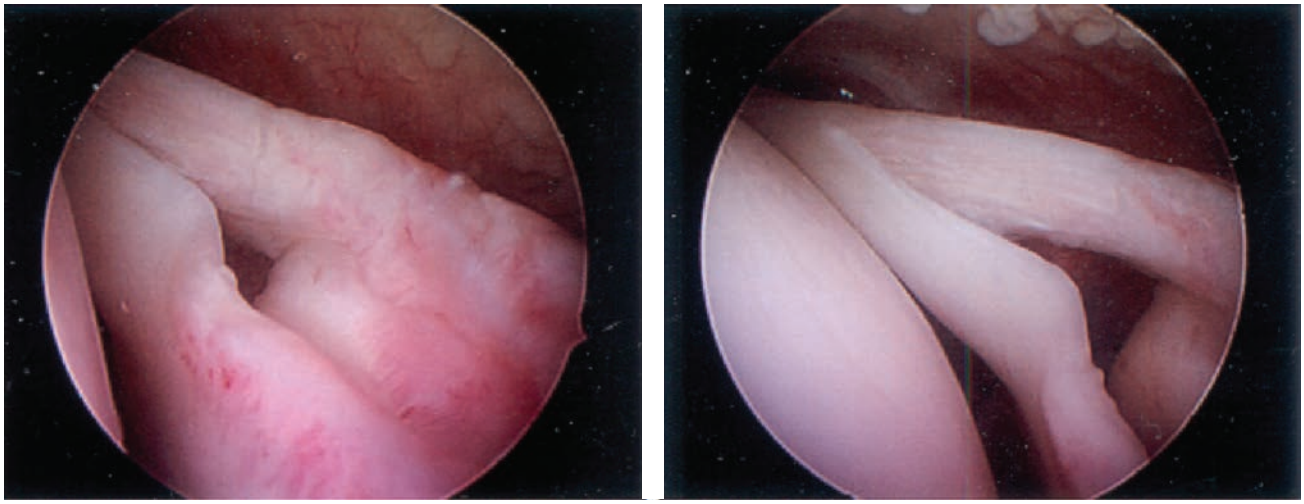


Figure 7-10 (*continued*)

Type IV: The central tear includes a portion of the biceps tendon (Fig. 7-10H). The biceps tendon is thus split longitudinally into an intact peripheral portion and a central portion displaced into the joint with the attached bucket-handle tear of the labrum.

Type II and type III lesions were the commonest in Snyder et al.'s series accounting for 41% and 33% of the cases, respectively. While type IV lesions were observed in 15%, type I lesions were the least common, accounting for 11% of the series.



H

Figure 7-10 (*continued*)

The original classification of SLAP tears by Snyder et al. has been expanded to include superior labral tears associated with glenohumeral instability. Maffet et al. reported a series of 84 patients with arthroscopically proven SLAP tears.[85] Thirty-two of the 84 patients (36%) were noted to have associated labral and/or capsular pathology that precluded standard classification. The majority of these tears were seen in patients with associated glenohumeral instability. The authors further classified these SLAP lesions into three additional categories. Type V SLAP tears involved detachment of the superior labrum and biceps anchor with propagation of the tear into the anteroinferior labral tear (Bankart lesion). Type VI SLAP tears were seen as displaced flap tears of the superior labrum with an intact biceps root. Type VII SLAP tear included a detached superior labrum associated with a distinct capsular tear.

CLINICAL EVALUATION

History

As with all other orthopedic conditions, an accurate and directed history and physical examination are basic and essential parts of the clinical evaluation. This is particularly true in the case of biceps tendon disorders as imaging studies are often nondiagnostic.

Patients with biceps tendinitis or pain from subluxation always have pain at the bicipital groove.[21,56,96,98] This pain, however, can be difficult to distinguish from anterior subdeltoid pain from impingement syndrome or rotator cuff tendinitis. In most people, the bicipital groove can be felt as a distinct entity between the greater and lesser tuberosities when the arm is placed in neutral rotation. The pain felt in this location should migrate laterally with external rotation of the arm. Often, this pain radiates down anteriorly to the biceps muscle belly. This is different than rotator cuff tendinitis pain, which generally radiates to the deltoid insertion. Generally, the patient experiences the pain with activities, particularly repetitive overhead types seen in sports. Resting pain is seen later in the disease progression and can be a significant component of night pain. When involved with calcific tendinitis, the pain can be of such intensity that it may mimic a septic shoulder.

In the case of bicipital tendinitis, it is almost always accompanied by rotator cuff inflammatory symptoms.[95] The patient will complain of pain in the anterior aspect of the arm, particularly with extension and internal rotation. Impingement signs such as those described by Neer and Hawkins are generally positive.[48,55,96,99] We have found the lift-off sign, as described by Gerber and Krushell, to be helpful not only for detecting subscapularis tears but also as a sensitive test for eliciting impingement-type pain in patients with bicipital tendinitis.[47] Additionally, those patients with rotator cuff tendinitis with more severe symptoms and signs, including restriction of shoulder motion, are those more likely to have a component of bicipital tendinitis. Whether they are in association with tendinitis or full-thickness tears of the cuff, inflammation of the biceps tendon can be a significant source of additional discomfort. A significant pain that can sometimes accompany inflammation of the long head of the biceps has led some authors to believe it to be a possible cause in the development of frozen shoulder. DePalma and Callery reported a 40% incidence of frozen shoulder in bicipital tendinitis, particularly in the 45- to 55-year-old age group.[33] Modern interpretation of those findings is that the intraarticular synovitis associated with frozen shoulder preceded the bicipital involvement. When long-standing, the pain from the long head of the biceps tendon can spontaneously resolve following a full-thickness rupture (Fig. 7-11A). This dramatic relief of symptoms has been

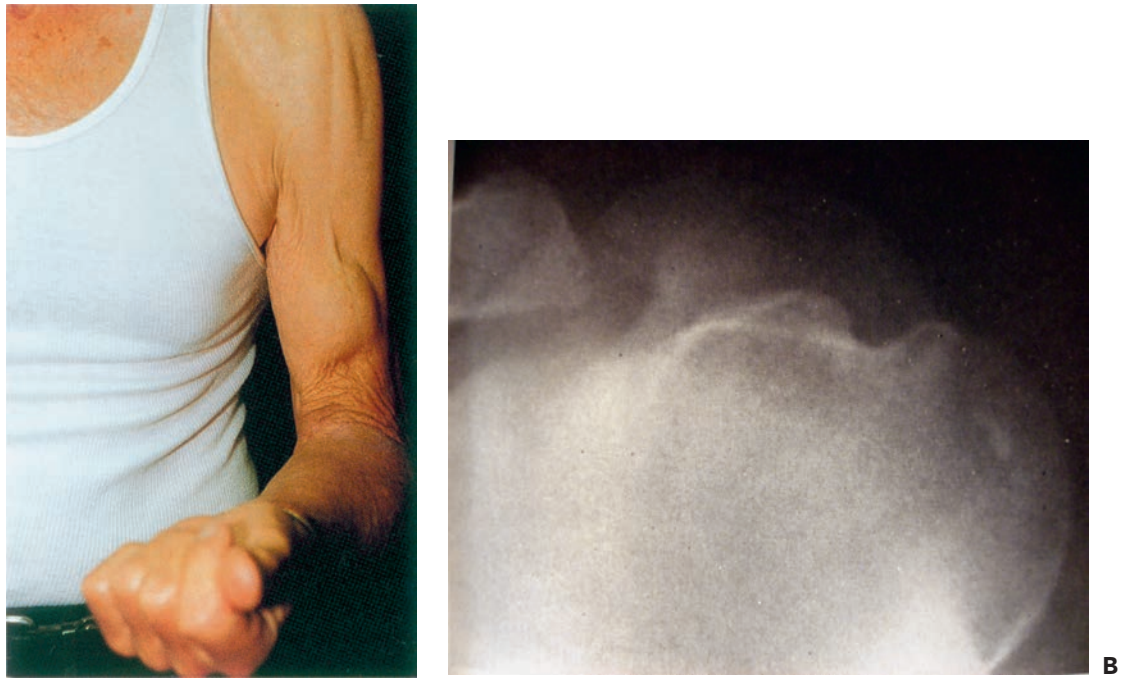


Figure 7-11 **A.** This is a 68-year-old patient with a long history of pain in the shoulder. The patient experienced a spontaneous resolution of the pain following a full-thickness rupture of the long head of the biceps tendon. The rupture of the long head of the biceps tendon is seen by a shortening of the lateral biceps muscle seen as a lump on the anterior lateral aspect of the arm. **B.** Biceps tendon view as described by Fisk. (Reprinted with permission, Fisk C. Adaptation of the technique for radiography of the bicipital groove. *Radiol Technol* 1965;37:47–50.)

termed rupture salvatrice, or "saving rupture." The rupture of the degenerated long head of the biceps is sometimes accompanied by bruising down the front of the arm along the biceps muscle belly in the presence of a lump on the anterior lateral aspect of the arm. When impingement-like pain is persistent following a rupture, further evaluation of the rotator cuff is warranted.

Stability of the biceps tendon in the form of subluxation is extremely difficult to assess nonoperatively. Patients that develop this condition are more likely to be young, overhead athletes.[102] They can often notice a painful snapping or clicking sensation in the shoulder, especially with overhead positions going from internal to external rotation. It is unclear why these symptoms occur as the tendon is generally fixed in a luxated location. As subluxation generally occurs in the presence of partial- or full-thickness surrounding rotator cuff defects, the symptoms are concurrent with those usually found with rotator cuff disease.

Frank dislocations of the long head of the biceps tendon are seen in the dominant shoulder of middle-age males.[106,107] The cause of this location is traumatic in the majority of cases. As with subluxation of the biceps, motion of the shoulder will aggravate the pain, especially in those of forced internal/external rotation as seen with throwing. Recurrent dislocation of the tendon of the groove can often be accompanied by a snapping sensation sometimes causing a sudden reflex dropping of the arm. The symptoms are similar to those seen in recurrent subluxation or dislocation of the glenohumeral joint. The series reported by Walch et al. highlights the fact that a large percent of patients with a dislocated biceps tendon will be seen in the context of a massive rotator cuff tear.[131] Approximately 40% of these patients were noted to have pseudoparalysis of the shoulder with attempted elevation.

Lesions of the superior labrum (SLAP) are similarly difficult to assess from history alone. These lesions are generally seen in younger patients that are extremely active in overhead activities. The cause is controversial but may involve posterior–inferior capsular contracture leading to a posterior–superior humeral head luxation. This luxation is proposed by Burkhart and Morgan to produce a sheer, peel-back mechanism for producing SLAP tears.[20] Alternatively, internal abutment of the articular surface of the supraspinatus against the labrum during maximal abduction, external rotation, and extension of the arm during throwing may be an alternative mechanism. A common cause is a fall on the outstretched arm in which the proposed mechanism is a compressive shearing of the humeral head against the superior labrum. A history of snapping or popping can also accompany these types of disorders.

Physical Examination

The most common physical examination finding for disorders of the long head of the biceps tendon is point tenderness over the intertubercular sulcus. This is most easily distinguished with the arm in 10 degrees of internal rotation where the biceps groove generally faces anteriorly and can be palpated approximately 7.5 cm distal to the acromion. In this position, the lesser and greater tuberosities can generally be palpated to locate the bicipital groove. The pain at the bicipital groove may be distinguished from anterior shoulder pain commonly associated with rotator cuff disorders by externally rotating the arm. Pain specific to the biceps tendon may move laterally with external rotation in the arm in contrast to subcoracoid pain.[48] Unless there is complete disruption of the transverse humeral ligament and luxation of the biceps tendon out of the groove, the long head of the biceps tendon can generally not be directly palpated. When dislocated, the biceps tendon can be rolled under the fingers of the examiner and this can exacerbate the tenderness.[33] Actual palpation of the tendon, however, is generally rare, and symptoms emanating from the bicipital groove are difficult to distinguish between tendinitis and instability.

Several specific physical examination tests have been described to isolate the biceps tendon as a source of pathology. These tests help to localize the tenderness to the biceps tendon but are not specific for any particular pathology. These tests include:

1. Speed's test or bicipital resistance test. The patient attempts to flex the arm with the forearm supinated and the elbow at approximately 30 degrees of flexion. A positive test is indicated by pain in the region of the bicipital groove.[50,96]
2. Yergason's test. Resisted supination of the forearm with the elbow flexed 90 degrees and the arm at the side can cause pain, specifically at the bicipital groove.[139]
3. Biceps instability test. In this case, dislocation of the tendon is elicited as a palpable and sometimes audible clicking as the biceps tendon is forcibly subluxated or dislocated. This is performed by placing the arm in full abduction and external rotation. The arm is then slowly brought down to the side in the plane of the scapula in progressive internal rotation.[1]
4. Shoulder compression test. This is performed with the patient supine and the shoulder is abducted to 90 degrees and the elbow flexed to 90 degrees. The axial compression force is applied to the humerus, which when rotated can sometimes elicit either pain or snapping of labrum. The test is analogous to the McMurray's compression test for meniscal tears in the knees.[120]
5. Modified biceps tension test. This test, designed to test superior labral lesions, is performed by holding the shoulder in 90 degrees of abduction and the forearm supinated. Pain is seen on application of a downward directed force applied to the distal forearm.[112]
6. Lift-off examination. Because instability of the biceps is so often associated with a loss of integrity of the subscapularis tendon, the lift-off test is an essential portion of the biceps evaluation. The lift-off test, as described by Gerber, is performed by placing the arm just short of maximal internal rotation and extension. The patient is then asked to actively lift the back of the hand off of the belt line or buttock depending on the amount of internal rotation achievable. The modified lift-off test (internal rotation lift-off sign) is performed by placing the arm in maximal internal rotation with the back of the hand posterior to the belt line or the buttock. A positive examination is seen when the patient cannot keep the hand in this position and it falls to the buttock or belt line. The hallmark of a positive test is a significant difference between active and passive maximal internal rotation and extension.[47] Positive examinations are highly suggestive of subscapularis tears. Additionally, as noted by Gerber, pain during this test serves as a sensitive indicator of impingement. Placement of the humeral head in internal rotation and extension positions the posterior rotator cuff under stretch and up against the acromial arch.
7. O'Brien sign. The shoulder is placed in sagittal plane forward flexion or slight adduction to the sagittal plane with the shoulder in full internal rotation. The examiner then pushes down against resistance when the patient is maintaining this position. The patient quantifies the level of pain caused. The examiner then positions the shoulder in full external rotation while otherwise maintaining the other positions of the shoulder and then forcibly pushes downward, and the patient quantifies the pain. A significantly greater amount of pain in the first position over the pain in the second position suggests biceps tendon, upper subscapularis tendon, and superior labrum pathology. Position one results in anterior internal glenoid impingement of these structures, and pathology of these tissues will elicit a position pain response.

Once these tests are performed, an essential part of the clinical evaluation for disorders of the long head of the biceps is the use of differential injections. Initially, a subacromial injection should be performed with lidocaine. The injection of a local anesthetic into a subacromial space is generally very effective for temporarily alleviating symptoms associated with rotator cuff tendinitis.[12,25,72] In the absence of a full-thickness tear, pain from the biceps tendon should remain persistent. Because the long head of the biceps occupies an intraarticular location, the use of a subacromial injection should not have a direct and immediate effect on pain associated with the tendon. In those cases with persistent pain, despite a subacromial injection, an

intraarticular injection of lidocaine is then administered.[12,21] Resolution of symptoms, particularly at the biceps groove or with any of the specific biceps examinations following an intraarticular injection, is relatively accurate for biceps tendon pathology.[12,98] When symptoms are not improved with an intraarticular injection, two clinical scenarios should be considered: (a) Marked inflammation within the intertubercular portion of the biceps tendon is preventing the infiltration of a local anesthetic in this location or (b) an alternate cause for anterior shoulder pain exists.

When a significant concern for biceps tendon pathology exists despite a negative intraarticular test, a direct injection of the biceps tendon sheath within the bicipital groove can be attempted. When done in a blind fashion, this injection can be difficult as direct injection of the tendon should be avoided.[72] Accuracy of this injection can be improved significantly by performing it under ultrasound guidance. If performed under high confidence, an injection of a local anesthetic should reliably relieve pain specific to the long head of the biceps tendon.

The clinical presentation of SLAP tears can vary significantly based on the age of the patient and the type of SLAP lesion. Kim et al. described the clinical presentation and arthroscopic findings of a series of patients with arthroscopic-proven SLAP tears.[73] One hundred and thirty-nine SLAP tears were diagnosed from a series of 544 (26%) consecutive primary shoulder arthroscopies. Most of the SLAP lesions were associated with other intraarticular pathology. Multivariate analysis showed that the majority of type I tears were seen in older patients and were associated with a positive Speed's test and the presence of a supraspinatus tear. The findings of those patients with type II SLAP tears differed according to the age of the patient. In older patients, type II SLAP tears correlated with a supraspinatus tear and osteoarthritis of the humeral head. In those under the age of 40 years, type II SLAP tears were associated with shoulder instability and Bankart tears. Type III and IV SLAP tears were seen in younger patients and were associated with high-demand occupations and a Bankart lesion.

The accuracy of physical examination tests commonly used for the diagnosis of SLAP tears is limited. McFarland et al. studied the accuracy of the active compression test (O'Brien's test), the anterior slide test, and the compression rotation test for the detection of SLAP tears.[87] The ability of these tests to correctly diagnose SLAP tears was examined in a group of patients with arthroscopically proven type II, III, and IV SLAP tears compared to a control group with a normal superior labrum or type I SLAP lesions. The incidence of positive results was not significant between groups. In those patients with SLAP lesions, the most sensitive test was the active compression test (47%) and the most specific test was the anterior slide test (84%). The test with the highest overall accuracy was the anterior slide test (77%) and with the lowest accuracy was the active compression test (54%).

DIAGNOSTIC IMAGING

Despite significant progress in shoulder diagnostic techniques such as magnetic resonance imaging (MRI) or high-resolution ultrasound, imaging for disorders of the biceps tendon and superior labrum has remained difficult and nonspecific. The value of plain films, MRI, ultrasonography, or arthrography has come more from the characterization of associated pathology such as rotator cuff disease. Although the long head of the biceps tendon and superior labrum can at times be well visualized by some of these methods, any finding must be strictly placed in the context of a history and physical examination. Conversely, the lack of positive findings does not rule out or even deem less likely the presence of significant long head of the biceps tendon pathology.

Plain Films

Imaging of a painful shoulder begins with plain films. In our institution a standard shoulder series of plain radiographs includes the anteroposterior (AP) view with the humerus in internal rotation, posterior oblique "true AP" view of the shoulder in external rotation, supraspinatus outlet view, and axillary lateral view. Plain radiography of the shoulder may demonstrate radiographic changes of an associated long-standing rotator cuff disease, with sclerosis of the greater tuberosity, anterior acromial spurs, and decrease in the subacromial space.[58] When a disorder of the long head of the biceps is suspected, specific additional views can be a useful adjunct.

While the tuberosities are visible on anteroposterior and axillary views, special views to visualize the bicipital groove have been described. The "groove view" described by Cone permits measurement of the depth and width of the groove as well as the slope of the medial wall and may demonstrate degenerative changes in half the patients with tendinitis[3–5,29a] (Fig. 7-11B). This study is obtained with the patient supine and the arm in external rotation. The x-ray beam is directed cephalad and 15 degrees medial to the long axis of the humerus toward the film cassette, which rests on the superior aspect of the shoulder. An alternative method of imaging the biceps groove was described by Fisk.[40] This view is obtained by positioning the patient in a semiprone position resting on the posterior aspect of the elbow. The shoulder is semiflexed and slightly externally rotated, the elbow is bent, and the cassette is positioned on the supinated forearm of the patient. The x-ray beam is directed from superior to inferior over the shoulder of the patient. This view also affords a direct longitudinal view of the biceps groove.

Arthrography

Arthrography can be a valuable adjunctive tool in the evaluation of biceps-related shoulder pain. The accuracy in the

diagnosis of rotator cuff disease has been well established when leakage of dye injected into the shoulder joint is seen in the subacromial space.[4] The ability to visualize the tendon sheath by arthrography has extended its indications to evaluation of the long head of the biceps tendon. There are two openings normally present in the shoulder capsule, the first being the subscapularis bursa and the second, the biceps tendon synovial sheath. Preliminary scout films are obtained, as are anteroposterior views in internal and external rotation and abduction of the shoulder, an axillary view, and a bicipital groove view. In obtaining anteroposterior views, the tube is tilted approximately 15 to 20 degrees caudad to allow visualization of the subacromial space without posterior overlap by the acromion.[115] Normally the entire shoulder should fill from the glenoid to the anatomic neck without any irregularities. When the biceps tendon sheath and tendon outline is easily visualized without any narrowing or vacuolization, significant inflammation is unlikely[99] (Fig. 7-12). Loss of the sharp delineation of the long head tendon may suggest the presence of associated synovitis. The main drawback of arthrography is that filling of the sheath of the biceps tendon is unreliable and may be absent in up to 31% of arthrograms, especially in the presence of full-thickness cuff tears.[91,92] The absence of filling of a normal tendon may therefore be difficult to differentiate from rupture. Although vacuolization of the sheath with narrowing of the contrast may be noted in some cases, often there may be no difference in the pattern of filling of the tendon sheath in cases of tendinitis compared to normal tendons.[3,5] Because of this, arthrography of the shoulder can be of limited value in the diagnosis of bicipital tendinitis without atrophy, fraying, or subluxation of the tendon.[3] Arthrography can be useful for demonstration of the bony configuration of the bicipital groove and sensitive for the detection of subluxation of the biceps tendon medially out of the groove.[96] The sensitivity for detection of subluxation or dislocation of the tendon can be enhanced with commuted tomographic (CT) arthrography.[56] Although useful at times, arthrography has become less popular in the age of MRI and ultrasonography because it is an invasive procedure with potential side effects, such as exacerbation of shoulder pain; complications, such as allergic reaction to the contrast medium; and infection.[57]

Ultrasonography

In the late 1980s, with improving technology and expertise, ultrasonography became increasingly applied for the diagnosis of shoulder pathologies. Ultrasonography has the advantage of correlating, in a dynamic fashion, abnormal findings with clinical sites of tenderness.[2,83,91,92] The test is noninvasive and relatively inexpensive, and offers the opportunity for bilateral examinations at the same sitting. The tendon can be best imaged by transverse scanning at the bicipital groove.[91,92] The normal tendon appears as an echogenic ellipse within the groove (Fig. 7-13). Proximal to the groove, the tendon is visible lying against the humeral head, covered by the supraspinatus posterosuperiorly and the subscapularis anteroinferiorly. On longitudinal imaging, the tendon appears as a narrow band of tissue between the humerus and the deltoid, differentiated from the latter due to its greater echogenicity. Absence of the biceps tendon in its sheath indicates rupture or dislocation out of the groove. Ultrasound can detect effusions within the sheath

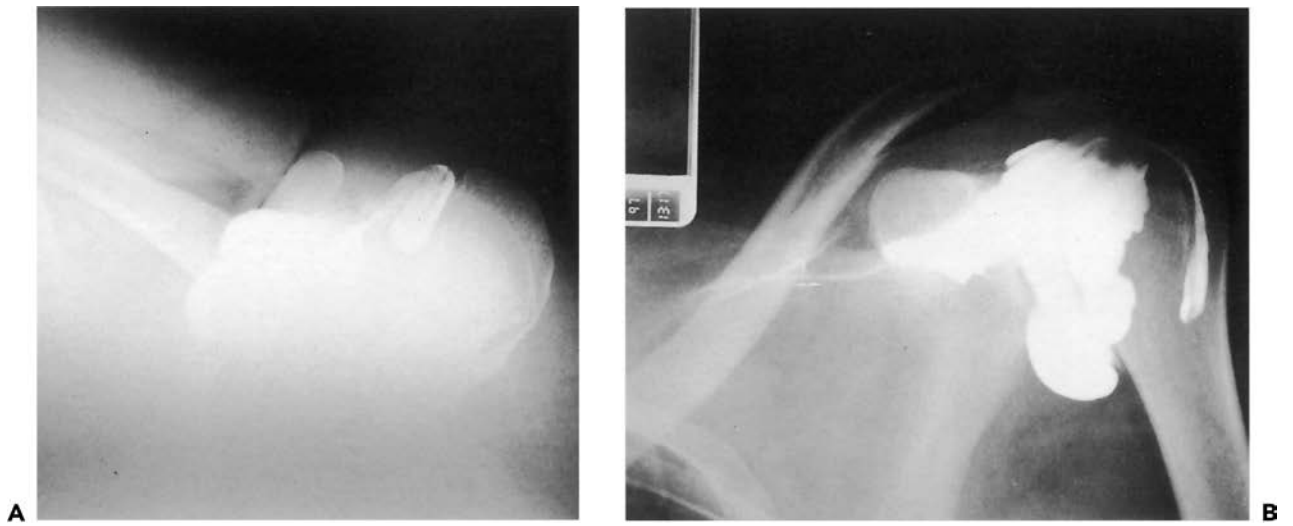
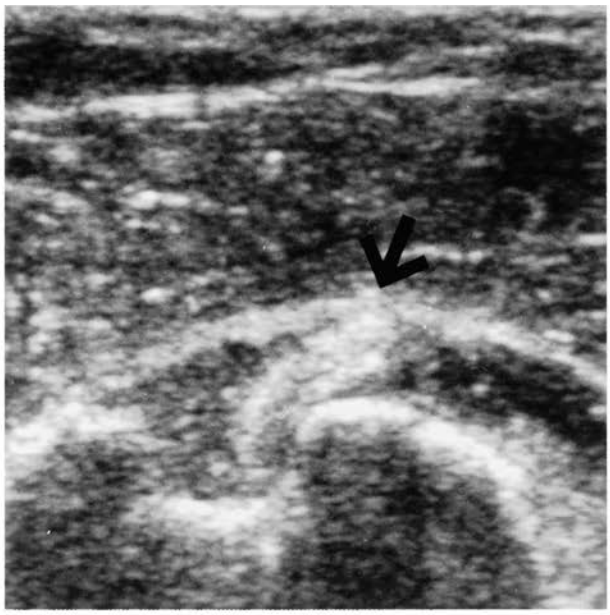


Figure 7-12 Arthrogram of the glenohumeral joint showing the relationship of the biceps tendon. **A.** Contrast dye is normally seen down the biceps tendon sheath and a tendon outline can generally be visualized. In absence of any significant narrowing or vascularization, this generally indicates that inflammation is unlikely. **B.** Dye in the bicipital groove can also be seen on the biceps view.



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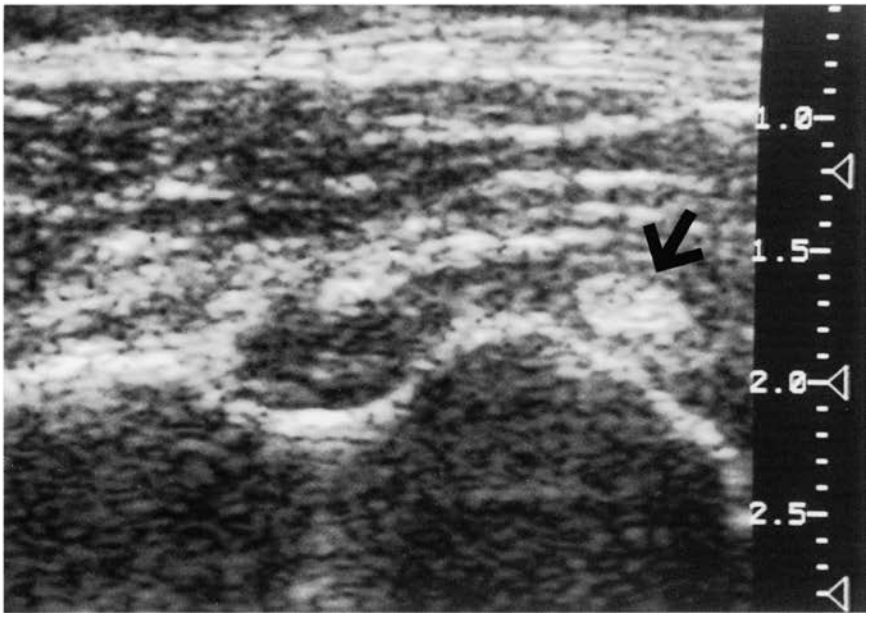

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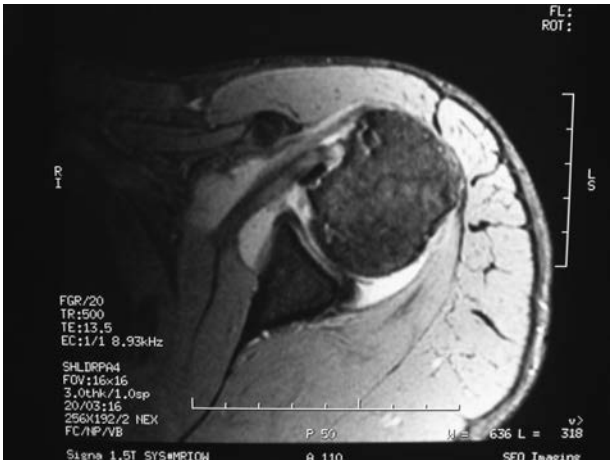

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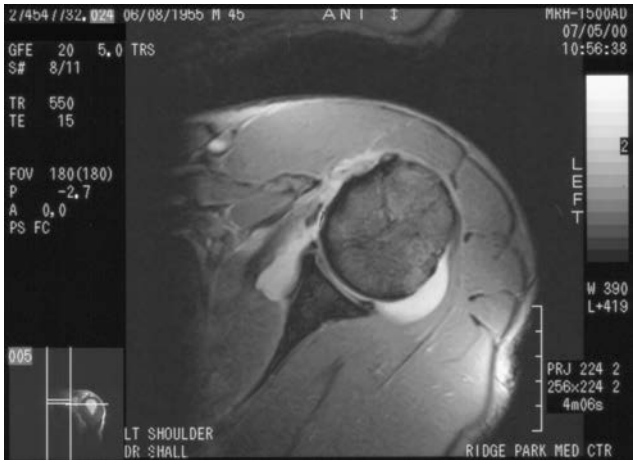

D

Figure 7-13 **A.** Ultrasound view shows a subluxated biceps tendon. The arrow is pointing to the hyperechoic (white) biceps tendon as it is perched on the lesser tuberosity. The transverse humeral ligament, which is also hyperechoic, is seen overlying the tendon. **B.** Ultrasound view showing a dislocated biceps tendon. The arrow is pointing to the hyperechoic biceps tendon, which is medially dislocated on top of the lesser tuberosity. **C.** Magnetic resonance image (MRI) of subluxated biceps tendon with partial subscapularis tear. **D.** MRI of dislocated biceps tendon and full-thickness subscapularis tendon tear.

of the biceps, and can detect other abnormalities in the groove such as osteocartilaginous loose bodies. In a prospective study of 80 patients, Middleton et al. compared the value of sonography and arthrography in the detection of lesions at the bicipital groove and distally in the biceps tendon.[92] In 20% of cases, with negative arthrograms for biceps lesions, effusions of the sheath of the biceps tendon were demonstrated by ultrasonography. Most of these patients had other associated pathology in the shoulder.

In our experience, ultrasonography has been extraordinarily accurate not only in defining the presence of associated rotator cuff defects, but also in precise characterization of the morphology.[125] Subtle partial-thickness or full-thickness defects in the anterior edge of the supraspinatus or superior edge of the subscapularis, seen with tendon instability, can be visualized with surprising accuracy. These improvements have followed recent technologic gains in the resolution of the scanners and refinements in technique gained with increasing clinical experience.

There are some limitations to ultrasonography. The test is highly dependent on the technical expertise of the radiologist and at this time is not widely available. Additionally, the viewing area is constrained by the bony anatomy. Because the acromial process cannot be penetrated, medial pathology at the joint line such as labral defects are not well visualized. Given these disadvantages, it is anticipated that ultrasonography will become increasingly popular secondary to significant advantages in cost effectiveness, patient tolerance, bilateral information, and associated accuracy.

Magnetic Resonance Imaging

Magnetic resonance imaging provides high-resolution soft tissue imaging in a noninvasive fashion and has the advantage of multiplanar study of the shoulder.[64] In addition, the morphology of the bicipital groove can be studied and bony osteophytes or other abnormalities detected.[24] Images in the axial plane and in the coronal oblique and sagittal oblique planes (with reference to the plane of the subscapularis) are used for a routine shoulder study. In axial images, the biceps tendon can be identified on T1-weighted images as a round, low-signal intensity structure contained in the bicipital groove. The tendon of the subscapularis muscle can be visualized sweeping anterior to the tendon to its insertion on the lesser tuberosity. In the coronal oblique sections, the tendon is seen as a linear structure between the greater and lesser tuberosities. Sagittal oblique sections can demonstrate segments of the tendon within the groove. In cases of bicipital tendinitis, an effusion may be noted in the tendon sheath with or without a corresponding effusion in the shoulder joint. Thickening of the tendon may be observed in some cases with hypertrophic tendinitis. An empty groove in the axial section suggests rupture or dislocation of the tendon. A medially dislocated tendon should be considered in these cases (Fig. 7-13B,C). It is important to follow the tendon on serial axial sections to avoid mistaking osteophytes in the groove for the tendon. As a high incidence of abnormalities or disruption of the subscapularis tendon is reported in association with luxation of the biceps tendon, special attention should be given to it. Because the long head of the biceps tendon is not visualized in continuity, it is easy to miss subluxations or even dislocations of the tendon between slices. Spritzer et al. examined the effectiveness of conventional MRI to detect the presence of biceps tendon instability. In this small series, MRI had a sensitivity of 67% and a specificity of 90% in detecting biceps tendon instability.[124] Another series reported the accuracy of MRI in detecting surgically confirmed lesions of the biceps tendon and their association with rotator cuff tears. The MR examinations were performed with IV contrast only. The sensitivity, specificity, and accuracy of unenhanced MRI for detecting biceps tendon tears were 52%, 86%, and 79%, respectively. When a tear was present in the biceps tendon, the prevalence of supraspinatus, infraspinatus, and subscapularis tendon tears was 96%, 35%, and 47%, respectively. Patients with biceps tendon tears were significantly more likely to also have subscapularis and supraspinatus tendon tears than patients with a normal biceps tendon.[8]

MRI has not proven useful in the diagnosis of superior labral lesions, with reports of a positive scan in only about a third of cases.[39] Magnetic resonance arthrography employing the intraarticular injection of gadolinium has been reported to provide better definition of labral lesions than conventional MRI.[41] Bencardino et al. evaluated the accuracy of MR arthrography for the detection of SLAP tears in a series of patients with confirmative surgical findings. MR arthrography showed a sensitivity of 89%, a specificity of 91%, and an accuracy of 90% for detecting SLAP injuries.[10]

The value of MRI appears to be more for defining associated shoulder pathology such as partial- or full-thickness rotator cuff tears.[64] However, it is an expensive and at times poorly tolerated test and is generally not recommended as the procedure of choice for imaging the long head of the biceps tendon or superior labrum.

NONOPERATIVE TREATMENT

Long Head of the Biceps Tendinitis

Nonoperative treatment of long head of the biceps tendinitis has generally been directed toward treatment of the rotator cuff. A failure to improve or an increase in symptoms is more often seen in those cases with a component of

biceps tendinitis. Biceps tendinitis is typically more resistant to treatment and does not respond to subacromial steroid injections.[21,96–98] For this reason direct tendon sheath injections have been suggested when subacromial ones have failed. DePalma and Callery, in a series of 18 cases treated for isolated bicipital tenosynovitis, reported improvement in 10 when given a series of hydrocortisone injections into the tendon directly under the transverse ligament.[33] More modern concerns for atrophic tendon changes from the steroid have led to recommendations for sheath injections in preference to intratendinous ones.[72] A 74% good and excellent result has been reported with this approach.[72] Technical difficulties associated with "blind" injection of biceps tendon sheath have led to recommendations for intraarticular injections as an alternative method.

Very little information is available about the nonoperative treatment of SLAP lesions or long head of the biceps instability. As tendon instability almost invariably follows the development of significant rotator cuff pathology, treatment is again directed primarily along those guidelines. With instability of the biceps, nonoperative measures are less likely to be successful and earlier operative intervention or prolonged activity restrictions are required.

Spontaneous or traumatic ruptures of the long head of the biceps tendon do not require operative intervention.[21,23,56] Although associated with a cosmetic defect, the long-term functional and symptomatic sequelae are minimal. Mariani et al. compared 26 patients who underwent early tenodesis of a rupture of the long head of the biceps with 30 patients who had nonsurgical treatment.[86] Residual pain was found to be infrequent for both groups. On biomechanical testing the nonsurgical group lost 21% of supination strength versus 8% for the surgical group. There was no significant difference in elbow flexion. The nonsurgical patients returned to work earlier. Similar results were reported by Warren, who showed no loss of elbow flexion strength and only 10% supination loss.[134] Although the vast majority of tears of the long head of the biceps are the result of attritional tendon changes in the elderly, there is a much lower incidence of traumatic tears in the younger and active patient. In these patients some (up to 30%) will have symptoms of cramping pain in the biceps muscle with strenuous activities. Some of these symptoms improve over time, but a percentage of these patients' symptoms persist. It may be advisable to tenodeseis the biceps in these younger and active patients.

Authors' Preferred Treatment—Long Head of the Biceps Tendinitis

Nonoperative treatment is primarily directed at the accompanying rotator cuff tendinitis. This includes rest, local modalities such as ice, nonsteroidal antiinflammatory medication, physical therapy, and local steroid injections.[93,108] When patients present with acute or significantly painful shoulders, initial treatment is directed toward inflammation control instead of strengthening. Starting physical therapy at this time may cause exacerbation of symptoms. Rather, a short period of rest or reduction of activities is started.

Nonsteroidal oral medication given at antiinflammatory doses is used in conjunction with local measures like ice. It should be noted here that many patients who present for orthopaedic consultation have some history of nonsteroidal use. However, dosages and compliance is often insufficient for anything more than an analgesic effect. To achieve maximal antiinflammatory benefit, nonsteroidal medication should be taken in a sustained fashion and at antiinflammatory doses.

Subacromial injections are given only in the context of severe night pain or a failure to improve with 6 weeks of treatment. Physical therapy is instituted only after some initial improvement in symptoms. It is directed toward range-of-motion stretching and rotator cuff strengthening. There is no attempt made to specifically strengthen the biceps muscle. If there are strong signs or suspicions of biceps-related pain, then an intraarticular injection of steroids is considered. Theoretically, to achieve maximum benefit, the steroid should be injected into the tendon sheath only and intratendinous injection avoided. Because this is technically difficult to achieve on a consistent basis, we prefer to inject the solution into the shoulder joint cavity, thereby addressing the intraarticular and extraarticular portions of the tendon.

We generally prefer to perform intraarticular injections from a superior approach analogous to the superior arthroscopic portal as described by Neviaser.[100] A spinal needle is required. The entry site is in the supraclavicular region. The needle is inserted 1 cm medial to the medial border of the acromion just posterior to the clavicle and anterior to the scapular spine. A "soft spot" can reliably be palpated there. The needle is advanced, aiming 30 degrees anteriorly and 30 degrees laterally. To assist in orientation, the arm is placed in 30 degrees of coronal plane abduction and the needle directed down the long axis of the arm. Upon contact with the humeral head, the needle is withdrawn by a few millimeters and a solution of local anaesthetic and water-soluble steroid suspension is injected. Immediate and temporary relief of symptoms is diagnostic and hopefully therapeutic.

The patient is monitored at 6-week intervals throughout the nonoperative treatment period (Fig. 7-14). At each of these follow-up consultations a reevaluation of progress is coordinated with indications for further workup or treatment changes. When significant improvements are made, a gradual tapering of nonsteroidal medication and progression of activities and physical therapy is attempted. When there are no significant changes, depending on the severity and nature of the symptoms, further observation without

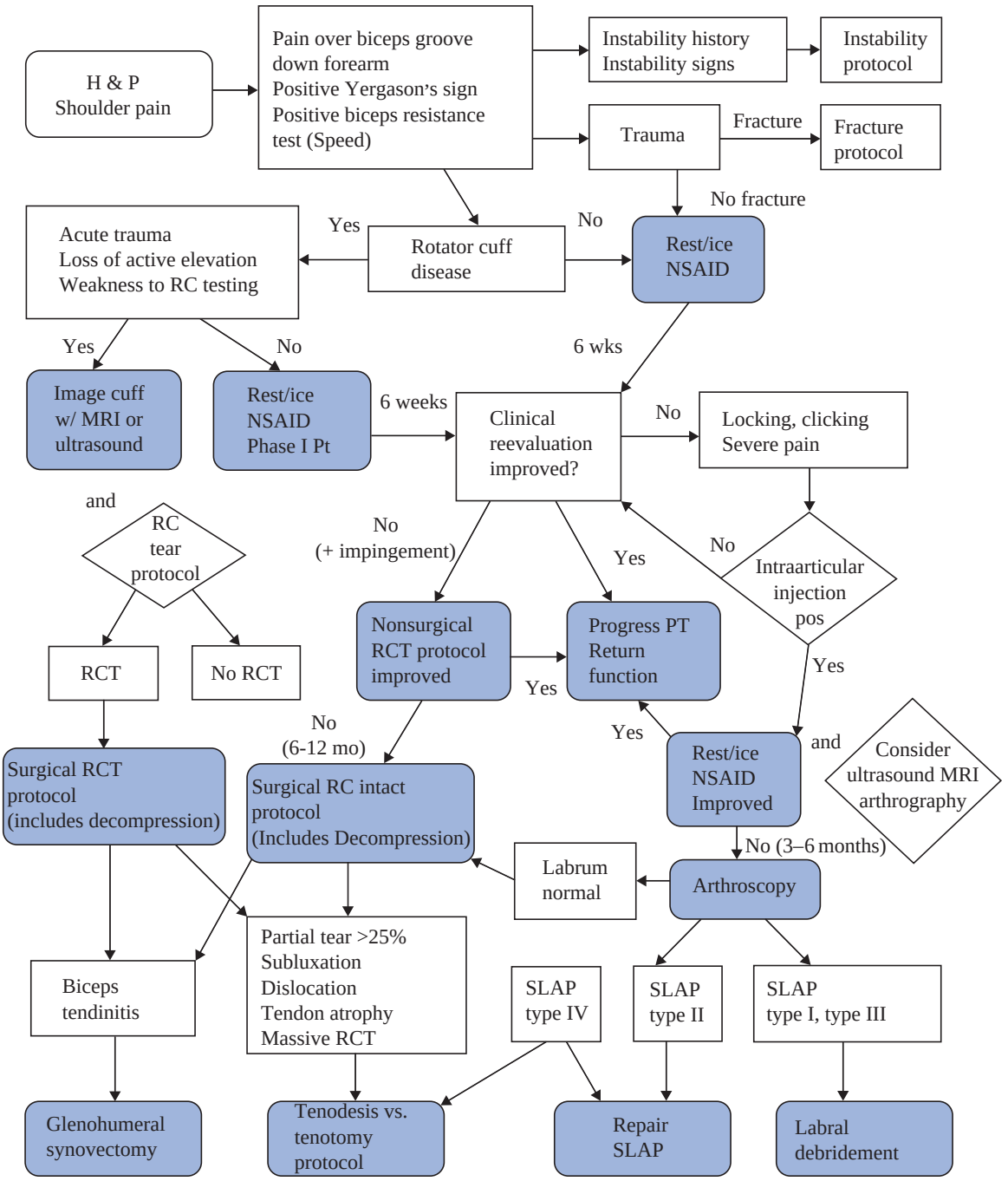

Figure 7-14 Algorithm for treatment of long head of biceps disorders. H&P, history and physical; MRI, magnetic resonance imaging; RC, rotator cuff; NSAID, nonsteroidal anti-inflammatory drug; RCT, rotator cuff tear; PT, physical therapy; SLAP, superior labral anterior posterior.

treatment changes is reasonable. The deterioration of symptoms or a continuation of severe symptoms should alert the treating physician that further workup or treatment modification may be indicated. Treatment modification should include differential injections, including intraarticular as described above. Further workup may include imaging for rotator cuff tears and biceps tendon subluxation. Ultrasound, MRI, and CT arthrography are examples of modalities that may provide information about both the rotator cuff and long head of the biceps tendon. If a strong suspicion remains for biceps-related pain despite a negative intraarticular injection, a bicipital groove injection is considered. In our institution, this injection can be performed with high accuracy with the use of ultrasound guidance. In the absence of significant findings with these studies, nonoperative treatment including repeat injections (maxi-

mum of three) is continued for a minimum of 6 months if the injections are positive (patient experiences some relief of symptoms). In the absence of copathology such as rotator cuff tears or tendon instability, a high percentage of patients (greater than 80%) are expected to experience satisfactory improvement. A negative finding for both imaging studies and injections should alert the treating physician for alternate causes of anterior shoulder pain including instability, adhesive capsulitis, glenohumeral arthritis, acromioclavicular arthrosis, coracoid impingement syndrome, neurogenic causes, and medical conditions.

Long head of the biceps instability and SLAP lesions are generally considered operative indications in those rare circumstances where a strong preoperative diagnosis is available and there is greater than 3 months of pain. Nonoperative treatment for these disorders is limited to rest and intraarticular injections.

SURGICAL TREATMENT

Reflecting remaining controversies about the functional and symptomatic significance of the long head of the biceps tendon, multiple and often contradictory treatment strategies and indications have been recommended. Options for surgical treatment include benign neglect with treatment of associated pathology only (i.e., rotator cuff, labral defects), inspection and synovectomy, repair of partial tears, tenodesis in the intertubercular groove, or simple intraarticular tenotomy. Historically, concerns about functional deficits created by loss of the long head of the biceps tendon have led to a prevailing operative strategy for avoidance of tenodesis whenever possible. If sacrifice of the biceps tendon is indicated, the decision to perform tenotomy versus tenodesis is based upon the age, functional demands, and cosmetic concerns of the patient.

Treatment for Biceps Pathology Associated with Rotator Cuff Disease

Pathophysiology

Surgery for rotator cuff disorders accounts for the majority of cases in which biceps pathology is encountered. The biceps tendon is susceptible to the same mechanical abutment seen with impingement of the rotator cuff tendons. Whether primary or secondary causes of impingement exist, the long head of the biceps occupies an anterior location within the impingement zone, which predisposes its involvement with rotator cuff disease. Besides these compressive forces, the biceps tendon is susceptible to inflammation and degenerative changes secondary to the synovitis initiated from primary rotator cuff disease.

Neer recommended routine inspection of the long head of the biceps and intertubercular groove as part of the surgical procedure for impingement syndrome.[95] Later reports by Neviaser have noted a high prevalence of pathologic changes in the biceps tendon macroscopically, as well as microscopically (in normally appearing intraarticular portions of the biceps tendon) in patients with rotator cuff disease.[94] Based on their experience, they recommended inclusion of biceps tenodesis with excision of the intraarticular segment as part of the surgical procedure for the treatment of impingement syndrome.[97] The work of Petersson and Murthi et al. has highlighted the strong association prevalence of biceps tendon pathology with rotator cuff disease.[94,107] In addition, the incidence of biceps pathology increases with advancing age the severity of the underlying cuff disease.

Treatment of Biceps Tendonitis Associated with Rotator Cuff Disease

While the high association of long head of the biceps tendon pathology has not been disputed, indications for surgical treatment have been varied and inconsistent. According to Crenshaw and Kilgore, the indications for tenodesis were pain present for an average of about 5 months, bicipital tenderness, and restriction of motion.[31] The authors reviewed 89 patients who had undergone surgery for bicipital tenosynovitis, with a minimum follow-up of 1 year. Average age at the time of surgery was 52 years. All patients were treated with bicipital tenodesis using various techniques. Maximum improvement was reached at about 12 months after surgery, with excellent and good results obtained in 87%. Pain relief was dramatic with relief from disabling pain in 80% by the end of the month, and in 95% by 3 months.

Neer highlighted the importance of mechanical impingement from the anterior acromial arch as the primary cause of anterior shoulder pain.[95] In a series of 50 shoulders with the preoperative diagnosis of biceps tenosynovitis, only 30% had significant biceps abnormalities and favorable results were obtained with acromioplasty alone. In this series, tenodesis was rarely performed, and biceps-related symptoms were reliably improved from decompression alone. Neer recommended avoidance of biceps tenodesis, whenever possible, to prevent a loss of head depressor effect.

Other early reports recommended tenodesis for shoulder pain unresponsive to conservative measures and regardless of the extent of operative findings. Lippmann observed a relief of symptoms in patients who developed spontaneous adhesion of the tendon in its groove and attempted to reproduce the same surgically in acute cases.[84] In this technique, the long head of the biceps tendon was anchored in its groove with several nonabsorbable sutures passed through drill holes in the lesser tuberosity. Hitchcock and Bechtol also described fixation of the tendon in the bicipital groove.[65] Their technique, however, created an osteoperiosteal flap raised from the floor of the groove. The tendon was placed deep to this flap and secured with nonabsorbable sutures. The transverse

humeral ligament was sutured over, reinforcing the repair. An alternative and popular technique for the long head of the biceps tenodesis after release of the intraarticular origin, as described by Froimson and termed the "keyhole" technique, involved placement of a rolled or knotted biceps tendon into a keyhole-shaped trough that had been drilled in the bicipital groove[43] (Fig. 7-15).

The importance of treating associated rotator cuff pathology was highlighted in several other series. In a retrospective review of 20 shoulders in 18 of patients who underwent biceps tenodesis without subacromial decompression, Dines et al. reported good results in 14 shoulders at a mean follow-up of 3 years.[34] The patients were categorized into two groups based on findings at surgery: those with inflammatory lesions in the bicipital groove and those with instability of the tendon. The majority of the patients were young with an average age of 33 and 35 years in the two groups, respectively. In addition to the biceps procedure, incision of the coracoacromial ligament was performed in 14 of 20 cases. Poor results were associated with younger age and failure to release the coracoacromial ligament. Of the six failures, four were attributed to impingement against the acromion and glenohumeral instability was observed in two. The authors recommended careful preoperative examination for the exclusion of glenohumeral instability in younger patients and suggested that acromioplasty be a major component of the surgical procedure in older patients. The authors did not report any complications, such as superior head migration, arising from tenodesis of the long head.

Becker and Cofield reported the long-term follow-up of 54 shoulders at an average of 13 years after surgical tenodesis of the long head of the biceps for the treatment of chronic tendinitis.[9] As part of the exposure, the coracoacromial ligament was routinely transected, the intraarticular portion of the long head was removed, and the remainder was tenodesed at the bicipital groove or to the short head. Acromioplasty was not performed. At latest follow-up, 22 shoulders had mild or no pain, whereas the other 22 continued to be moderately[113] or severely[137] painful. Twenty-nine patients needed additional treatment in the form of steroid injections or surgical procedures (8%) including rotator cuff tear repairs, anterior acromioplasty, and excision of the distal clavicle. They advised against bicipital tenodesis without decompression as the primary surgical procedure due to deteriorating long-term results. In their opinion tenodesis was indicated if, during a surgical procedure directed primarily at the rotator cuff, degenerative changes were encountered in the biceps tendon or if the tendon was unstable or displaced from its groove.

In contrast to the Becker and Cofield study, tenodesis of the long head of the biceps has been reported to have good results even when not performed in conjunction with a decompression of the coracoacromial arch. Post and Benca showed 94% excellent results in a series of 13 patients with primary bicipital tendinitis.[110] Berlemann and Bayley reported improved results of biceps tenodesis with long-term follow-up.[12] Of interest, 8 of the 15 cases in this series had persistent pain following decompression. Six of the eight then had complete resolution of the symptoms following biceps tenodesis.

While the long-term results as reported by Becker and Cofield would suggest a relatively poor outcome for an isolated biceps tenodesis, no studies have shown adverse results when performed in conjunction with a decompression of the coracoacromial arch. Neviaser and coauthors reported their experience with 89 patients who underwent routine biceps tenodesis as part of the procedure for subacromial decompression.[97] They operated on 89 patients, the average age being 42 years, and evaluated the results at 2 to 8 years. Patient results were based on pain relief and range of motion. All but one patient reported no pain (86%) or pain with unaccustomed exertion (13%). One patient was diagnosed to have adhesive capsulitis postoperatively and continued to have pain at night and intermittently during the day. The authors did not report any functional loss secondary to tenodesis of the biceps tendon.

Despite the excellent results reported for biceps tenodesis performed in conjunction with acromioplasty, the routine employment of a tenodesis is unsupported.[95,115] Multiple studies in which biceps tenodesis was generally avoided during treatment of the rotator cuff have shown good results with decompression and cuff repair alone.[13–16,29,36,37,46,60,62,78,127] It is, however, unclear how many of the failures may have been secondary to persistent biceps-related pain.

Recent advances in equipment and surgical techniques now allow biceps tenodesis to be performed arthroscopically. A variety of tendon fixation techniques have been described using suture anchors, tenodesis screws, or soft tissue tenodesis alone. Many of these techniques are technically demanding but offer the advantage of secure tendon fixation via small, cosmetic incisions.

Gartsman and Hammerman described a technique of arthroscopic biceps tenodesis using suture anchors.[45] The intraarticular portion of the biceps tendon is pierced with a spinal needle at the proximal aspect of the bicipital groove. The arthroscope is directed into the subacromial space and the needle is used to identify the biceps tendon. The biceps sheath is open and débrided with a shaver through the lateral cannula. The biceps groove can be deepened with a burr. One or two suture anchors are placed into the biceps groove. A Caspari punch is used to place a shuttle suture through the biceps tendon via the lateral cannula. One limb of the anchor suture is then shuttled through the tendon using the shuttle suture. These steps are repeated with the remaining limb of the anchor suture creating a mattress stitch, which is then secured arthroscopically. The biceps tendon is then divided and the intraarticular portion of the tendon is removed arthroscopically.

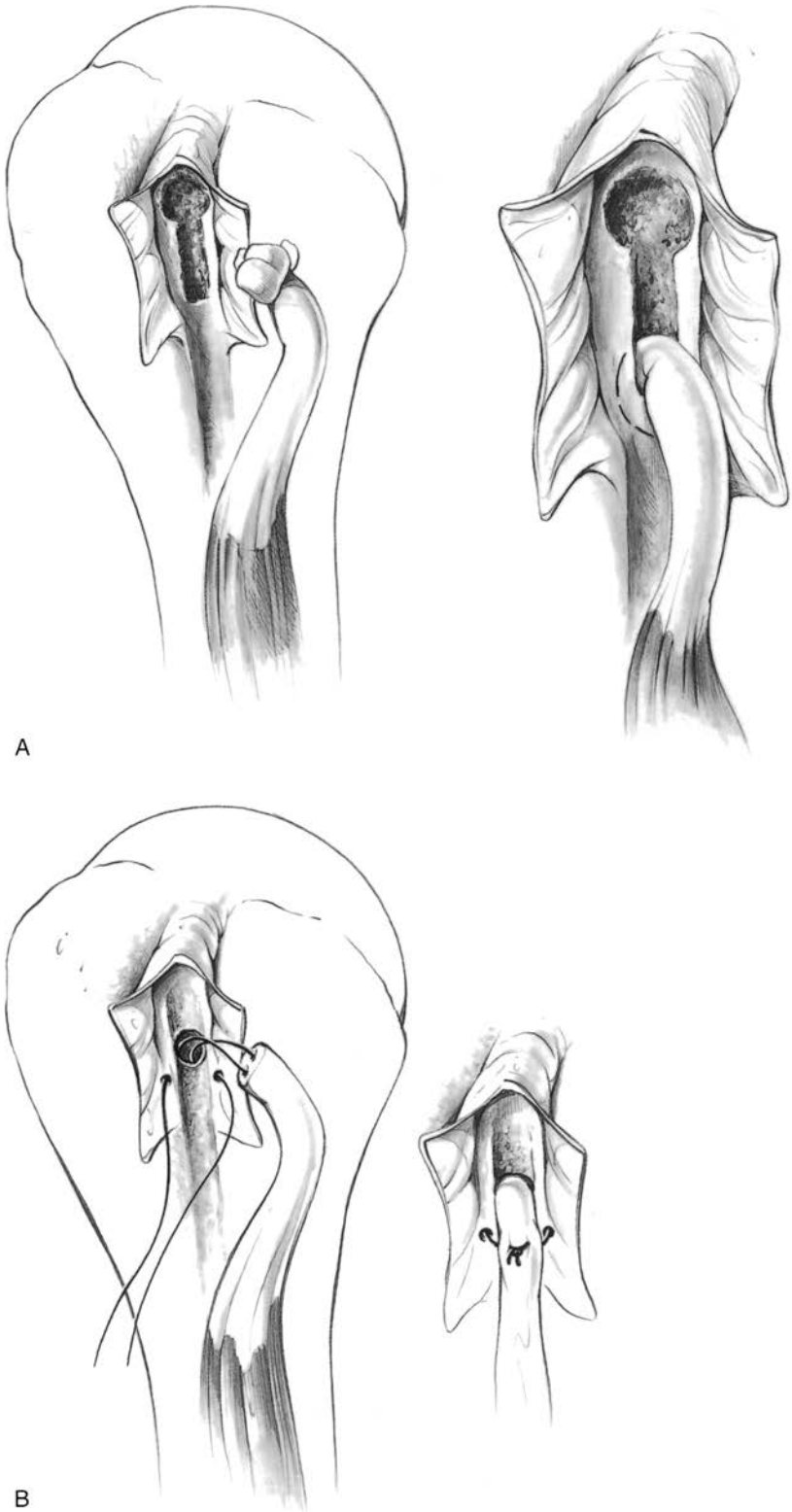

Figure 7-15 A schematic representation of two popular techniques for open biceps tenodesis. **A.** In the keyhole technique, as described by Froimson, the knotted proximal end of the biceps tendon is placed into a keyhole slot, which had been drilled into the bicipital groove. **B.** In the Post technique, the proximal portion of the biceps tendon is inserted into a round hole drilled into the bicipital groove. Tendon grasping stitch, which had been placed through the proximal end of the tendon, is brought into the hole and out through two drill holes. The tendon is then sutured into itself.

Boileau et al. and Klepps et al. have described techniques of arthroscopic biceps tenodesis using bioabsorbable interference screws for tendon fixation. Boileau et al. reported a technique of interference screw fixation after pulling the tendon into a tunnel in the proximal aspect of the bicipital groove with sutures that have been delivered out the back of the shoulder by drilling through the proximal humerus with Beath pins.[18] Results of this technique in 43 patients were recently reported.[19] The majority of cases were performed in conjunction with débridement of an irreparable massive cuff tear. The absolute Constant score improved from 43 to 79. Two failures of the tenodesis were noted. Elbow motion was full and biceps strength was 90% of the opposite side.

Klepps et al. described a technique of interference screw fixation utilizing a suture anchor at the base of the tunnel to feed the biceps tendon into the bone.[74] The intraarticular portion of the biceps tendon is tagged with a suture at the proximal aspect of the bicipital groove using a spinal needle. The origin of the tendon is released from the superior labrum. The arthroscope is then directed into the subacromial space. A lateral portal is established, through which a bursectomy and subacromial decompression are performed. An anterolateral portal is created directly over the biceps groove. After the biceps groove is opened and débrided, the biceps tendon is exteriorized through the anterolateral portal, shortened, and tagged with a heavy, nonabsorbable suture in a running fashion. The appropriate tunnel location within the bicipital groove is marked with a pin through the anterolateral cannula. The tunnel is enlarged with a cannulated reamer. The reamer size should be the same size or slightly larger than the width of the biceps tendon. The size of the interference screw should be the same width of the biceps tendon or 1 mm larger. If a "biotenodesis tray" is not available, a suture anchor can be used to shuttle the tendon into the tunnel and held while the interference screw is placed (Fig. 7-16). The anchor is placed into the base of the tunnel. One limb of the anchor suture is placed through the biceps tendon. The other limb of the anchor suture is pulled, thereby feeding the tendon into the tunnel. The Arthrex biotenodesis tray (Arthrex, Naples, FL) contains a cannulated screwdriver that will feed the tendon into the tunnel as the interference screw is placed. A flexible guidewire is used to feed one limb of the tendon suture through the driver. This suture is tensioned while the tip of the screwdriver guides the tendon into the tunnel (Fig. 7-17). The interference screw is advanced into the tunnel through the anterolateral cannula. The two limbs of the biceps tendon suture, one limb now inside and one outside the interference screw, are then tied, providing further fixation of the tendon to the screw.

An alternative method of arthroscopic biceps tenodesis can be performed using soft tissue fixation only. Soft tissue tenodesis can be accomplished quicker than those techniques requiring bone fixation. In addition, soft tissue tenodesis is less demanding technically and is associated with

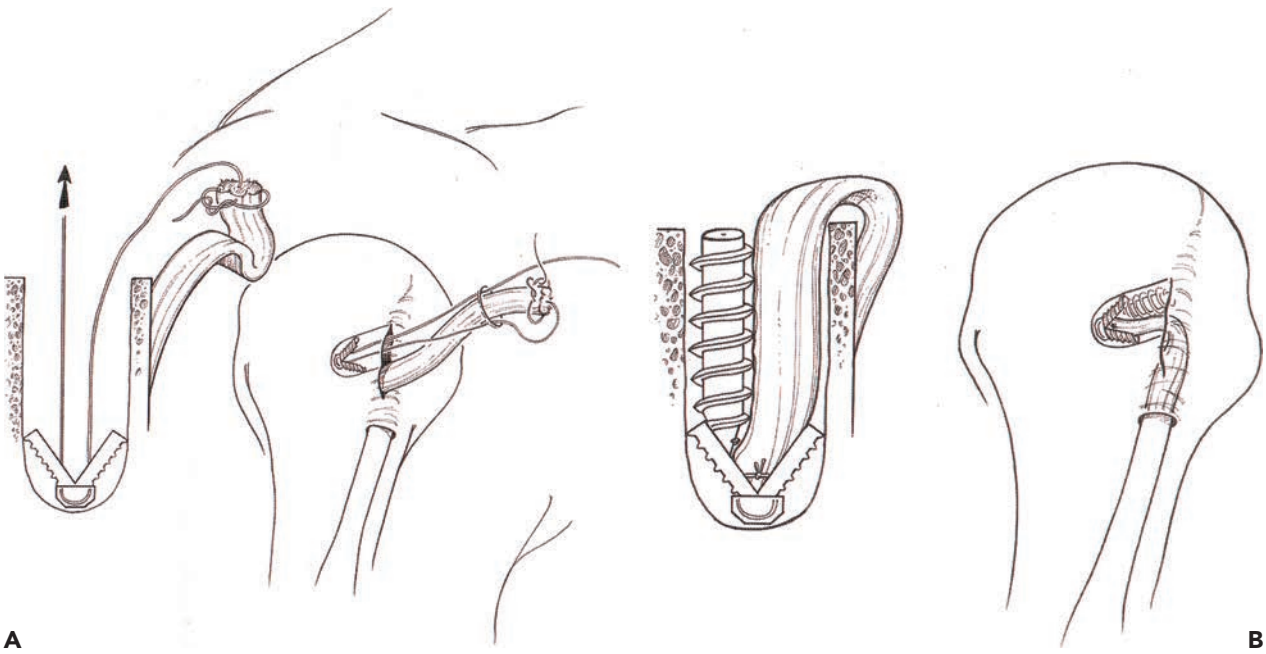

Figure 7-16 Illustration of suture anchor being used as a pulley system for interference screw tenodesis. **A.** Suture anchor at the base of the tunnel serves as a pulley to draw the tendon into the tunnel. **B.** The tendon is delivered to the base of the tunnel and secured with an interference screw. (Reprinted with permission, Kuo W, Gladstone JN, Flatow EL. Biceps tenodesis. In: Miller MD, Cole BJ, eds. *Textbook of arthroscopy.* Philadelphia: Saunders, 2004:187–201.)

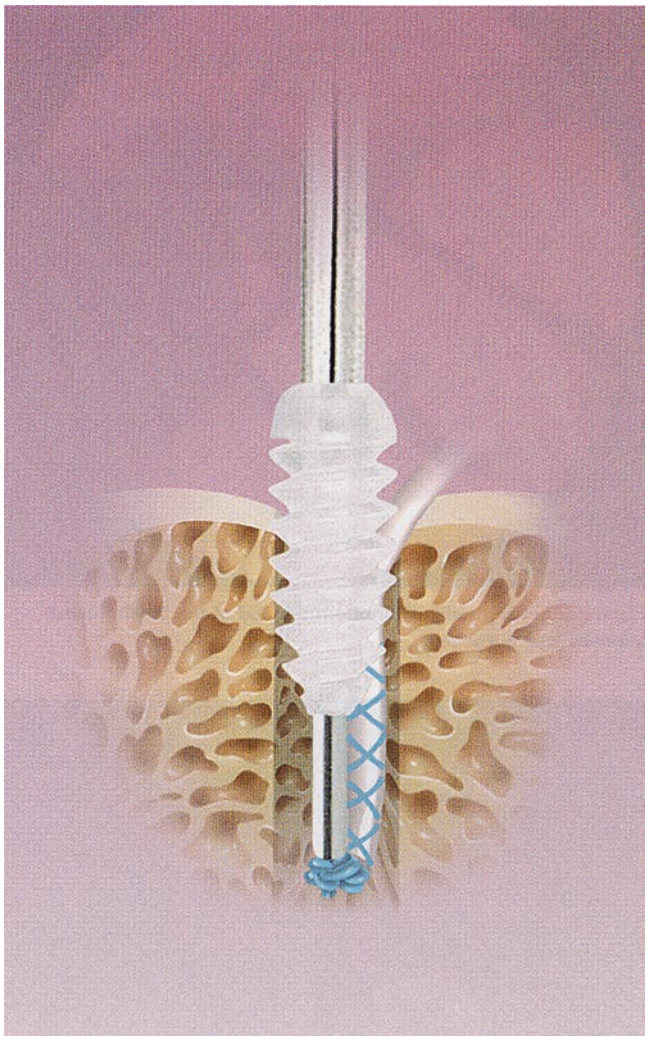

Figure 7-17 Illustration of the interference screw and biceps tendon being advanced into the tunnel. The tendon is firmly held against the tip of the driver through tension applied to one limb of the tendon whipstitch that has been delivered through the cannulated driver. (Reprinted with permission, Kuo W, Gladstone JN, Flatow EL. Biceps tenodesis. In: Miller MD, Cole BJ, eds. *Textbook of arthroscopy.* Philadelphia: Saunders, 2004:187–201.)

less soft tissue dissection than bone fixation techniques, thereby taking advantage of local tendon adhesions within the groove. Tendon scarring within the groove and proximal tendon hypertrophy may explain why some spontaneous ruptures or biceps tenotomies are not associated with significant distal retraction of the tendon stump. The primary concern with soft tissue tenodesis is limited strength of the repair. Wolf and coauthors compared the strength of simple tenotomy versus tenodesis with interference screw fixation in a cadaver model.[136] The biceps tendon was loaded in a cyclic manner with 50 N and observed for migration of the tendon distally. Load to failure was then performed in those shoulders without evidence of biceps migration. Interestingly, only 4 of the 10 tenotomized specimens failed after cyclic loading compared to none of the tenodesed specimens. The ultimate load to failure for the tenotomy group (110 N) was significantly lower than the tenodesis group (310 N).

Biceps Tenodesis Versus Tenotomy

Considerable controversy persists in the decision to perform tenodesis or tenotomy for the treatment of biceps tendon pathology. Factors that play a role in the decision making include the age and activity level of the patient and cosmetic concerns. The potential benefits of simple tenotomy must be tempered against the potential for residual biceps pain and spasm as well as a cosmetically unacceptable result. Ultimately, the decision to perform a tenodesis or tenotomy is based on the surgeon's personal preference and philosophy.

Historically, biceps tenodesis has been the preferred method of treatment for long of head biceps tendon disease. Numerous authors have reported good results following biceps tenodesis in various patient populations.[12,19,31,97,102,110] The advantages of biceps tenodesis in favor of tenotomy include improved cosmesis and potentially decreased incidence of biceps-related pain, spasm, and fatigue. These concerns are significant in younger patients and those participating in occupational and recreational activities that are demanding. Recovery following biceps tenodesis requires protection of the surgical repair. Active elbow flexion and forearm supination must be minimized while the repair is healing. In addition, biceps tenodesis increases the complexity of surgery, especially when performed arthroscopically. Residual tenderness in the biceps groove following tenodesis has been a concern as well.[75]

The presence of significant biceps muscle spasm and fatigue discomfort following tenotomy is a matter of debate. The literature regarding biceps-related pain following tenotomy is conflicting. Koening et al. reported the results of biceps tenotomy compared to tenodesis in a retrospective review of 61 patients.[75] Thirty-seven (mean age 48 years) had undergone a tenodesis and 24 (mean age 60 years) had either a tenotomy or spontaneous rupture. Follow-up ranged from 12 to 48 months. No significant differences were noted in the mean American Shoulder and Elbow Surgeons (ASES) score (74 vs. 72), pain (2.5 vs. 2.0), or activities of daily living scores (21 vs. 20) between the tenodesis and tenotomy groups. Fifty-three percent of the tenodesis group had local tenderness over the tenodesis site, but none had biceps muscle symptoms otherwise. In contrast, 68% of the biceps tenotomy and rupture patients did not like the cosmetic appearance and 72% had pain and cramping in the biceps muscle during activities requiring resisted supination and flexion of the elbow. Osbahr and coauthors reported the results of a retrospective review of 160 patients following either biceps tenotomy (mean age 58 years) or tenodesis (mean age 54 years).[103] The indications to perform tenodesis or tenotomy

were based on the age and physical demands of the patients and associated rotator cuff pathology. The average duration of follow-up was 20 months in the tenodesis group and 23 months in the tenotomy group. Patients were asked to subjectively report the degree of anterior shoulder pain, muscle spasms in the biceps, and cosmetic deformity. The authors found no significant difference between the groups with any of the measured outcomes. In addition, there were no differences in biceps-related symptoms between males and females.

In recent years, biceps tenotomy has become more popular for the treatment of patients with biceps tendon pathology, particularly older or low-demand individuals.[103,131] Potential benefits of tenotomy over tenodesis include ease of surgery and rapid postoperative recovery. The primary concern with tenotomy is the risk of residual biceps pain and spasm as well as cosmetic deformity. Numerous authors have reported successful results after biceps tenotomy combined with rotator cuff débridement in older patients with massive rotator cuff tears.[71,117,126] Walch et al. advocated release of the biceps tendon in cases of subluxation and dislocation associated with rotator cuff tear.[131]

Gill and coauthors reported good results in a group of relatively young patients following isolated biceps tenotomy.[51] Thirty patients with a mean age of 50 years were followed for an average of 19 months after surgery. All patients underwent arthroscopic biceps tenotomy for the treatment of biceps tendon tenosynovitis, partial rupture, and dislocation. Two patients underwent a concomitant arthroscopic subacromial decompression. The mean postoperative ASES was 81.8. Ninety-seven percent of patients returned to their previous occupation and 87% of patients were satisfied with the procedure. Four (13.3%) of the patients had poor results: one patient with an unacceptable cosmetic deformity, two cases of residual subacromial impingement, and one patient with residual shoulder pain.

Isolated biceps tenotomy is also effective when combined with other shoulder procedures in young patients. Kelly and coauthors reported the results of 40 of 54 patients with anterior shoulder pain treated with biceps tenotomy.[70] Only 9 of the 40 tenotomies were performed as isolated procedures. The mean patient age was 48 years. Seventy percent of patients were noted to have a Popeye sign on examination. The mean postoperative ASES score was 78. Sixty-eight percent of patients rated themselves as good or excellent. There were 7 of 40 self-rated poor results, all of which had concomitant procedures: five high-grade osteoarthrosis débridements, two rotator cuff repairs, and two acromioplasties. Overall, 95% of patients reported relief of tenderness on palpation of the biceps groove. However, 37.5% (13 patients) reported fatigue discomfort (soreness) isolated to the biceps muscle. The presence of residual biceps symptoms correlated with younger age. Seven of 11 patients under the age of 40 years were noted to have residual biceps symptoms following tenotomy compared to 8 of 19 between the ages of 40 to 60 years. No patients older than age 60 were noted to have residual biceps-related symptoms.

Treatment of Biceps Instability

Similar to biceps tendinitis, rotator cuff pathology is usually coexistent with tendon instability. Contrary to surgical treatment of tendinitis alone, sacrifice of the biceps tendon is the procedure of choice with biceps tendon subluxation or dislocation.[35,131] The rationale of tenotomy or tenodesis depends on the factors previously discussed. Biceps instability, both subluxation and dislocation, is known to occur in a variety of patient populations. However, a commonality to these patients is the presence of a rotator cuff tear, usually involving the subscapularis.[35,56,107,131] Those with frank biceps tendon dislocation have a high likelihood of both a full-thickness tear of the subscapularis tendon as well as the posterior cuff.[131] Walch et al. advocated biceps tenotomy for biceps instability associated with rotator cuff tear.[131]

O'Donoghue performed tenodesis of the biceps tendon in the groove by the technique of Hitchcock as a treatment for unstable biceps tendon in a young population involved in athletic pursuits.[102] Fifty-six operations were carried out in 53 patients with an age range of 15 to 35. Seventy-seven percent could throw satisfactorily and resumed sports. Edwards et al. reported the results of 84 patients following repair of a subscapularis tendon tear.[35] The mean age of the patients was 53 years. Fifty-four shoulders were noted to have medial subluxation or dislocation of the biceps tendon and 10 had complete rupture of the tendon. Forty-eight shoulders underwent concomitant tenodesis of the biceps tendon, while another 13 shoulders underwent concomitant tenotomy. The mean Constant score improved from 55 preoperatively to 79.5 postoperatively. Tenodesis or tenotomy of the biceps tendon at the time of subscapularis repair was associated with improved subjective and objective results, independent of the preoperative condition of the biceps tendon.

More recently, biceps instability concerns seen in the context of rotator interval lesions have been treated with attempted reconstruction of the coracohumeral ligament and rotator cuff tear.[132] In a series of 14 shoulders with subluxated biceps tendons an attempt at reinsertion of the tendon and repair of torn structures resulted in secondary rupture of the biceps tendon in 25%. When rupture is not seen there can still be concerns with autotenodesis with this operative strategy. Bennett attempted arthroscopic repair of the biceps sling in a series of 18 patients with interval lesions and partial-thickness rotator cuff tears, and otherwise healthy appearing biceps tendons. Improvement in shoulder pain and function was noted in the majority of patients.[11] One patient suffered rupture of the biceps tendon and two others had recurrence of biceps-related pain.

Treatment of Long Head of the Biceps Ruptures

Spontaneous or traumatic ruptures of the long head of the biceps do not generally require operative intervention.[23] While little information is available about operative treatment of long head ruptures, one study showed a 21% supination and 8% elbow flexion strength deficit.[89] Pronation, grip, and elbow extension strength were normal. When comparing operative versus nonoperative patient groups in 27 patients, residual pain was uncommon for both and nonoperative patients returned to work faster.

Treatment of Superior Labral Disorders (SLAP Lesions without Glenohumeral Instability)

Since the initial description of superior labral lesions, associated with the biceps origin by Snyder, treatment has generally been dependent on the type of pathology encountered.[32,121] Seen at most around 5% to 6% of the time, SLAP lesions without glenohumeral instability have been divided into four types as previously described.[122] Treatment options for these various types of SLAP lesions include simple débridement, suture repair, or tenodesis. In general, the type of treatment has been dependent on the type of pathology encountered.

Snyder, in his original description of SLAP lesions, described treatment for type I lesions as being simple débridement.[122] In type II lesions, the superior labrum and biceps tendon anchor was débrided at the bone–labral junction to obtain a bleeding base. Initially, further stabilization with a screw or suture was not performed secondary to an absence of available techniques. Type III lesions were treated with an excision of the bucket-handle portion of the tear. In type IV lesions, the torn portion of the biceps tendon and labrum were débrided. If more than 50% of the tendon was torn, then a tenodesis was performed. The short-term results were considered promising with 88% considered good or excellent. In a follow-up report by these same authors of 140 SLAP lesions treated arthroscopically, 21% were type I, 55% were type II, 10% were type III, and 5% were type IV.[120] Treatment included débridement alone for type I; débridement with glenoid abrasion alone for half of the type IIs and débridement with repair for the other half; débridement for type III; and débridement for type IV in half and repair in half. Repeat arthroscopy in 18 of the shoulders showed that three of five type II lesions treated with débridement alone had healed in comparison to four of five treated with repair. Type IV lesions that had been repaired all appeared healed on repeat arthroscopy. Additional data and outcome were unavailable.

Yoneda et al. described the use of an arthroscopically placed staple for type II lesions.[140] They performed the procedure in 10 young athletes and had a good or excellent result in eight. The two failures were thought to be secondary to persistent subacromial bursitis and multidirectional instability.

Field and Savoie prospectively treated 20 consecutive patients with type II and IV SLAP lesions of the shoulder with arthroscopic suture repair.[39] Sixteen patients had a history of a significant injury, the commonest mechanism being fall on the outstretched abducted arm. Arthroscopy revealed detachment of the superior labrum–biceps tendon anchor. The superior glenohumeral ligament was connected to the unstable fragment. A large number of patients had additional pathology including partial rotator cuff tears (eight cases), impingement syndrome (five cases), and acromioclavicular arthritis (three patients), which were addressed at the same time. All patients underwent arthroscopic débridement and reattachment of the lesion using transglenoid sutures. At an average follow-up of 21 months, the authors reported excellent results in 80% and good results in 20%. While the scores for motion, strength, and stability were improved after surgery, the increase in the pain and function scores was statistically significant.

Authors' Preferred Treatment—Surgical Management

Arthroscopic Evaluation

In the treatment of biceps tendon pathology, the initial treatment is generally arthroscopic. Arthroscopy provides a valuable tool to accurately visualize both intraarticular and intertubercular biceps pathology as well as any associated disorders such as rotator cuff tears. As we perform arthroscopy in the beach-chair position, open approaches can be easily conducted during the same sitting. Beach-chair arthroscopy is performed on a standard operating table. The patient is positioned as far lateral on the table as possible using a universal joint head holder and lateral pad to secure the patient safely. Arthroscopy is then initially performed in the glenohumeral joint through the standard posterior portal. The rotator interval is easily identified within a triangle demarcated by the biceps tendon superiorly, the subscapularis inferiorly, and the glenoid medially (Fig. 7-18). An anterior portal is established in an outside-in fashion by first placing a spinal needle just lateral to the coracoid tip. Needle placement is verified intraarticularly with arthroscopic visualization. A cannula is then placed with a sharp plastic trocar from outside to in. A standard diagnostic arthroscopy is performed with the arm under slight in-line traction. The articular surfaces of both the humerus and glenoid are thoroughly evaluated. The anterior glenoid labrum is visualized from the midportion of the glenoid to inferior observing for labral fraying or detachment that may suggest instability. Glenohumeral ligaments, particularly the inferior glenohumeral ligament, are then inspected. The scope is then brought to the rotator interval, the superior subscapularis muscle, and the tendon.

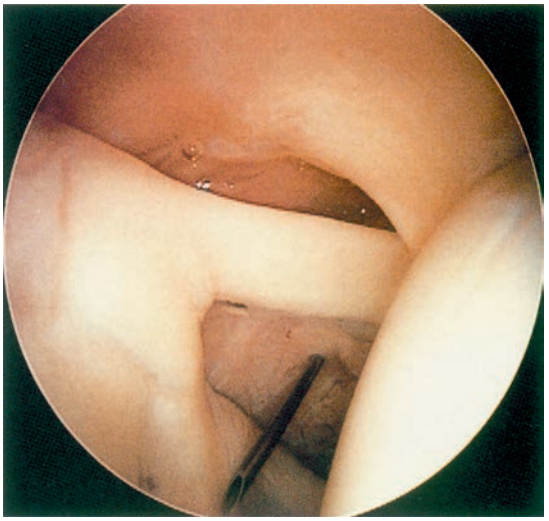

Figure 7-18 An arthroscopic view showing the rotator interval. A needle is placed just lateral to the coracoid under arthroscopic visualization to verify the location of the anterior portal.

From this position, the 30-degree scope is pointed laterally out toward the intersection of the biceps tendon and subscapularis (Fig. 7-19). In this fashion, the entire portion of the superior subscapularis is inspected. Internal rotation of the shoulder improves visualization of the subscapularis insertion. The scope is then brought posterior to the biceps tendon over the top of the humeral head to visualize the supra, infra, and teres minor insertions and the bare area of the humeral head. Following this, the scope is then brought up through the posterior aspect glenoid, inspecting for any posterior labral defects or any posterior–superior inflammation suggestive of internal

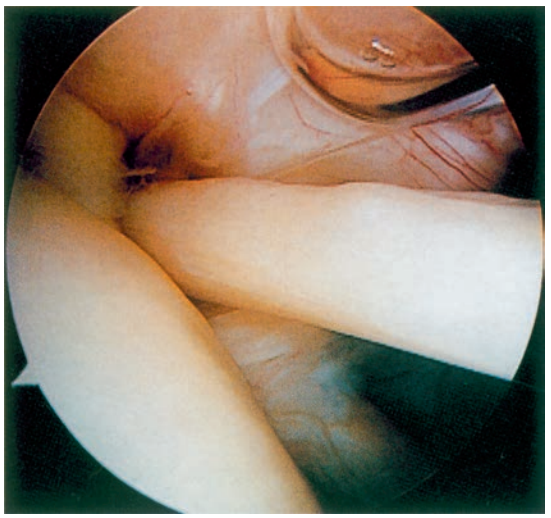

Figure 7-19 Arthroscopic view of the entrance to the bicipital groove. The intersection of the subscapularis and supraspinatus with the biceps tendon can be seen at the proximal portion of the bicipital groove. Pathology in this location may indicate a disruption of the rotator interval sling.

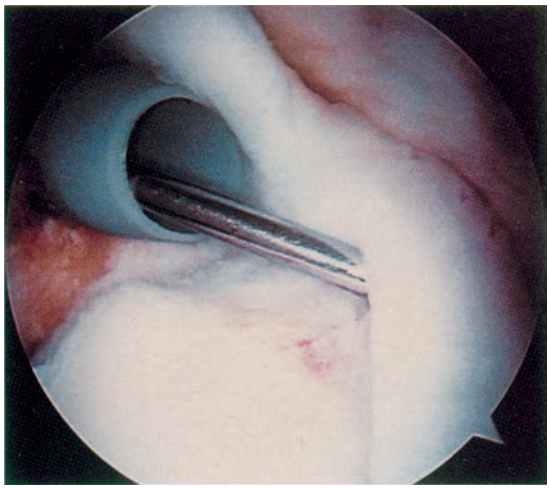

Figure 7-20 An arthroscopic view of a type II superior labral anterior and posterior (SLAP) lesion. A probe is placed under the anterior superior labrum to detect any detachment of the biceps origin. The probe is placed in from the anterior portal. Notice that easy access for abrasion of the glenoid rim can be achieved from this same portal.

impingement. The biceps tendon is then inspected at length from its superior labral attachment laterally to the bicipital groove. Under normal conditions, the entrance of the biceps tendon into the groove distally can easily be visualized. The probe is then placed anteriorly under the anterior–superior labrum and the origin of the biceps examined for any detachment suggestive of a superior labral lesion (Fig. 7-20). The probe is then placed over the top of the biceps tendon to pull the tendon farther into the joint to inspect the portion normally within the intraarticular groove (Fig. 7-21). The humeral head is brought from a position of external rotation into internal rotation to observe whether the biceps tendon is subluxated from the bicipital groove.

Treatment of Bicipital Tendinitis

We determine appropriate surgical treatment for the long head of the biceps tendon according to observations made during surgery and the preoperative physical examination findings. We feel that the biceps tendon can be a significant source of shoulder pain if not specifically addressed during a surgical procedure. This is based in part on multiple observations of patients who had spontaneous resolution of shoulder pain upon rupture of the long head. Additionally, as previously described in other studies, we have seen patients who have failed subacromial decompressions for chronic rotator cuff tendinitis or rotator cuff tears who later can achieve satisfactory pain relief from a revision surgery in which a biceps tenodesis is performed. However, we do not believe that routine biceps tenodesis is indicated. Given the symptomatic concerns of a significant biceps tendon disorder, we also do not believe that a policy

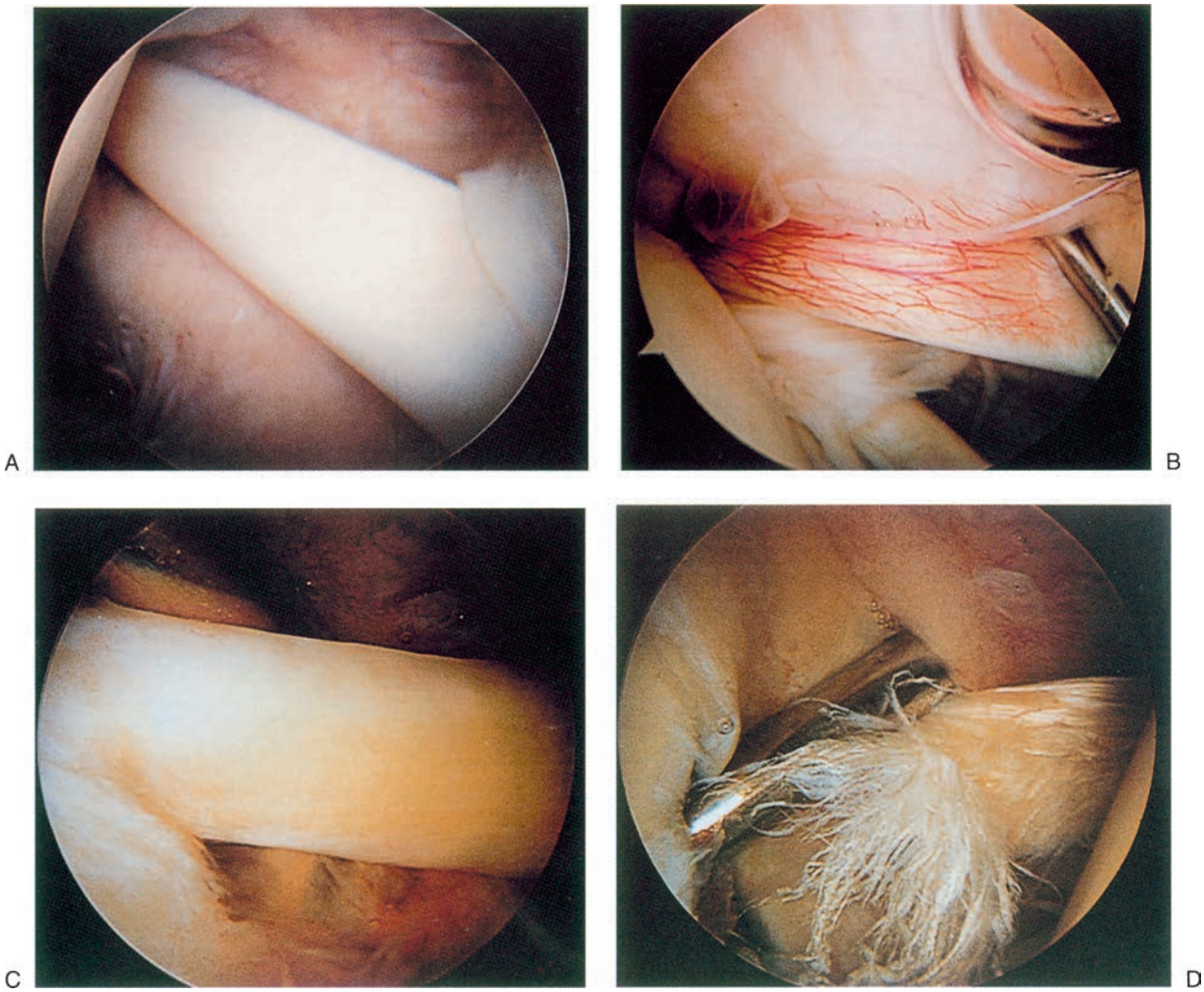

Figure 7-21 Inspection of the biceps tendon must include the intertubercular groove portion. This is inspected by placing a probe over the top of the biceps tendon and drawing the intertubercular groove portion into the joint. **A.** Shows a normal-appearing biceps tendon. **B.** When the intertubercular groove portion is drawn into the joint with a probe, a marked amount of synovitis is recognized. **C.** Again, the biceps tendon appears to be normal in the intraarticular portion. **D.** When a probe is used to draw in the intertubercular groove portion, a significant partial tear is detected.

of avoidance of tenodesis whenever possible is appropriate. Rather, avoidance of tenodesis is employed whenever it is felt that the inflammatory changes to the biceps tendon are reversible. In general, the criteria for a biceps tendon with reversible changes include less than 25% partial tearing from a normal-width tendon, a normally located tendon within the bicipital groove, and normal tendon size regardless of associated synovitis. Those surgical observations consistent with a biceps tendon with irreversible changes include partial-thickness tearing or fraying of the tendon greater than 25% of the normal width of the tendon, any luxation of the biceps tendon from the bicipital groove, any disruption of the associated bony or ligamentous anatomy of the bicipital groove that would make autotenodesis likely, and any significant reduction or atrophy in the size of the tendon greater than 25% of the normal width of the tendon (Fig. 7-22). Relative indications for sacrificing the biceps also include any biceps pathology in the context of a failed acromioplasty or any significant biceps pathology in the context of a SLAP lesion.

Inflammation of the biceps tendon or involvement of the biceps tendon within the marked inflammation seen with rotator cuff disease is treated in the standard fashion for rotator cuff disease alone. It is generally anticipated that adequate treatment of a rotator cuff tendinitis will resolve concurrent bicipital tendonitis. Generally, a florid subscapular synovitis is present, descending from the rotator interval to encompass the anterior–superior and posterior–superior labrum and bicipital groove portion of the biceps tendon (see Fig. 7-8). We have found a wide dispersion cautery device, such as the multifilament bipolar device (Arthrocare), quite useful in ablating the synovitis. The cautery

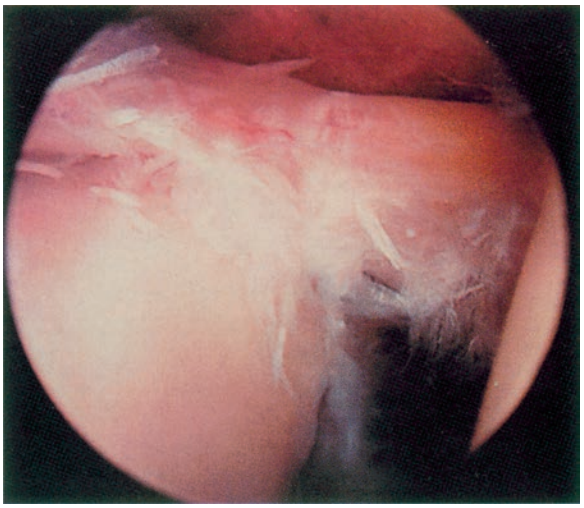

Figure 7-22 Arthroscopic view of a biceps tendon with greater than 25% fraying in the width of the tendon. In this situation, significant, chronic, or posttraumatic changes have occurred, which makes healing of the tendon less likely and concerns about persistent pain more significant. This is considered an indication for tenodesis.

device is used to vaporize the red, inflamed tissues on the undersurface of the rotator cuff without any penetration through the capsule to the cuff itself. No attempt is made to débride the inflamed synovium overlying the bicipital groove portion of the long head of the biceps.

If sufficient biceps pathology exists to warrant sacrifice of the tendon, the next decision in the treatment algorithm is biceps tenodesis versus tenotomy (Fig. 7-23). Preoperatively, we discuss the potential need to perform a biceps-related procedure in all patients undergoing surgery for the treatment of rotator cuff tendonitis or cuff tear. Specifically, the risks of cosmetic deformity and residual biceps spasm following tenotomy alone and the necessary biceps precautions observed with tenodesis are discussed. Review of patient preferences and concerns by the clinician as well as improved patient education play important roles in achieving a good outcome following surgery.

We prefer to perform a biceps tenodesis in younger patients (under 60 years); in those who remain physically active, at work or with recreation; and when cosmesis is a significant concern. When a biceps tenodesis is indicated, based on the above criteria, an arthroscopic-assisted approach is generally employed. In this technique, the biceps tendon is first tagged with a #1 polydioxanone (PDS) suture. This is accomplished by placing a spinal needle anterolaterally through the rotator interval. Under arthroscopic guidance, the needle is placed through the substance of the tendon near the bicipital groove entrance (Fig. 7-24). A #1 PDS suture is then threaded through the 18-gauge spinal needle into the joint. A suture grasper is inserted and the suture brought out through the anterior portal. In this fashion, the suture prevents any excursion of the long head of the biceps tendon down the arm. Once the biceps tendon is secured with the suture, the origin is released with either electrocautery or an arthroscopic scissor. Generally, after release of the origin, the excursion of the tendon is only 1 to 2 cm. The stay suture is only a preventive measure as the biceps tendon is wider at the base and generally will not travel down the bicipital groove unless atrophic changes are present.

Our preferred method of tenodesis employs bony fixation of the tendon under the pectoralis major tendon. We reserve this technique for young patients that are physically active. A subpectoral tenodesis is advantageous because the tenodesis site is well covered by soft tissue in this location. A 4-cm incision is placed in the axilla, centered on the inferior border of the pectoralis major tendon. The majority of the incision should be in the axilla, when the arm is at the side. This incision made along this line is quite cosmetic and for the most part not visible. Subcutaneous dissection is then taken to the deltoid pectoral interval, and this is then followed to the bicipital groove. The inferior portion of the transverse humeral ligament is then divided in line with the bicipital groove. The tendon is then visualized and drawn out of the groove using a curved clamp. The tendon is shortened 2 to 3 cm inferior to the PDS suture to maintain appropriate tension within the biceps. A tendon-grasping stitch is woven through the biceps with a heavy nonabsorbable suture. Soft tissues are dissected from the bicipital groove under the upper margin of the pectoralis tendon insertion. Fixation is accomplished using a bioabsorbable interference screw (Arthrex, Naples, FL). A tunnel is drilled with a cannulated reamer over a pin that has been placed in the desired location within the bicipital groove. The tendon and interference screw are then advanced into the tunnel using a cannulated tenodesis screwdriver. The two limbs of the tendon suture are then tied over the screw, further securing the biceps tendon.

If a mini-open rotator cuff repair is being performed, then tenodesis is accomplished through the mini-open deltoid splitting approach. The arm is externally rotated at the side to bring the bicipital groove out laterally underneath the deltoid split. The transverse humeral ligament is then divided in line with the groove. The biceps tendon is then delivered out through the deltoid split with the PDS suture in place (Fig. 7-25). The floor of the groove is then roughened with a curette. The length of the tendon in the tenodesis is determined by the stay suture, which was placed close to the insertion of the biceps tendon in the groove. The tenodesis is accomplished with an interference screw as previously described. The transverse humeral ligament is then sutured on top of the secured tendon. The excess intraarticular portion of the tendon is then excised.

An alternative technique of biceps tenodesis can be performed using soft tissue fixation alone. This technique is performed arthroscopically and is technically easier and faster than interference screw fixation. Because the biceps tendon is not dissected, adhesions within the bicipital

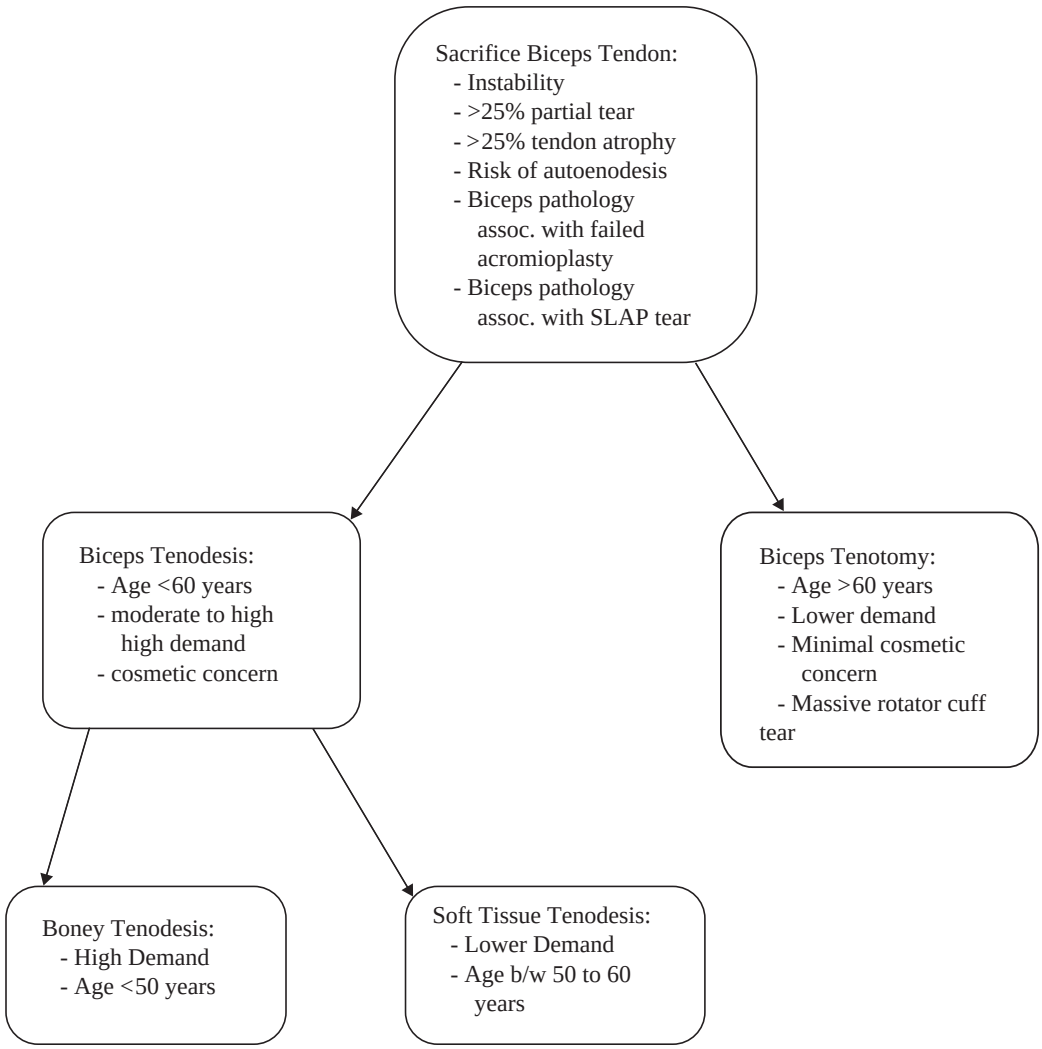

Figure 7-23 Algorithm for the treatment of biceps tendon pathology: tenodesis versus tenotomy.

groove may contribute to the strength of the tenodesis. We reserve this method of fixation in cases where tenodesis is preferred but the patient is less physically active. The tendon is tagged with multiple #1 PDS sutures through a percutaneous spinal needle at the apex of the bicipital groove. The biceps tendon is released from the labrum and transected just proximal to the tagging suture. The PDS sutures are then brought out through the anterior cannula and used to shuttle nonabsorbable sutures through the tendon in a closed-loop fashion. The arthroscope is directed into the subacromial space. After subacromial débridement, the nonabsorbable sutures are then identified and retrieved through the anterior–lateral cannula. The tendon is then secured to the transverse humeral ligament with an arthroscopic knot.

Tenotomy is the preferred method of treatment of biceps tendon pathology in older (older than 60 years) patients who are low demand. Tenotomy is often performed in the context of massive rotator cuff repair or débridement. Patients are counseled preoperatively regarding cosmetic deformity, which is often minimal in this group, as well as the possibility of residual biceps muscle spasm or pain. Tenotomy is accomplished arthroscopically by releasing the attachments to the superior labrum and glenoid. The tendon retracts 1 to 2 cm after release but usually remains visible within the joint. Excessive residual biceps tendon can be débrided if necessary.

Treatment of SLAP Lesions

We prefer the lateral decubitus arthroscopy position for all procedures that are primarily intraarticular. The patient is positioned with the affected side up and supported by a deflated bean bag. The patient is generally placed slightly diagonal on the table such that the affected shoulder is closest to the side of the table where the surgeon will be standing. Additionally, the head of the patient is generally as close to the top of the table as possible to allow the surgeon to access the shoulder from above. The patient is then

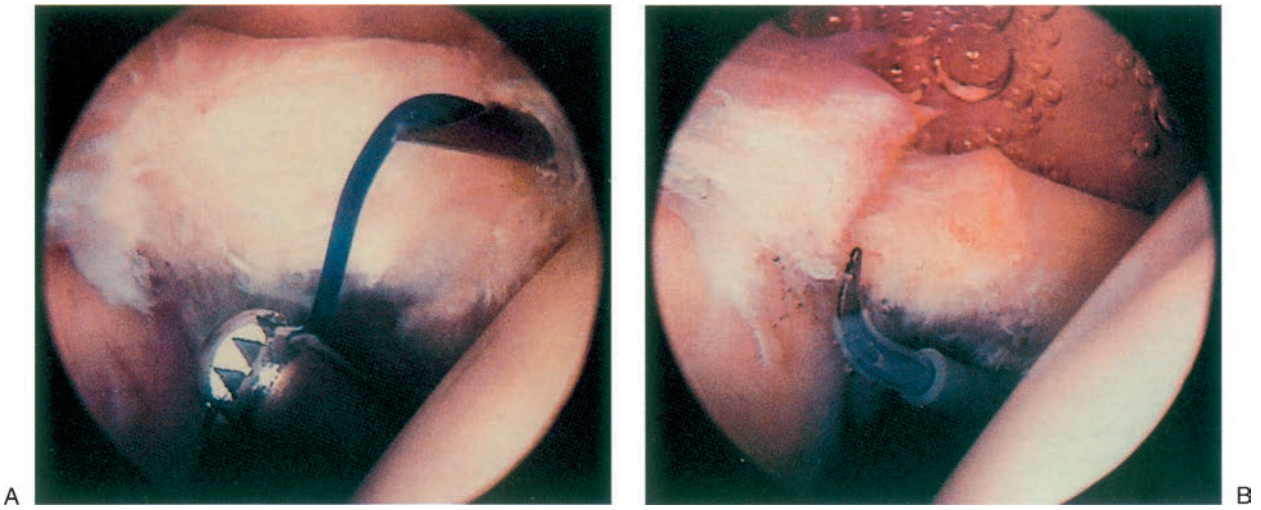

Figure 7-24 Arthroscopic views from a biceps tenodesis. **A.** A spinal needle is used to insert a #2 polydioxanone (PDS) suture into the biceps tendon near its entrance to the bicipital groove. This suture is then grasped and then brought out through the anterior portal. **B.** The origin is then released with an electrocautery device.

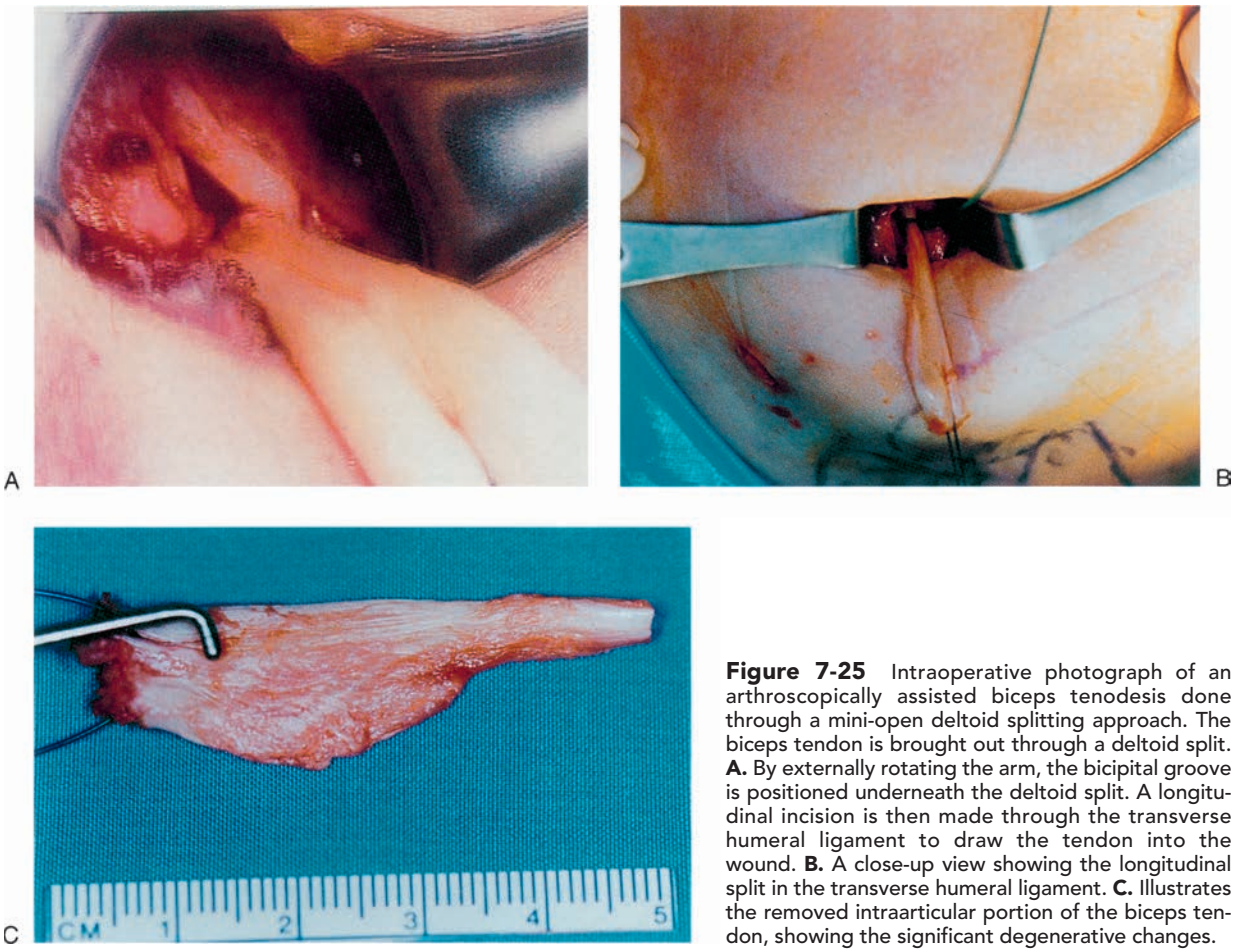

Figure 7-25 Intraoperative photograph of an arthroscopically assisted biceps tenodesis done through a mini-open deltoid splitting approach. The biceps tendon is brought out through a deltoid split. **A.** By externally rotating the arm, the bicipital groove is positioned underneath the deltoid split. A longitudinal incision is then made through the transverse humeral ligament to draw the tendon into the wound. **B.** A close-up view showing the longitudinal split in the transverse humeral ligament. **C.** Illustrates the removed intraarticular portion of the biceps tendon, showing the significant degenerative changes.

generally rotated approximately 30 degrees toward posterior to level the glenohumeral joint parallel to the floor.

Prior to prepping and draping, an examination under anesthesia is performed. Load-and-shift maneuvers are performed in both the anterior and posterior directions. The most consistent finding seen with labral abnormalities is a popping or clicking sensation as a humeral head luxates to the glenoid rim. Any associated laxity should be noted and considered as a potential source of symptomatology.

After routine prep and drape, the arm is then positioned in a lateral traction boom. We prefer a traction boom that allows both inline traction of the arm and lateral distracting traction to open up the glenohumeral joint (Arthrex, Naples, FL). This boom places the arm in foam padding for skin traction inline along the long axis of the upper extremity. It also has a second attachment that goes underneath the arm to provide a lateral distracting force. Generally, 15 lb of traction is employed and a lateral distracting force is applied equivalent to distracting force obtained when an assistant lifts the arm at the level of the axilla.

The glenohumeral joint is then located with a spinal needle and infiltrated with 10 mL of 0.25% Marcaine containing epinephrine. All bony landmarks are carefully outlined and portal sites drawn. Generally, four portal sites are used for SLAP repair. A standard posterior portal is employed approximately 2 to 3 cm inferior to the posterior border of the acromion. It is generally best to err this portal slightly laterally. The next two anterior portals are drawn, one lateral to the coracoid process and one just anterior to the acromion approximately 5 mm lateral to the acromioclavicular joint. More experienced surgeons may be comfortable doing SLAP repairs through a single anterior portal placed lateral to the coracoid process. Finally, a lateral portal just distal to the anterior lateral corner of the acromion is employed for anchor insertion. We generally do not prefer placement of a formal portal in this location. Alternatively, this location is used as a starting point for percutaneous entrance of a drill guide for anchor insertion. A cannula is not placed to avoid damage to the rotator cuff tendon or muscle.

Glenohumeral arthroscopy is initiated from posterior. After visualization is established from the posterior portal, the anterior portals are then established under direct visualization from outside to in. We prefer the use of guidewire and a cannulated portal expanding system (Arthrex, Naples, FL). The use of guidewire allows precise placement of portals, which are important for verifying utility of each of the portal sites for both suture passing and glenoid access. After establishment of the two anterior portals and posterior portal, a glenohumeral inspection is performed (Fig. 7-26). Treatment of superior–labral lesions of the biceps is generally done according to the recommendations of Snyder et al. Type I lesions are seen as significant fraying about the superior labrum and are débrided with a shaver through the anterior portal. No further treatment is necessary. Type II lesions in which the biceps origin and superior labrum are detached from the bony base of the glenoid are treated by arthroscopic repair. Care should be taken to properly identify a type II lesion as opposed to an extensive fraying seen with a type I lesion. The anatomy of the biceps and labral origin should be well recognized. It should be noted that articular cartilage generally overhangs the articular face of the glenoid, and because of this there will be a 2- to 3-mm area of detachment of the labrum laterally, which is normal. Medial detachment of the labrum more than 4 mm and fraying of the tissues on the undersurface of the labrum and biceps tendon anchor are abnormal. Type III lesions can be treated by débridement of the bucket handle tear alone if the remaining labrum is still firmly attached. Type IV lesions, which include extension into the substance of a biceps tendon, generally require both a repair of the labrum and a tenodesis/tenotomy of the long head of the biceps tendon. Type V lesions require superior labral repair, which then also encompasses normal anterior–inferior instability repair techniques.

In performing a repair of the labrum, a shaver is inserted from the anterior portal and the area of tear débrided fully to expose some bleeding bone and remove degenerated tissues. Once débridement is performed in the area of the tear, the percutaneous portal distal to the anterior lateral corner of the acromion is established for suture anchor placement (Fig. 7-26). The portal is generally performed approximately 1 to 2 cm distal to the anterior–lateral corner of the acromion for superior and anterior superior tears. A suture anchor placed in this location through a percutaneous drill guide will penetrate through the muscle belly of the supraspinatus, but not the tendon. The angle will give you good access to suture anchor placement around the biceps origin from 2 cm posterior to 1 cm anterior to this location. If further posterior anchors are required, a second portal may be required, which is 1 to 2 cm posterior to the previous portal and is near the posterior–lateral corner of the acromion. We generally like to establish this portal by first placing the percutaneous guide pin to verify accurate location. At this point, the guide pin can be removed and a drill guide inserted through a percutaneous technique using a trocar (Arthrex, Naples, FL). Alternatively, the guide pin can be left in place and a "mini-portal" formed by expanding over the guidewire as if forming the anterior portals. The drill guide can then be passed over the guidewire to expand tissues. In any case, at this point, all required suture anchors are generally placed into the superior–posterior glenoid. The average SLAP repair requires a minimum of two suture anchors. We prefer a bioabsorbable implant containing nonabsorbable high-strength sutures (Arthrex, Naples, FL). Once suture anchors are inserted into superior glenoid shuttle, sutures are then placed into the superior labrum to deliver one of the two suture strands from each anchor around the labrum. Generally, we employ the use of a suture-passing angled hook (Spectrum Instrument, Linvotec, FL). A zero

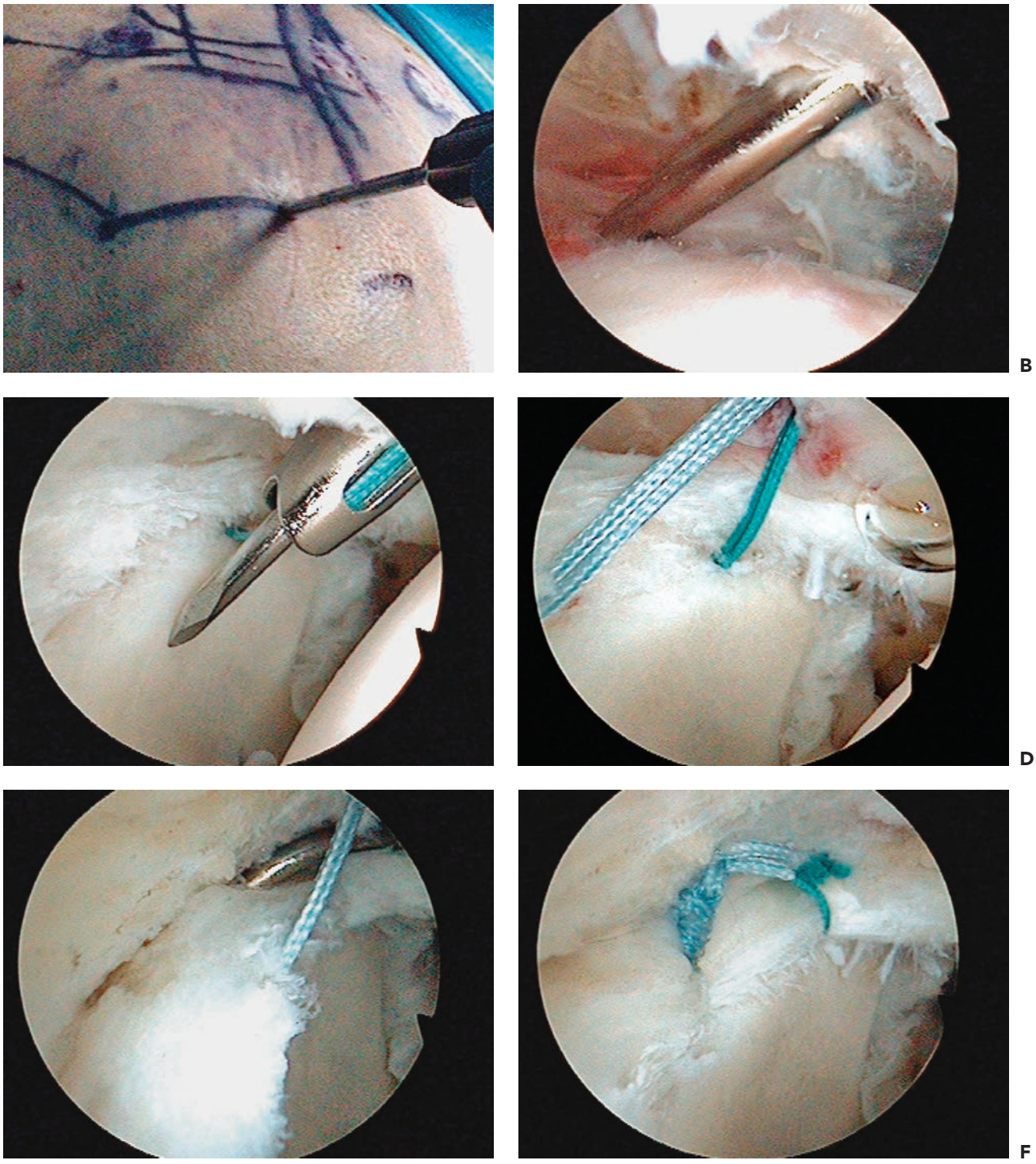

Figure 7-26 Intraoperative photographs from an arthroscopic repair of a superior labral anterior and posterior (SLAP) lesion. **A.** The superior–lateral portal is made just lateral to the anterior–lateral acromion and directed diagonally toward the glenoid. **B.** Penetration of the rotator cuff to enter the joint from this portal is through the muscle belly of the supraspinatus and not in the rotator cuff tendon that is more lateral. **C.** A drill guide is then inserted over the guidewire to maintain proper orientation for suture anchor insertion. **D.** Usually, a minimum of two suture anchors are required to fix most superior labrum tears. Both anchors are inserted prior to any suture passing. **E.** An angled, curved suture passing device is then inserted into the labrum from medial to lateral for shuttling of a single limb of suture from each of the anchors into the labrum. **F.** Standard knot tying is performed superior to the labrum. An effort should be made to keep the knot away from the articular surface.

Prolene stitch is delivered through the labrum and then used to shuttle a suture limb around the labrum. Knot tying is then performed from the anterior–superior portal.

Postoperative Rehabilitation

Postoperative rehabilitation for surgical treatment of the biceps tendon follows guidelines established for surgical treatment of the rotator cuff. Early range of motion is instituted in a passive to active assisted fashion. When a biceps tenodesis is performed, any resisted active motion of the elbow, either in flexion or supination, is avoided. This restriction remains for 6 weeks.

For patients with SLAP repairs, the arm is usually immobilized for 6 weeks in a sling. Passive rotation at the side is allowed as well as forward elevation to 90 degrees during the first 6 weeks. After 6 weeks, progressive active assisted elevation is performed with pulleys, and therapy until full elevation is achieved. Rotator cuff strengthening is started at 12 weeks.

Complications

Few complications have been recognized specific to surgical treatment of the long head of the biceps tendon. The primary complication noted has been spontaneous rupture and shortening of the lateral biceps. Although this may result in a significant cosmetic defect in the brachium, the functional effects of this are minimal. Thus, an intolerable cosmetic defect is the primary indication for revision surgical intervention. A rare case of heterotopic ossification has also been reported.

There are theoretical complications associated with arthroscopic repair or SLAP lesions. These include injury to the suprascapular nerve, which resides 1.6 cm medial to the superior glenoid labrum. Additionally, hardware such as suture anchors placed in this location may fail or loosen, resulting in intraarticular hardware. To date, these complications have not been reported in the literature but are a concern and have been recognized in clinical practice.

SUMMARY

Without a clear understanding of the functional role for the long head of the biceps tendon, treatment recommendations have been subject to controversy. An objective review of the available information would suggest that some humeral head stability may be imparted through the tendon. However, the magnitude of this function is likely to be small and possibly insignificant. In contrast, the symptomatic significance of the long head of the biceps is less controversial. It clearly can be an important source of shoulder pain that may remain persistent when not specifically addressed with either nonoperative or operative treatment. When present, persistent pain from the long head of the biceps is likely to have more negative functional consequences than loss of the tendon itself. Given these concerns, evaluation and treatment of patients with long head of the biceps disorders should be individualized based on the likelihood that biceps-related pain will resolve. Tenodesis is recommended in the context of irreversible structural changes such as atrophy, partial tearing of the tendon width, any luxation out of the bicipital groove, or surrounding bony abnormalities that make autotenodesis likely. When structural changes are not present, the tendon inflammation is likely to resolve and routine tenodesis is not supported.

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8 Traumatic Muscle Ruptures

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INTRODUCTION

Traumatic muscle ruptures involving the shoulder girdle are relatively uncommon. Excluding the rotator cuff and biceps, which are discussed in other sections of this text, the pectoralis major and triceps tendons are the most prone to avulsion. Traumatic injuries to the deltoid, trapezius, latissimus dorsi, teres major, coracobrachialis, and short head of the biceps are unusual but are also discussed.

Muscle injuries may vary in severity from minor strains to complete disruption of muscle fibers. These injuries involve different locations within the musculotendinous unit, including the site of origin, within the muscle belly itself, at the musculotendinous junction, and complete tendinous avulsion from bone. Clinical factors, such as mechanism of injury, rate of loading, and the specific anatomic features of a given muscle, determine the site of injury.[76,121] It has been shown experimentally that normal tendons are the strongest link of the muscle–tendon–bone construct.[76] Direct trauma to a contracted muscle generally causes disruption of fibers within the muscle belly.[45,75,113] Indirect trauma from which the affected extremity is subjected to an overwhelming force against a maximally

contracted muscle usually results in distal tendon avulsion from the bony insertion.[31]

Although traumatic ruptures of the shoulder musculature are uncommon, the physician's heightened awareness and understanding of the pathomechanics, combined with a careful clinical evaluation and judicious use of imaging studies, will lead to prompt diagnosis and optimal management.

PECTORALIS MAJOR RUPTURES

Surgical Anatomy and Biomechanics

The pectoralis major is a thick triangular muscle, with a broad origin including the medial clavicle, anterior surface of the sternum, costal cartilages down to the sixth rib, and aponeurosis of the external oblique muscle of the abdomen.[48] The muscle converges laterally to form two distinct heads: the smaller clavicular head and the larger sternocostal head (Fig. 8-1). The architecture of the tendinous insertion consists of two laminae that cross over the long head of the biceps and insert on the lateral edge of the bicipital groove. The upper clavicular head constitutes the anterior lamina, whereas the lower and deeper fibers of the sternocostal head form the posterior lamina. The sternocostal head spirals on itself approximately 180 degrees, inserting proximally underneath the clavicular portion. This rolled edge creates the anterior axillary fold. Kretzler and Richardson found that the actual pectoralis tendon was about 1 cm long on its anterior surface and 2.5 cm long on its posterior surface.[57]

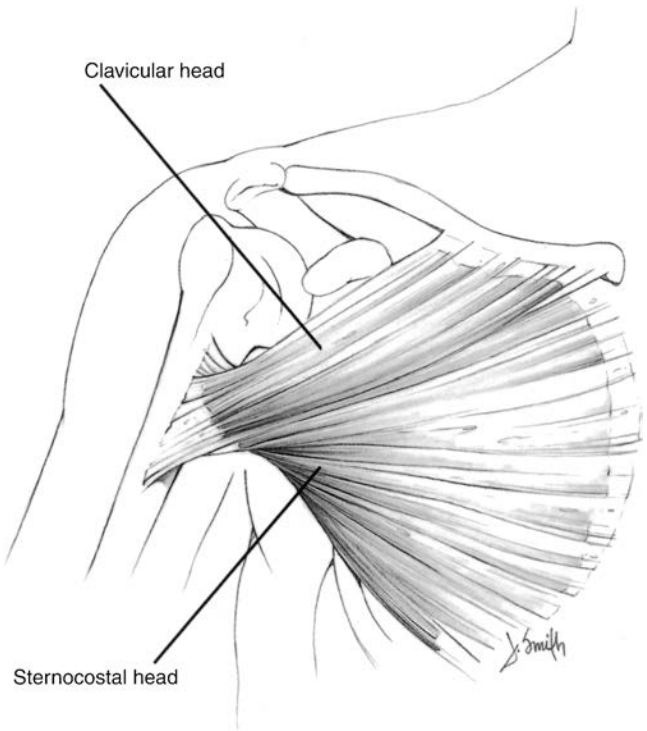


Figure 8-1 Anatomy of the pectoralis major. The triangular muscle converges to form two distinct segments: the smaller clavicular head and the larger sternocostal head. The sternocostal portion spirals on itself to insert proximally and underneath the clavicular portion of the muscle.

The function of the pectoralis major depends on the arm's relative position to the chest at the time of contraction. For example, the muscle functions to flex the humerus if it is extended behind the plane of the body; whereas the pectoralis major's lower fibers will extend the shoulder when the contraction is initiated with the arm in a flexed position. The pectoralis is also a powerful adductor and internal rotator of the humerus.[73] Weight lifting, and particularly the bench press, is a commonly reported mechanism of injury for pectoralis major rupture. Wolfe et al.[123] examined muscle fiber lengths at various points along the muscle, both at rest and during a simulated bench press maneuver in a cadaveric model. They demonstrated that the short inferior fibers of the muscle lengthened disproportionately during the final 30 degrees of humeral extension when compared with the superior fibers of the sternal head. Therefore, these inferior fibers are theoretically placed at a mechanical disadvantage in the eccentric phase of the lift, predisposing them to injury.[123]

The pectoralis major is innervated by the lateral and medial pectoral nerves, which principally serve the clavicular and sternocostal heads, respectively. These nerves course from a superior to inferior direction and enter the muscle along its posterior surface. The muscle's innervation is not in jeopardy during mobilization of the tendon for surgical repair.

Incidence and Pathophysiology

Rupture of the pectoralis major muscle is relatively uncommon, first reported in 1822 by Patissier.[86] Since that time approximately 200 cases have been reported, but many were not confirmed by exploration for surgical repair.[24] McEntire and associates published a literature review of pectoralis major ruptures in 1972 and added 11 new cases to the 45 already in the literature at that time.[75] However, only 22 of these 56 cases were confirmed surgically. Larger series of new cases have more recently been reported. Kretzler and Richardson, in 1989, reported 19 new cases, of which 16 were repaired.[57] Wolfe et al., in 1992, published 14 new cases, with seven being managed surgically.[123] Bak et al.[9] performed a meta-analysis of 112 cases of pectoralis major rupture. Hanna et al. reported on 22 cases, with 10 surgical repairs, in 2001.[43] Aarimaa et al. retrospectively reviewed 33 operatively treated cases of total or near-total pectoralis major ruptures at their own institution, along with a meta-analysis from the literature on the treatment and outcome of pectoralis major ruptures.[1] Most of these injuries occur in men during the third or fourth decade.[53]

The mechanism of injury usually described involves an overwhelming extension force applied to a maximally contracted pectoralis muscle, resulting in a complete tendinous avulsion from bone. The single most common mechanism causing pectoralis rupture has been the bench press exercise.[10,50,53,57,64,94,116,123,125] Other sports activities, such as boxing, football, ice hockey, sail boarding, skiing, water skiing, and wrestling, have been associated with cases of pectoralis rupture. Recently, rappelling in an active-duty soldier was responsible for acute rupture of the pectoralis major tendon.[119] There is also one report of pectoralis major rupture in a paratrooper when entanglement of the aircraft risers led to excessive traction in a malpositioned shoulder.[55] The mechanism of injury involves either an indirect sudden force applied to the upper extremity or, in a small number of cases, a direct blow to the contracted muscle. Reaching out to break a fall or grabbing a railing to prevent a fall are typical mechanisms.

Pectoralis major ruptures may be complete or partial. Partial injuries, though more common, are treated nonsurgically and, consequently, the pathoanatomy of these lesions has not been well documented.[44] Partial injuries of the musculotendinous unit may occur in the muscle belly, at the musculotendinous junction, or at the tendinous insertion site. Partial tendinous disruption at the bony insertion site most often involves a complete rupture of the sternal portion of the muscle, while the smaller clavicular part remains intact. Palpation of the intact clavicular head may be interpreted as an incomplete injury not requiring surgical intervention. However, the sternal portion constitutes approximately two-thirds of the muscle mass and its repair is warranted in active individuals. This pattern of disruption is consistent with the common mechanism of injury involving the terminal eccentric part of the bench press where the lower muscle fibers of the pectoralis are stretched disproportionately. Complete rupture of both the sternal and clavicular head is less common, but has been reported.[64,68,75,85,116,123] Additionally, Potter et al. reported one case of simultaneous bilateral rupture of the pectoralis major tendon, suffered while the patient was performing dips on a wide-grip parallel bar.[90]

A survey of the literature shows that most reported cases involving the pectoralis major are distal disruptions at the insertion site and musculotendinous junction, rather than proximal ruptures of the muscle belly. Proximal muscle belly ruptures occur occasionally and tend to result from a direct blow to the contracted muscle.[52,75] Interestingly, there are few cases in the literature where a bone fragment from the humerus was avulsed at the time of injury.[83,117] In one case the bone fragment was small and inconsequential. In the other, a large fragment of humeral cortex was reinserted with screws and spiked washers.[117]

Recently, there have been several reports in the literature regarding pectoralis major rupture in the elderly, all from Beloosesky et al.[11–14] In one paper, they reported on pectoralis major rupture in a 97-year-old woman.[11] These authors noted that these injuries are probably more common in the elderly population than generally believed, as indirect trauma to the muscle may occur with common nursing procedures such as transferring, positioning, and dressing when the upper extremity is placed in particular positions. Further, soft tissue in elderly individuals is frequently stiffer, more atrophic, and further at risk from a general decrease in muscular activity and changes in diet and nutrient intake. Lastly, an unfortunate but real consideration is that of elder abuse, suspected by Beloosesky et al. in some patients in their series.[12]

Evaluation

The principal clue to the diagnosis of a pectoralis major rupture is the patient's history of an acute traumatic event associated with burning pain and a tearing sensation of the upper arm and chest. Most often the mechanism of injury described by the patient involves an overpowering extension force on the extremity. An audible pop is not usually appreciated.

The specific physical findings depend on the exact site and extent of muscle rupture. The more common distal ruptures will have swelling and ecchymosis of the lateral chest and upper arm region. The muscle retracts medially and superiorly, although the absence of the anterior axillary fold may not be immediately obvious in the acute setting. With complete avulsion of the sternal head, one may still be able to palpate the thin, tendinous clavicular insertion. Zeman et al. described one patient who, at surgery, had a complete avulsion at the musculotendinous junction, but a persistent overlying fascia layer had clinically masqueraded as a segment of intact tendon.[125] Weakness of adduction and internal rotation with a medial muscle bulge that is accentuated with resistance can usually be appreciated.

Conversely, proximal or medial muscle injury causes ecchymosis and swelling on the anterior part of the chest wall. The muscle belly retracts toward the axillary fold and a visible and palpable medial defect is seen. Again, these injuries are often a result of a direct blow or crush injury. A useful clinical test involves having the patient firmly press his or her hands together in front of the chest to allow for simultaneous inspection and palpation of the involved and uninvolved pectoralis muscles.

In the chronic setting, the diagnosis is very straightforward. The patient will complain of fatigue, ache, and weakness to strenuous internal rotation and adduction activities. Weight lifters and body builders, in particular, will complain of the gross asymmetry compared with the uninvolved side. Weakness on manual muscle testing of adduction and internal rotation can usually be appreciated. Isokinetic testing has been used to quantify and document this weakness.[57,65,98,102,123]

Occasionally, associated injuries will present with pectoralis major ruptures. Injuries to the anterior deltoid, rotator cuff, latissimus dorsi, and brachial plexus have all been seen.[52,75] Interestingly, pectoralis major disruption has only been reported in combination with an anterior traumatic dislocation of the shoulder in one case.[6] The mechanism for these two anterior shoulder injuries is similar, albeit with traumatic instability usually occurring at greater degrees of abduction and external rotation. It is important to consider this differential diagnosis when assessing football players who have been injured while tackling with their arm in an abducted and extended position.

Radiographic evaluation of the shoulder and chest is usually negative, except in the rare case of an avulsion of a small bone fragment.[83,117] The subtle finding of a loss of the normal pectoralis major shadow has been described.[73,85,125] Magnetic resonance imaging (MRI) is potentially helpful in the early evaluation of this injury.[78] In the acute setting, an MRI can identify the extent and site of muscle injury when the clinical diagnosis is unclear because of swelling and pain (Fig. 8-2). In most cases, an MRI is unnecessary, as subsequent examination within 7 to 10 days after the acute event will usually clarify the diagnosis. This brief delay in diagnosis has no influence on decision making or the ultimate result. Some authors do advocate the utility of MRI in the early postinjury period to potentially delineate the extent of the injury and assist with surgical planning. Carrino et al. reported on 10 patients referred for MRI after injury to the pectoral muscle, and found that MRI is accurate and useful in detecting and grading tears.[23] In an unusual report, Povoski and Spigos presented an MRI of the breast, ordered to evaluate a suspected breast mass in an 87-year-old woman, which revealed a partial tear of the pectoralis major.[91] Ultrasound is another modality that has become increasingly popular for the diagnosis of pectoralis major tears.[13,96,120] These studies generally support the use of sonographic evaluation as a complement or alternative to MRI for high-resolution imaging with low cost and superior practicality.

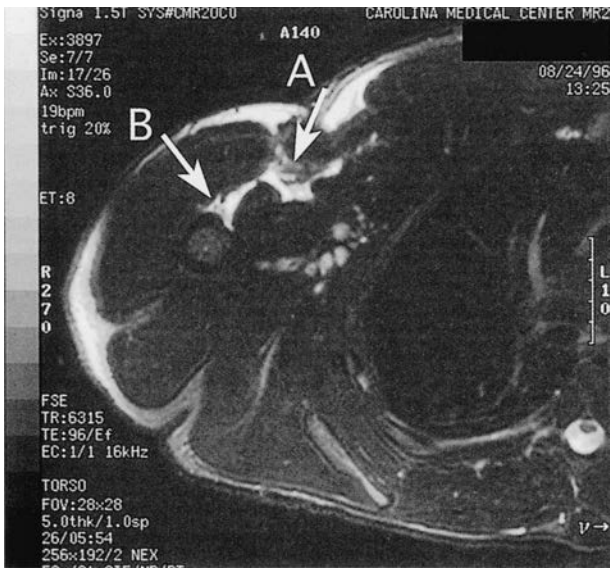


Figure 8-2 Magnetic resonance imaging depicting the retracted pectoralis major with adjacent hematoma *(A)* and the tract leading from the humeral insertion underneath the deltoid *(B)*.

Treatment

The treatment of pectoralis major muscle ruptures is determined by the specific location and degree of injury. Tears from the origin or within the muscle belly, whether a mild strain, partial tear, or complete disruption, are not amenable to surgical intervention and respond satisfactorily to conservative treatment.[24,45] There are several case reports in the literature of wrestlers who sustained medial pectoral injuries and successfully returned to competition. Injuries at the musculotendinous junction are usually of partial severity, and conservative management will yield acceptable function despite the residual cosmetic defect.[75,98,112] MRI may help the surgeon decide on operative versus nonoperative management by providing information on the exact location of a distal injury and the degree of muscle involvement.[29,78] Distal tendinous avulsion may be complete involving both heads of the pectoralis major, or partial where just the larger inferior sternal portion is detached. Most authors would recommend surgical repair of these injuries, particularly in athletes and laborers, who require optimal function and prefer upper body muscular symmetry (Fig. 8-3).[10,16,33,50,57,64,65,71,73,75,85,125] This recommendation is based on subjective complaints of weakness interfering with recreational or occupational function and cosmetic deformity. Isokinetic testing has demonstrated 25% to 50% muscle deficits in adduction and internal rotation in both preoperative patients and in those treated nonoperatively.[57,65,98,102,123]

The literature is divided in the recommendations for treatment of chronic ruptures. A few authors have suggested nonoperative treatment for the chronic tear, based on satisfactory functional recovery in a few cases while avoiding the potential difficulties of late repair.[73,98] The more recent literature tends to favor late repair of complete disruptions, with satisfactory results achieved with surgery done up to 5 years after rupture.[10,57,64,65,102,123,124] Anbari et al. reported on one patient who underwent successful repair of pectoralis major rupture 13 years after the initial injury, restoring strength and function as well as improving contour and cosmesis of the muscle complex.[3]

Nonoperative Treatment

Nonoperative treatment is recommended for medial ruptures of the pectoralis origin, muscle belly, and partial injuries at the musculotendinous junction. Conservative

Figure 8-3 **(A)** Preoperative photograph of a professional football player who had sustained a complete pectoralis major avulsion while reaching out to make a tackle. Note the web appearance of the anterior axilla. **(B)** Postoperative photograph showing restoration of the anterior axillary fold after a pectoralis major repair.

management is also appropriate for complete distal ruptures in lower-demand individuals, particularly if the nondominant extremity is involved. Initial treatment includes immobilization in a sling and icing to control pain and to allow stabilization of the hematoma. Gentle active and passive range-of-motion exercises are begun at 7 to 10 days postinjury. In the partial distal injury, care is taken to avoid humeral extension and abduction to protect the vulnerable inferior fibers of the sternal portion of the muscle. Resisted exercises are begun at 6 weeks. Isokinetic exercises for horizontal adduction can assist in the rehabilitation to develop strength and endurance. Depending on the severity of the initial injury and the specific occupational or athletic demands, patients may return to unrestricted activity at between 8 and 12 weeks.

Surgical Management

The goal of surgical intervention is to achieve optimal functional recovery in high-demand individuals requiring full strength. Surgery is best suited for complete tendinous avulsion of both the clavicular and sternal heads or complete disruption at the musculotendinous junction. Serious consideration for surgical repair should be given for partial tendinous avulsions in active athletes or heavy laborers in whom the sternal head has been completely avulsed but the smaller clavicular head remains. Surgery is not indicated for proximal tears of the muscle origin or muscle belly.

Anatomic repair is accomplished using heavy nonabsorbable suture placed in Bunnell fashion in the distal tendon passed through drill holes lateral to the biceps tendon at the humeral insertion site.[57,123] Other technical options reported include using pullout wires,[75] a barbed staple,[35] and, most recently, suture anchors.[78] Disruptions within the distal tendon itself occur rarely, but should also be secured directly to bone.

Repair of musculotendinous junction tears is more difficult because of the inability to obtain secure suture fixation in the torn muscle. For this reason, most partial tears are probably best managed nonoperatively. Very large defects or complete musculotendinous junction disruptions are accomplished with standard suturing techniques. Less vigorous postoperative rehabilitation is recommended in this setting.

Late primary repair for chronic ruptures—generally defined as greater than 2 weeks since injury—can be accomplished, even up to 5 years after the initial injury, although surgical dissection through scar and mobilization of the chronically retracted muscle is more difficult.[10,57,64,65,95,102,123] Schepsis et al. report on 17 patients with distal pectoralis major rupture, with 13 patients (six acute injuries and seven chronic injuries) undergoing surgical fixation while four patients were treated nonoperatively.[100] These authors concluded that there were no significant subjective or objective differences between patients treated operatively for acute or chronic injuries, though both groups of patients fared better than the group treated nonoperatively.

Postoperative management is relatively aggressive. The arm is immobilized for 1 to 2 weeks and then gradually weaned out of the sling by 4 weeks after surgery. Progressive active exercises and gentle use out of the sling are begun at 2 weeks postoperatively. Passive stretching is avoided. Resisted exercise begins at 6 weeks. Isokinetic machines can offer an objective assessment of strength and endurance and are used at 8 to 10 weeks in anticipation of return to full activities by 3 to 4 months after surgery. The postoperative rehabilitation program must be less aggressive in those cases of musculotendinous junction repair.

Results

Although nearly 200 cases are presented in the literature, the results of treatment, whether nonoperative or surgical, are frequently described anecdotally. There are very few

large series in the literature that offer objective criteria with which we can critically analyze results. In 1970 Park and Espiniella reviewed 30 cases of pectoralis major ruptures. The results in those patients treated with surgical repair were 80% excellent and 10% good. These results were superior to the 17% excellent and 58% good outcomes in patients treated nonoperatively.[85] McEntire et al., in 1972, presented 11 new cases and combined the results with the 45 cases that were already in the literature. They identified a similar trend favoring surgical management. In those patients for whom outcomes could be determined by the data available, surgical treatment yielded 77% excellent and 11% good results. By comparison, results in those patients who received conservative treatment or no formal treatment at all were 29% excellent and 54% good.[75]

Zeman et al., in 1979, reported on nine athletes with ruptures of the pectoralis major. Four cases were treated surgically and all had excellent results. The five patients managed nonoperatively had residual weakness, with two weight lifters and one professional boxer dissatisfied with their ability to perform at return to athletic competition.[125]

Kretzler and Richardson, in 1989, repaired 16 distal avulsions, and 81% achieved full return of range of motion, strength, and normal contour. Two patients were repaired 5 years after injury and, although they had a satisfactory clinical result, both patients have persistent weakness in horizontal adduction of 16% and 20%, respectively, by Cybex evaluation.[57]

Wolfe et al., in 1992, evaluated 14 sports-related ruptures, seven treated surgically and seven conservatively. Cybex testing demonstrated marked strength and work deficits in those patients treated conservatively, compared with normal strength in the repaired group.[123] Bak et al.,[9] in their meta-analysis of 112 cases, determined that prognosis was unrelated to patient age or location of the rupture, and that surgical treatment—preferably within the first 8 weeks after injury—had a significantly better outcome than repair after protracted delay or nonoperative management.

Surgical repair of chronic pectoralis ruptures is technically feasible, even up to 5 years after injury. Acceptable results can be obtained, albeit somewhat inferior in recovery of strength compared with those repaired immediately.[10,50,57,64,65,102,123] Jones and Matthews, in 1988, reviewed the literature and concluded that ruptures repaired within 7 days had 57% excellent and 30% good results, but those patients with delayed repair had no excellent and 60% good results.[50] Although delayed primary repair of chronic ruptures is possible, patients should be told that a satisfactory outcome is probable but that there is a likelihood of mild persistent weakness and cosmetic asymmetry.

Complications

Complications from surgical intervention are infrequent. One patient was slightly limited in abduction postoperatively,[57] and another had postoperative fourth and fifth digit paresthesias of unknown cause.[123] Interestingly, there have been several early reports of complications from pectoralis major rupture related to the associated hematoma in patients treated nonoperatively. Sepsis from an infected hematoma caused the death of one patient and precipitated the death of a second with pneumonia.[81,86] Additionally, a pseudocyst had formed from a hematoma,[99] and another reported case involved a partial rupture with subsequent hematoma that became infected, leading to persistent, low-grade morbidity prior to drainage.[25] Finally, myositis ossificans was reported to develop in a patient 4 months after rupture.[94] Interestingly, rerupture after surgical repair has not been documented.

Authors' Preferred Treatment

Conservative treatment is indicated for all proximal injuries involving the pectoralis major origin and muscle belly. Partial injuries at the musculotendinous junction can be similarly managed nonoperatively. One may wish to consider operative intervention if a large defect at the musculotendinous junction occurs in a high-demand individual or body builder who requires symmetry and optimal strength. An MRI can be helpful in this setting to determine the exact location and extent of the defect when the clinical evaluation is obscured by swelling and hematoma.

Acute surgical repair is recommended not only for complete avulsion, but also for partial distal injuries that involve the entire sternal head of the pectoralis major and leave the clavicular head intact. We prefer approximating the tendon to a bony trough with heavy, nonabsorbable, braided sutures passed through cortical bone tunnels. The humeral cortex in young, active individuals is fairly thick and the bone holes should be drilled in a convergent orientation. Suture passage can be facilitated using commercially available suture passing devices or alternatively a contoured loop of a 24-gauge wire. Sutures passed in mattress fashion through these tunnels maximize the tendon-to-bone contact (Fig. 8-4). Suture anchor fixation is another option, but cinching the tendon firmly down to the bone may be difficult, and to date there is only one case in the literature advocating this technique.[78]

Postoperative rehabilitation is fairly aggressive, with early mobilization and return to unrestricted activity by 3 to 4 months after repair.

DELTOID RUPTURES

Surgical Anatomy and Biomechanics

The deltoid is the largest muscle of the shoulder girdle and its integrity is critical to shoulder function. It consists of three major parts, with the anterior deltoid taking origin

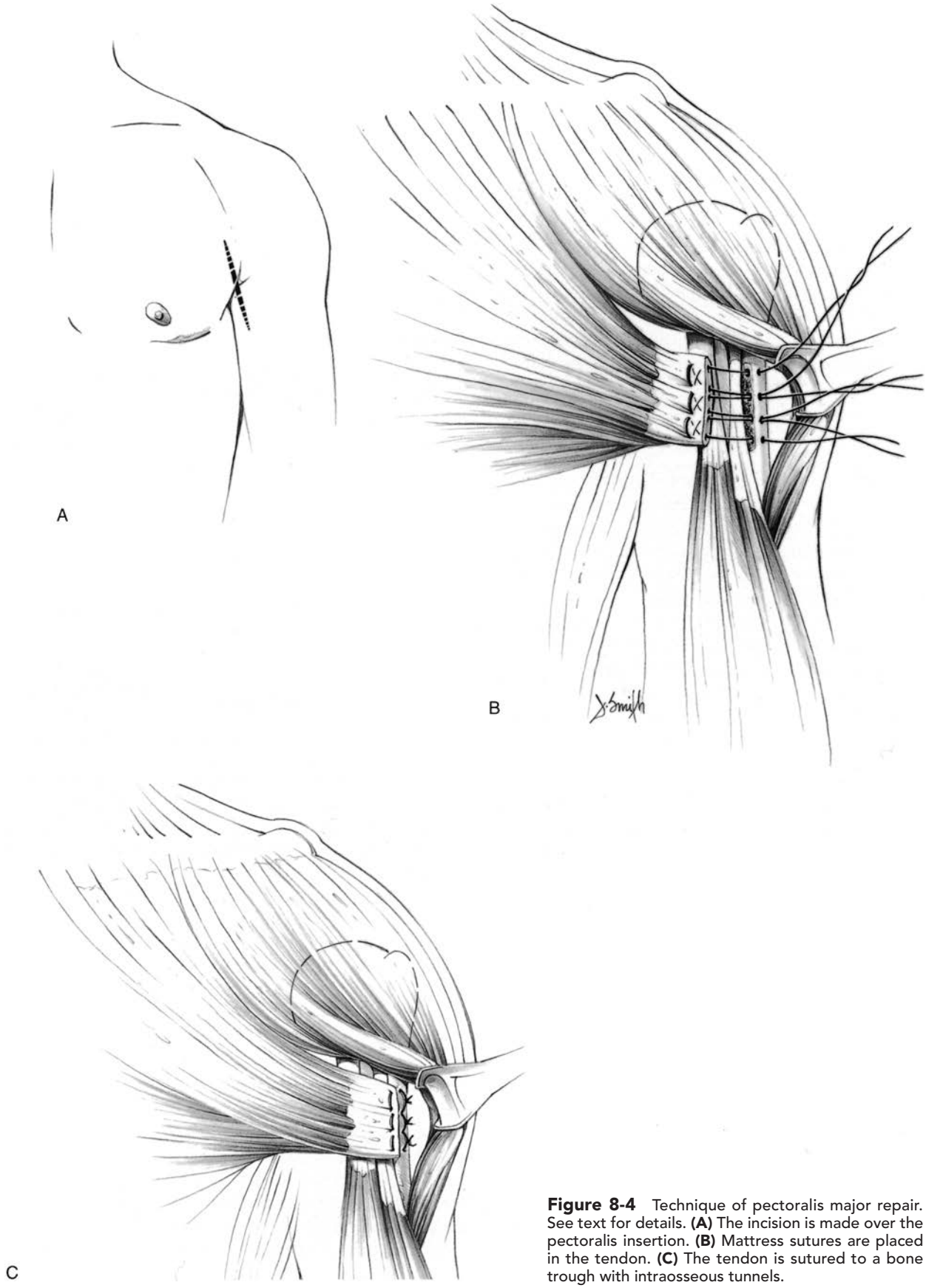


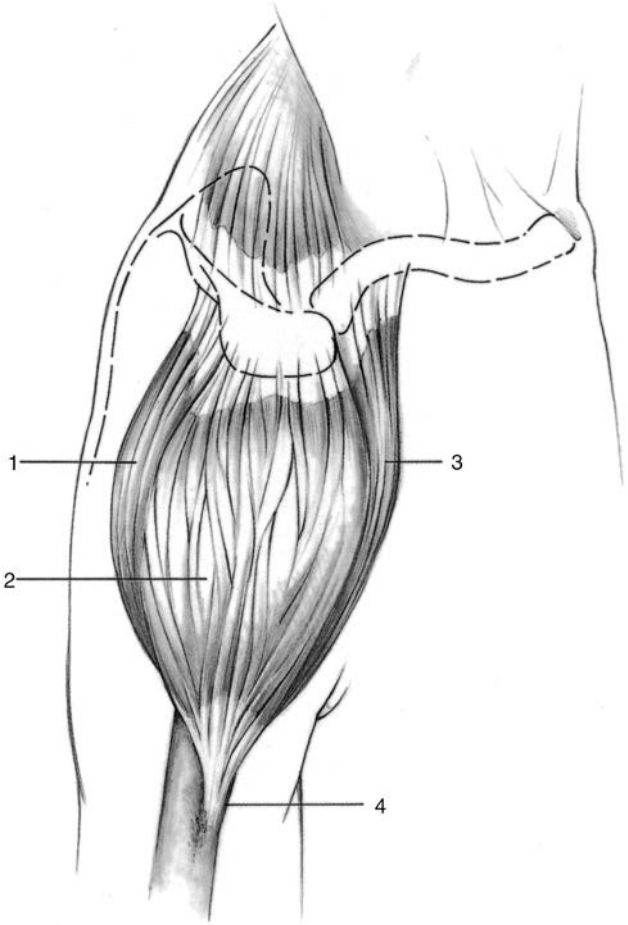
Figure 8-4 Technique of pectoralis major repair. See text for details. **(A)** The incision is made over the pectoralis insertion. **(B)** Mattress sutures are placed in the tendon. **(C)** The tendon is sutured to a bone trough with intraosseous tunnels.

from the anterior and superior surfaces of the outer third of the clavicle and anterior acromion, the middle deltoid from the lateral margin of the acromion, and the posterior deltoid from almost the entire scapular spine (Fig. 8-5).[48] The fibers converge from this wide origin to insert into the deltoid tuberosity on the lateral humeral shaft. Anterior and posterior fibers are essentially unipennate, meaning they run parallel to each other. The more powerful middle part is multipennate, with an internal structure consisting of tendinous septae serving as both origin and insertion sites for its relatively short and multiply oriented muscle fibers.[48] The axillary nerve innervates the muscle after exiting the quadrangular space posteriorly and courses along its undersurface from posterior to anterior.

The most important function of the deltoid is forward elevation in the scapular plane. The anterior and middle deltoids contribute to this motion. All three components of the muscle, but particularly the middle third, abduct the humerus, whereas the posterior deltoid extends the humerus.

Incidence and Pathophysiology

Traumatic rupture of the deltoid is rare. There are few reported cases in the literature from which we can draw information concerning the cause and management of this injury. Deltoid disruption appears to be a result of a sudden force applied to the muscle, sometimes associated with a concomitant direct blow. The first case was described by Clemens in 1913 and occurred while the patient was carrying a heavy rail.[28] Gilcreest and Albi reported two cases in 1939; one was sustained in an automobile accident and the other occurred with an overhead lifting accident.[39] McEntire et al. published a case of a deltoid rupture associated with a pectoralis major tear.[75] Caughey and Welsh, in review on this subject, described a chronic deltoid detachment in a 62-year-old guide who sustained the injury when a snowmobile rolled over him.[24] Davis, in 1919, reported a case not of traumatic cause, but of a deltoid origin detachment caused by osteomyelitis of the distal clavicle.[32] Allen and Drakos, in 2002, described



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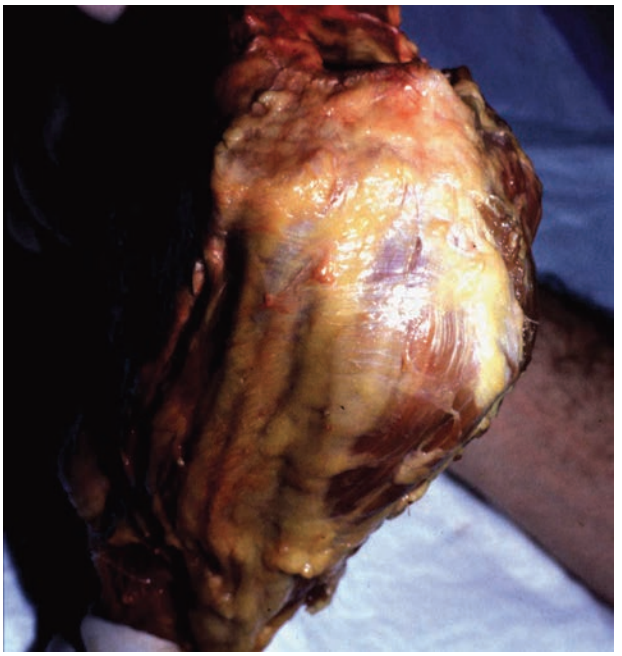


B

Figure 8-5 Illustration **(A)** and Cadaveric Specimen **(B)** depicting the anatomy of the deltoid muscle. **(1)** Posterior one-third deltoid; **(2)** middle one-third deltoid; **(3)** anterior one-third deltoid; **(4)** deltoid tuberosity of humerus.

partial detachment of the posterior deltoid in a professional cricket player in the absence of a rotator cuff injury; the mechanism was thought to be due to fast-bowling movements associated with the sport of cricket.[2] Hydrocortisone injections may play a role in the cause, though in this case the patient denied such history. Finally, Lin and Nagler described partial tear of the posterior deltoid muscle in an elderly woman.[63] It was thought that the mechanism of injury in this case was due to repetitive motions associated with the patient's preferred hobby, golfing, on the backswing with the club. Combined with an older patient with more friable, weaker tissue, repetitive microtrauma may best explain a partial, acute-on-chronic partial tear of the right posterior deltoid in this left-handed patient. Blazar et al., in 1998, described four shoulders in three patients who suffered spontaneous detachment of the deltoid origin; all patients had chronic massive rotator cuff tears who then presented with the acute onset of shoulder weakness.[18] This type of tear is most likely due to the attritional changes of the fibers of the deltoid origin secondary to the superior migration of the humeral head associated with massive rotator cuff deficiency. Deltoid detachment as a complication of chronic cuff tear arthropathy or previous shoulder surgery is addressed in Chapter 5.

In a general orthopedic practice, minor strains of the deltoid are not uncommon and are often related to participation in athletic activity, particularly throwing sports. Contusions from direct blows may be associated with a subcutaneous hematoma, but usually do not involve significant muscle fiber disruption. Complete traumatic disruption as evidenced by the foregoing literature accounts is extremely rare and is caused by an abrupt external force applied either directly or indirectly to the contracted deltoid muscle.

Evaluation

Physical examination findings are dependent on the site of rupture and extent of muscle involvement. The clinical presentation is also determined by whether the injury is acute or chronic.

A mild strain or direct contusion will be manifest by local tenderness, mild swelling, and decreased active range of motion secondary to pain. The degree of weakness in either forward flexion or abduction will reflect the exact location and magnitude of the injury.

Acute complete deltoid rupture may occur in the context of a severe traumatic event to the upper extremity or in conjunction with a multiple trauma case. Rupture from the acromial origin can involve an isolated anterior, middle, or posterior head of the deltoid. Acute swelling may mask the proximal muscle defect. Resisted deltoid contraction will accentuate the distal muscle retraction and reveal the asymmetrical deltoid contour and muscle defect (Fig. 8-6).

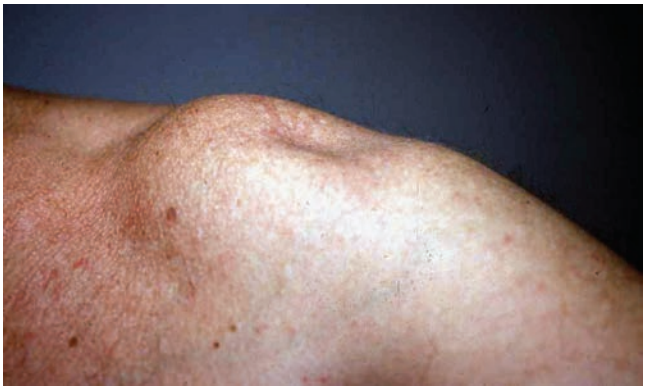


Figure 8-6 Spontaneous detachment of the middle deltoid origin in a patient with a massive chronic rotator cuff tear.

Passive range of motion may be only slightly limited because of pain. Weakness in forward elevation, abduction, or extension reflects injury to the anterior, middle, and posterior heads, respectively. If significant weakness is present but no defect is appreciated, the possibility of axillary nerve injury must be entertained, particularly if there is a history of concomitant shoulder dislocation. Serial examinations, MRI, or electromyography may be helpful if the diagnosis is uncertain (Fig. 8-7).

Treatment

Deltoid strains and contusions are treated nonoperatively with early icing and passive range-of-motion exercises to avoid stiffness. After resolution of the acute injury phase, heat, electrical stimulation, and graduated stretching and strengthening exercises are instituted. Full recovery is anticipated within 4 to 6 weeks of injury.

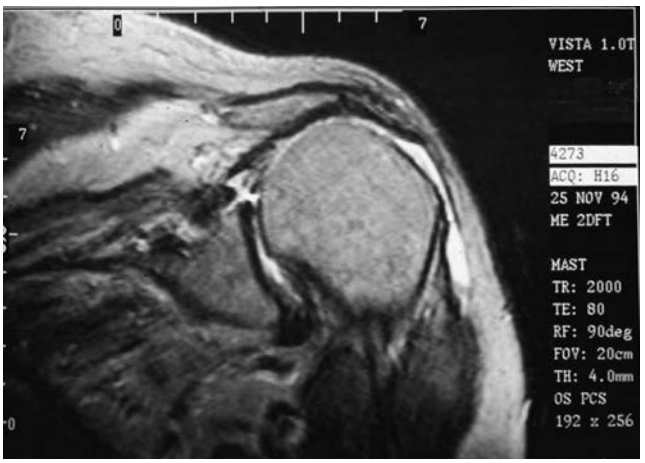


Figure 8-7 Magnetic resonance imaging of the same patient in Fig. 8-6 with a spontaneous attritional detachment of the middle deltoid origin.

There is limited published information with which to base recommendations for treatment of acute traumatic ruptures. Disruptions involving an entire segment of the deltoid, particularly the anterior or middle portions, will significantly alter shoulder function and should be surgically repaired back to the acromion. An MRI may help quantify the extent of deltoid origin involved. Prompt surgical repair is necessary to achieve an optimal result. A midsubstance muscle tear is certainly more difficult to repair securely, raising the question of whether repair should be attempted for these injuries. If severe soft tissue and muscle injury of the shoulder girdle presents in association with a closed head injury, one may wish to consider prophylactic use of indomethacin (Indocin) to avoid heterotopic ossification.

Postoperatively, the deltoid repair should be protected in an abduction pillow or splint for 4 to 6 weeks. Passive range of motion in forward elevation and internal and external rotation from the abduction pillow or brace should begin immediately. Humeral extension or internal rotation with the arm behind the back must be avoided for 6 weeks. Progressive active exercises are started at 6 weeks postoperatively.

Complications

A miss or a delay in diagnosis of an acute deltoid rupture will significantly compromise the surgeon's ability to restore normal shoulder function. Complications of deltoid detachment more commonly pertain to dehiscence of the deltoid repair following shoulder surgery and are discussed in Chapter 5 of this text.

TRAPEZIUS, LATISSIMUS DORSI, AND TERES MAJOR STRAINS AND RUPTURES

Surgical Anatomy and Biomechanics

Trapezius

The upper anterior border of the trapezius forms the posterior border of the posterior triangle of the neck, with the muscle completely overlying the supraspinatus and the majority of the rhomboids. The lower fibers of the trapezius typically overlie the upper fibers of the latissimus dorsi as well. The triangle of auscultation is an area between the trapezius, latissimus, and rhomboids whereby a triangle of variable size exists, particularly during scapular protraction.[48]

The trapezius has an extremely broad origin, extending medially from the superior nuchal line on the occipital bone, from the external occipital protuberance, from the ligamentum nuchae, and from the seventh cervical and all thoracic vertebral spinous processes. The insertion of the trapezius is less broad than its origin yet is still substantial. The upper fibers insert posterosuperiorly on the distal third of the clavicle, while the lower fibers insert into the base of the scapular spine as they form a triangular, flat tendon.[48]

The primary functions of the trapezius are elevation of the lateral scapular angle and scapular retraction; for the former, this is the only muscle capable of performing this function as no other muscle has a downward insertion onto the lateral scapular angle. The upper muscle fibers exert upward pull on the tip of the shoulder while the lower fibers have a downward pull on the root of the scapular spine; these fibers work synergistically to assist in upward rotation of the scapula.

Innervation of the trapezius is provided by the spinal accessory nerve (cranial nerve XI) and several cervical nerves that send fibers into the muscle. The spinal accessory nerve, after passing deep or through the sternocleidomastoid, passes through the posterior triangle of the neck to supply the trapezius. The nerve is joined by cervical nerve fibers arising from supraclavicular branches of the third and fourth cervical nerves. The ascending branch of the transverse cervical artery accompanies the spinal accessory nerve as it runs downward on the deep surface of the muscle.[48]

Latissimus Dorsi

The latissimus dorsi, like the trapezius, also has a broad origin. The muscle originates from the spinous processes of lower six thoracic vertebrae and from the spinous processes of the lumbar and sacral vertebrae; from an aponeurosis stemming from the iliac crest; and by muscular slips arising from the lower four ribs. The fibers converge as they run laterally and begin to spiral around the lower border of the teres major. The muscle fibers themselves end in a tendon in the axilla, along with the tendon to the teres major, that passes around to the medial surface of the humerus to insert into the medial wall and bicipital groove of the humerus; the insertions of the teres major and latissimus are quite close together with the teres major insertion located just medially to the insertion of the latissimus dorsi tendon on the anterior aspect of the humerus.[48]

Primary functions of the muscle are adduction, internal rotation, and arm extension. Secondarily, through its pull of the humerus on the scapula, it can participate in depression or downward scapular rotation, as is used when a crutch is placed in the armpit. The thoracodorsal nerve innervates this muscle, along with contributions from the seventh cervical nerve and frequently from the sixth and eighth cervical nerves as well.[48]

Teres Major

The origin of the teres major comes from the dorsal scapula from the medial third of the lateral border. As it

passes laterally it is in close contact with the latissimus as the two spiral together around to the anteromedial side of the humerus, where it inserts on the medial lip of the intertubercular groove in close proximity to the latissimus.[48] The muscle similarly functions as an internal rotator, adductor, and arm extensor. The teres major forms the inferior border of both the triangular space and the quadrangular space. Innervation comes from the lower subscapular nerve, which arises from the posterior cord or the axillary nerve.

Incidence, Evaluation, Treatment, and Case Reports

To our knowledge, isolated complete ruptures of the trapezius have never been reported. While the overwhelming majority of these injuries are intramuscular strains, work-related upper-extremity disorders, fatigue syndromes, psychosocial stressors, and many other pathologies can contribute to trapezius strain and fatigue, which may then mimic or present with unusual findings. Unusual symptoms, protracted pain, or other clinical concerns should warrant evaluation with MRI. Acute trapezius injuries are often typically associated with acromioclavicular (AC) joint separations and/or distal clavicle fractures. If the clavicle has perforated through the trapezius creating a button-hole perforation, surgical recommendation includes appropriate reduction and repair of the AC or distal clavicle injury along with reapproximation of the trapezius perforation with resorbable sutures. Lastly, lesions of the spinal accessory nerve lead to clinical scapular winging and show denervation edema on MRI and electrophysiological changes on electromyography (EMG). The recommended surgical management for symptomatic trapezius palsy secondary to spinal accessory nerve injury is the Eden-Lange procedure.[17,34,58,59] In this procedure, the levator scapula is transferred laterally to substitute for the upper trapezius and the rhomboids are advanced to compensate for the loss of the middle and lower trapezius function (Chapter 34).

Similar to the trapezius, complete ruptures of the latissimus dorsi are quite rare and reportable. The majority of injuries also represent intramuscular strains. Spinner et al. reported on a 38-year-old golfer who sustained an avulsion injury to the conjoined tendons of the latissimus dorsi and the teres major from the humerus. With conservative management, the patient was doing well 3 years postinjury.[108] Henry and Scerpella, in 2000, reported on a 42-year-old athletic patient who sustained an acute traumatic tear of the latissimus tendon from its insertion; the patient underwent successful surgical repair, with the only clinical deficit noted to be a 14% reduction in adduction strength compared to the uninjured side 6 months later.[46] Other isolated case reports of latissimus rupture[21,66] also exist in the literature. One of the editors (JPI) has had one case of traumatic latissimus dorsi rupture in an athletic young man injured in a surfing accident. The physical examination demonstrated asymmetry (Fig. 8-8A) and rupture at the tendon bone attachment site was confirmed on MRI (Fig. 8-8B). Surgical treatment was performed within a week of the injury through two incisions (Fig. 8-8C,D) with sutures passed through bone tunnels. The clinical result was excellent (Fig. 8-8E).

As seen with trapezius and latissimus dorsi injuries, partial tears or strains of the teres major far outweigh complete ruptures in their prevalence. MRI is the imaging modality of choice. Maldjian et al. reported on one case of isolated teres major rupture in a waterskiing accident, due to the acceleration of the tow rope,[70] leading to avulsion of the tendon.

CORACOBRACHIALIS AND SHORT HEAD OF BICEPS RUPTURES

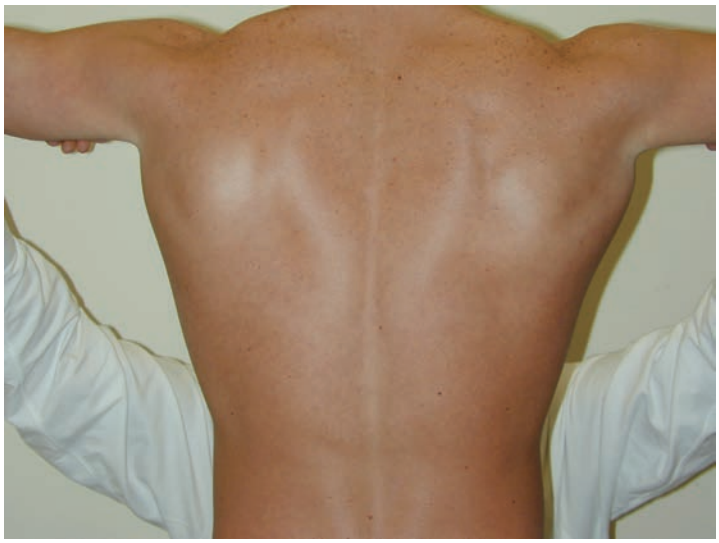
Surgical Anatomy and Biomechanics

The coracobrachialis muscle originates underneath and medial to the short head of the biceps on the coracoid process with a fleshy, tendinous attachment. It inserts on the anterior medial surface of the middle of the humerus.[48] The short head of the biceps takes origin from the lateral tip of the coracoid. The muscle belly of the short head fuses distally with the larger long head, ultimately inserting by a singular stout tendon across the elbow into the radial tuberosity.

The musculocutaneous nerve penetrates the coracobrachialis muscle and runs between the biceps and brachialis muscles before emerging distally as the lateral antebrachial cutaneous nerve.[48] The innervation of the coracobrachialis is supplied by both a small direct branch from the lateral cord and the musculocutaneous nerve. The main nerve enters the coracobrachialis between 3.1 and 8.2 cm from the tip of the coracoid. Small nerve twigs may enter the muscle as close as 1.7 cm from the coracoid.[38] The coracobrachialis is a flexor and adductor of the arm. The short head of the biceps contributes to arm adduction, but is principally an elbow flexor.

Incidence and Pathophysiology

Rupture of the coracobrachialis muscle and short head of the biceps is extremely rare. Few case reports of coracobrachialis rupture exist in the literature. Gilcreest and Albi, in 1939, described a case of coracobrachialis muscle belly rupture caused by direct trauma, which was confirmed at surgery.[39] The second case, recorded by Tobin et al. in 1941, occurred in a parachutist who sustained a complete tear of the coracobrachialis and short head of the biceps as a result of direct trauma to the arm by his static line.[113] Gilcreest, in 1934, listed two cases of short head of biceps tendon rupture, but no additional clinical information was



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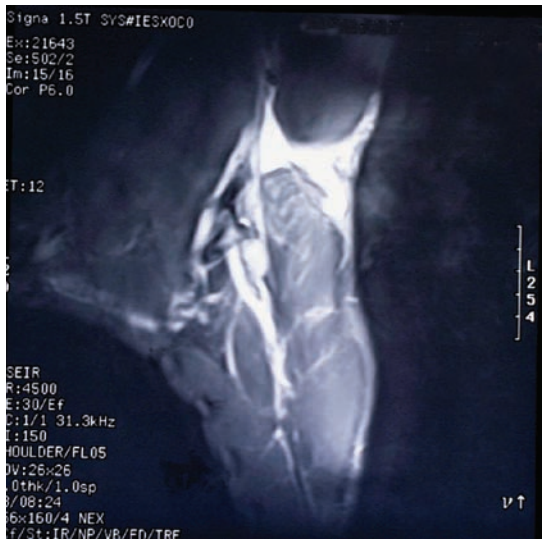

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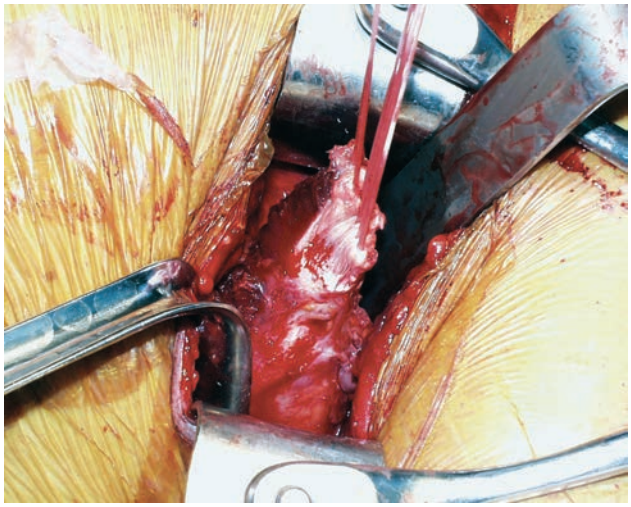

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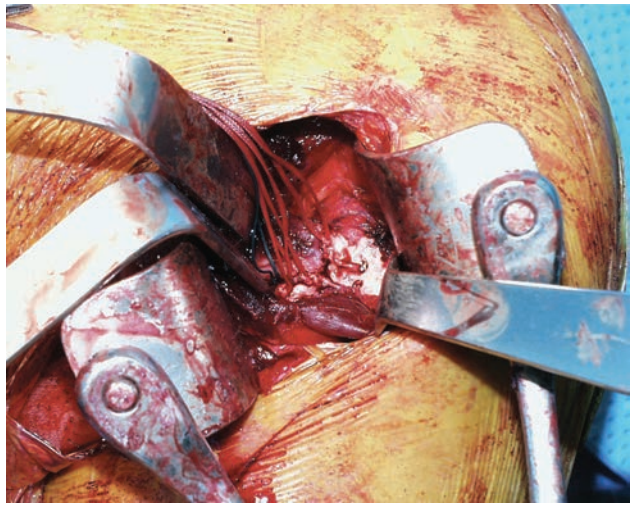

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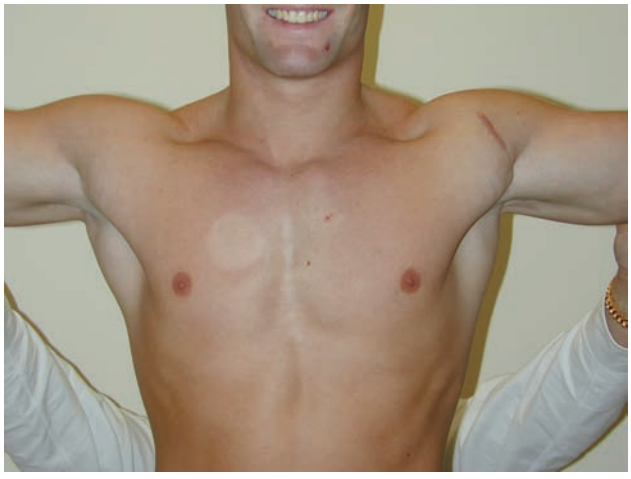

E

Figure 8-8 **(A)** A 21-year-old male involved in a surfing accident showing asymmetry of the latissimus dorsi due to a traumatic detachment at the tendon to bone attachment site. **(B)** Magnetic resonance imaging showing the detachment. **(C,D)** Intraoperative view of the tendon mobilized in the posterior wound and the attachment in the anterior wound to bone. **(E)** Postoperative function was normal.

provided.[41] Postacchini and Ricciardoi-Pollini, in 1977, reported on short head of the biceps rupture in a 67-year-old farmer in the absence of any direct trauma; the cause was felt to be severe degenerative changes in the quality and tensile strength of the tendon.[89] Finally, Shah and Pruzansky, in 2004, reported another case of a ruptured short head of the biceps that occurred in a 21-year-old man during a motor vehicle accident with the patient's arm out of the window where it struck the open door of a parked car. The isolated short head of the biceps rupture was identified by MRI and confirmed at exploration.[104]

The mechanism of this unusual injury most commonly appears to be direct trauma to a contracted muscle. Avulsion of these muscles' origin from the coracoid as a result of indirect forces is even more unusual.

Evaluation

The clinical presentation of a coracobrachialis or short head of biceps rupture is presumed to be a history of direct trauma to the arm associated with tenderness, swelling, and possibly a palpable defect in the muscle belly. Radiographs should be obtained to rule out a coracoid fracture. MRI may be very useful in identifying this unusual injury.

Treatment

The paucity of information available on this injury makes it difficult to give any firm recommendations for treatment. Gilcreest and Albi performed early surgical repair of the muscle belly and a complete recovery ensued.[39] It is not unreasonable to suggest exploration and appropriate surgical repair in those cases of direct trauma to the anterior arm when a significant muscle defect is appreciated by examination or MRI evaluation. Clinical factors, such as arm dominance and the patient's physical demands, should also play a role in the choice of treatment.

TRICEPS TENDON AVULSION

Surgical Anatomy and Biomechanics

The triceps muscle occupies the entire posterior aspect of the arm and comprises three distinct heads. The long head takes origin from the infraglenoid tubercle of the scapula and courses downward anterior to the teres minor and posterior to the teres major muscles. It serves as the medial border of the quadrangular space through which the axillary nerve and posterior humeral circumflex vessels emerge. The lateral head arises from the posterior surface of the humerus above the radial groove. The long and lateral heads converge distally to create the V-shaped triceps contour that can be appreciated superficially on the back of the arm. The medial head has a broad origin along the entire posterior surface of the humerus below the radial groove, and it fuses with the deep surface of the combined long and lateral heads. Ultimately, the entire muscle then transitions to form the substantial triceps tendon that inserts into the olecranon. Some of the superficial fibers of the tendon proceed over the surface of the olecranon and extend into the fascia of the forearm.[48]

The radial nerve innervates the triceps brachia, giving off multiple small branches to all three heads as it spirals downward along the posterior aspect of the arm. Although the nerve is at risk with posterior approaches to the humerus, it is not in jeopardy during the distal exposure associated with repair of a triceps avulsion.

The triceps muscle is essentially the sole extensor of the elbow, assisted only by the small anconeus muscle. The integrity of the triceps function is necessary to perform many activities of daily living, such as getting out of a chair and pushing objects away from the body.

Incidence and Pathophysiology

Anzel et al. reviewed a Mayo Clinic series of 1,014 tendon injuries and found only eight cases of triceps tendon injury, four of which were from a direct laceration.[5,80] The literature contains a few relatively small series, including Levy et al. (16 cases),[62] Pantazopoulos et al. (seven cases),[84] and Tarsney (seven cases).[110] Many of the remaining cases in the literature are presented as individual case reports.

Bach et al.[8] cumulated the cases of triceps tendon avulsion in the English literature up to 1987, and their study produced interesting epidemiologic data. The mean age at the time of injury was 26 years, ranging from 7 to 72. Males constituted 71% (29 of 41) of the patients. There was no correlation with the patient's dominant side.[8] Mair et al.[69] reported on a series of 10 partial and 11 complete ruptures of the triceps tendon in professional football players. Of the 19 players injured, 15 were offensive or defensive linemen. All complete tears were acutely repaired, while partial tears were managed either conservatively or operatively; it was not possible to draw firm conclusions about which partial tears would require surgery from their data.

The mechanism of injury for triceps tendon avulsion is a fall on an outstretched arm in most cases. The pathomechanics have been described as "a deceleration stress, superimposed upon a contracting triceps muscle with or without a concomitant blow to the posterior aspect of the elbow."[36] An abrupt, forceful, eccentric contraction of the triceps causes most triceps avulsions. Less frequently, a concomitant direct blow to the posterior aspect of the triceps at its insertion has been described in the mechanism of injury.[4,15,30,36,87,105,110] Additionally, there is one reported case of triceps avulsion during acceleration stress in a pilot in a high G-force training environment.[19] Recently, four

cases of triceps tendon avulsion injury have been reported in body builders and power lifters during the act of weight lifting, specifically the bench press or military press exercises.[8,22,47,51] The occurrence of this injury in body builders raises a question of the potential role of anabolic steroids in this injury.[8,47] Although steroid use was denied in these cases, this issue has been considered for predisposition to pectoralis major and distal biceps ruptures. Another study by Sollender et al. reported triceps rupture in four weight lifters, all of whom had taken oral anabolic steroids prior to injury.[107] The deleterious effects of either local steroid injections on tendons[54,115] or systemic anabolic steroid use on the strength of tendons and ligaments[49,56,77] have been well documented. A recent case report illustrates this point. A body builder who had received a series of six steroid injections for olecranon bursitis sustained an intrasubstance rupture of the triceps tendon while bench pressing.[109] In addition, he had a 5-year history of systemic anabolic steroid use. Individuals who use anabolic steroids are at risk for tendon ruptures because their abnormal muscular strength is applied to tendons that are stiffer and absorb less energy.[77]

The literature contains a number of systemically compromised patients in whom triceps tendon rupture has occurred spontaneously or as a result of trivial trauma. Medical conditions reported in association with tendon ruptures include renal osteodystrophy and secondary hyperparathyroidism,[26,37,67,72,74,82,92,93,103,114] chronic acidosis,[82] Marfan's syndrome,[101] and steroid treatment for lupus erythematosus.[114,122] The pathophysiology is not well understood, but calcification within the tendon from the chronic hypercalcemia of secondary hyperparathyroidism has been implicated.[93]

The site of triceps muscle injury may be at the muscle belly, musculotendinous junction, or osseous tendon insertion. The vast majority of authors have described tendo-osseous avulsions usually associated with a small fleck of bone.[36,80,110] Ruptures of the musculotendinous junction[40,47,79] and muscle belly tears[7,79,87] are unusual, having been noted in only a few cases. Although complete disruption is the rule, incomplete or partial tears do occur occasionally.[22,36,110] It is important to differentiate a partial tear from a complete rupture because surgical repair is necessary for the complete injury, whereas partial lesions are treated nonoperatively.[30,80]

Triceps tendon avulsions are sometimes associated with other upper-extremity injuries, resulting from the fall on the outstretched arm. Levy et al.[61,62] documented 16 patients with radial head fractures associated with triceps rupture. Lee[60] described a case of triceps rupture occurring in conjunction with a wrist fracture.

In the adolescent population, triceps avulsions involve complete separation of the olecranon epiphysis.[88,97] The secondary ossification center of the olecranon appears in the area of the triceps insertion at about 9 years of age. The physis fuses in an anterior to posterior direction at about 14 years of age.[20,106] The triceps expansion extends beyond the epiphysis, attaching to the surface of the metaphysis.[97] The injury sustained when the physis is incompletely fused produces a Salter-Harris type II fracture.[42]

Evaluation

The diagnosis of a complete triceps tendon avulsion is not difficult. The patient will relate a history of falling on the outstretched arm causing acute pain, a tearing sensation, and subsequent swelling and ecchymosis of the arm. The patient's complaint and physical findings will depend on whether the triceps is partially or completely torn. With a complete disruption, an indentation of the posterior contour of the arm can be seen, particularly as swelling subsides. A defect in the triceps mechanism is usually palpable. If a partial injury has occurred, tenderness is usually localized to the musculotendinous junction, and a discrete defect may not be appreciated. A complete tear results in the loss of extension against gravity, whereas an incomplete lesion is manifested by weakness, but the ability to actively extend the elbow is maintained.

In the acute setting, extension strength can be difficult to assess because of pain. In 1990, Viegas described a modification of the Thompson test[111] (traditionally used to identify complete ruptures of the Achilles tendon) for the assessment of a triceps injury.[118] The modified test can be performed either with the patient seated and the arm draped over the back of the chair or with the patient in the prone position, letting the forearm hang over the table. In the normal extremity, squeezing the triceps muscle belly produces slight elbow extension, but no motion will occur if a complete rupture is present.[118] This test is helpful for distinguishing between partial and complete triceps injuries.

The presence of associated injuries must be considered, particularly when falling on an outstretched hand is the mechanism of injury. Radial head fractures have been most frequently associated with triceps avulsions,[61,62,110] and distal wrist injuries[60] have also been reported. The differential diagnosis of triceps weakness includes nontraumatic causes. A C-7 nerve root lesion will cause isolated triceps weakness. As noted previously, spontaneous ruptures or injuries resulting from trivial forces may be the result of chronic medical conditions, including renal osteodystrophy with secondary hyperparathyroidism and long-term steroid use associated with systemic lupus erythematosus.

A plane lateral radiograph of the elbow will often confirm the diagnosis. A small avulsion fracture of the olecranon, called a "flake sign"[36,110] is estimated to be present in two-thirds of cases[47] and implies a complete rupture (Fig. 8-9). Additional studies, such as an MRI or ultrasound,[22] may be useful for defining the extent of partial lesions, but

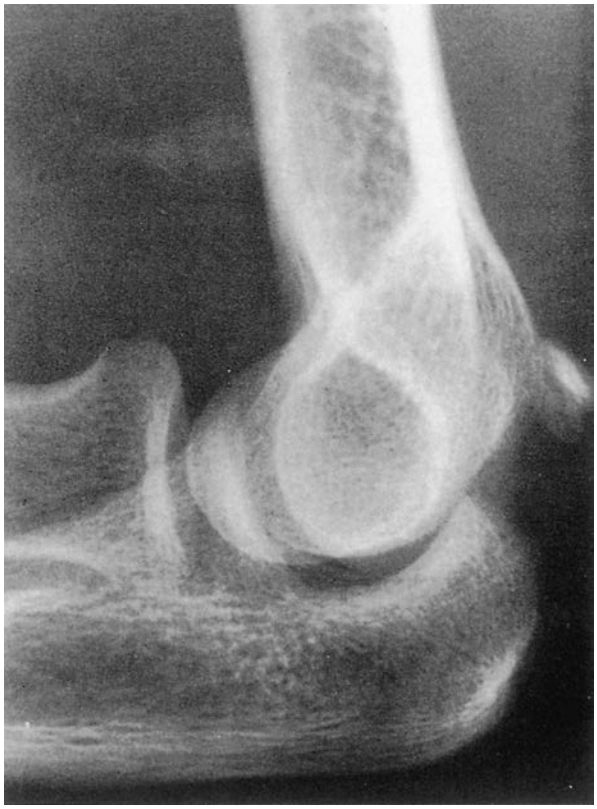

Figure 8-9 "Flake sign": A small avulsion fracture of the olecranon, best seen on a lateral radiograph of the elbow, is pathognomonic of a complete triceps avulsion. (Adapted with permission from Morrey B. Tendon injuries about the elbow. In: Morrey B. ed. *The elbow and its disorders.* Philadelphia: WB Saunders,1993: 492–504.)

are usually not necessary in cases of complete rupture. Zionts and Vachon[126] reported one case where MRI was necessary to secure the diagnosis in an adolescent boy with severe soft tissue swelling and no "flake sign."

Treatment

Surgical repair is the recommended treatment for complete avulsions. As opposed to cases of pectoralis major or distal biceps tendon ruptures for which other muscles adequately compensate for the loss of these specific muscle functions, no compensatory muscle exists to substitute for triceps function. Even in those chronically ill patients who are suboptimal surgical candidates, triceps function is essential for transfers and the use of ambulatory aids. Operative repair is, therefore, necessary in this group as well.

The surgical technique universally proposed in the literature involves anatomic reattachment of the triceps tendon through drill holes in the olecranon, most commonly using a nonabsorbable suture. Primary repair can be accomplished, even with delays in diagnosis and treatment of up to 6 months. Several case reports involving 3-, 4.5-, and 6-month delays in surgical treatment have achieved excellent results.[8,105,110] Other authors have suggested augmentation of the repair when faced with poor-quality tissue or in the setting of a late repair.[27,36,80] Bennett described reinforcing the repair by reflecting a proximally based flap of the posterior forearm fascia and suturing it to the triceps.[15] Clayton and Thirupathi supplemented a triceps repair using an inverted tongue of triceps fascia in an elderly patient with chronic bursitis.[27] Carpentier et al. described Z-plasty lengthening of the triceps to avoid undue tension when repairing a chronic rupture.[22]

The results of operative repair are uniformly excellent in the literature. Authors usually have based their results on the patients' or physicians' subjective assessment. Range of motion is almost always reported as normal. Occasionally a loss of approximately 5 degrees of terminal extension has been seen, but this trivial loss of motion has not affected the functional outcome.[20,74,106] Cybex evaluation has provided objective confirmation of strength recovery in two case reports.[8,105]

Ulnar neuritis has been seen in conjunction with triceps injury in two cases. In both instances, a delay in surgical intervention resulted in the formation of scar tissue encompassing the ulnar nerve.[4,47] Anterior transposition of the nerve at the time of triceps repair has produced satisfactory results.

Nonoperative treatment is appropriate for a partial triceps injury. The rate of progression with range of motion and strengthening exercises following 2 to 3 weeks of immobilization is dependent on the degree of muscle involvement. With adequate rehabilitation, essentially full restoration of strength and function can be anticipated after a partial tear.[7,22,36]

Authors' Preferred Treatment

Early anatomic repair through bone holes in the proximal ulna is strongly recommended for all patients with acute complete avulsions.

A posterior approach to the arm and elbow can be accomplished with the patient in the supine, lateral, or prone position. A tourniquet may be used to facilitate the dissection, particularly around the ulnar nerve. However, it must be deflated to permit maximal advancement of the triceps mechanism in the latter stages of the procedure. A posterior linear incision is used, passing lateral to the olecranon and parallel to the lateral border of the ulna (Fig. 8-10A). The ulnar nerve must be identified and protected, but formal mobilization or transposition is not necessary. The small bone fleck often associated with triceps avulsion is excised. If a large fragment has been avulsed, which usually occurs in the adolescent population, repair is amenable to tension band technique or screw-and-washer fixation.

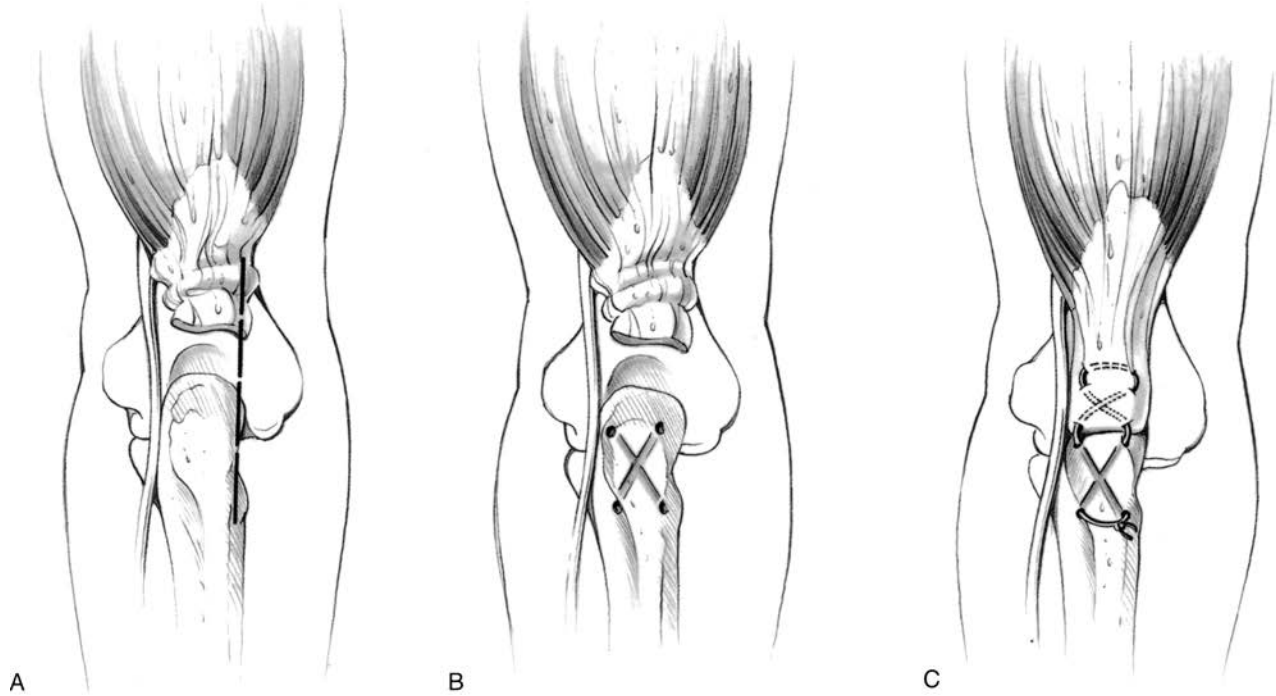

Figure 8-10 **(A–C)** Surgical technique for repair of triceps tendon avulsion. See text for details [79]. (Adapted with permission from Morrey B. Tendon injuries about the elbow. In: Morrey B. ed. *The elbow and its disorders.* Philadelphia: WB Saunders, 1993:492–504.)

A nonabsorbable no. 5 suture is woven through the freshened tendon in Bunnell fashion. Criss-crossed bone tunnels in the olecranon, as described by Morrey, is an excellent method to secure the suture (see Fig. 8-10B).[80] Again, care must be taken not to tether or entrap the ulnar nerve, which lies immediately adjacent to the repair. The suture is tied with the tourniquet deflated and the arm extended. Placing the knot on the lateral side of the ulna will help avoid ulnar nerve irritation and local tenderness when the arm is resting on a hard surface (see Fig. 8-10C). After routine closure, the arm is splinted at 30 to 45 degrees of elbow flexion, depending on the quality of tissue and tension on the repair.

We would recommend immobilizing the arm in a posterior splint for approximately 2 to 3 weeks postoperatively. The duration of postoperative immobilization in the literature ranges from 10 days to 6 weeks, averaging 3 weeks.[8] Gentle active elbow flexion and passive extension are begun 2 to 3 weeks after surgery. Resisted exercises are initiated at 6 weeks. Return to full unrestricted activity occurs between 4 and 6 months after surgery and is determined by the quality of the tissue repaired and the patient's specific functional demands.

Complications

No significant complications, such as rerupture or ulnar nerve injury, have been reported with operative repair of triceps avulsions. Wire sutures did cause a bursa over the olecranon in one case and, therefore, should probably be avoided.[84] Not a single case of failed repair was found in the literature.

SUMMARY

Complete muscle ruptures about the shoulder girdle are relatively uncommon. Unrecognized or untreated rupture of the deltoid origin will severely affect shoulder function. Complete pectoralis major rupture from its tendinous insertion will significantly impair strength in high-demand athletes and laborers. The triceps is the sole extender of the elbow, and therefore its integrity is essential for normal upper-extremity function. In general, anatomic repair of acute tendon ruptures is recommended to preserve normal shoulder function. It is difficult to predict the consequences of an isolated coracobrachialis or short head of biceps disruption. Traumatic injuries to the trapezius, latissimus dorsi, and teres major are usually intramuscular strain, although rare cases of tendon avulsion have been reported. The management of muscle belly ruptures is determined by the specific muscle involved and the extent of injury. Awareness of these injuries is the first step toward proper diagnosis and treatment.

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Glenohumeral Instability

II

9 Anatomy, Biomechanics, and Pathophysiology of Glenohumeral Instability

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INTRODUCTION

The complexity of the shoulder joint is best appreciated from our understanding of how shoulder anatomy and biomechanics are intrinsically related to the pathophysiology of shoulder instability. A multidisciplinary collaboration between surgeons, biomechanical engineers, anatomists, biochemists, and several other basic scientists is responsible for recent progress in these areas. Advances in experimental and clinical testing protocols have improved the current understanding of shoulder anatomy and biomechanics tremendously. No longer is our understanding of shoulder instability based on anecdotal and qualitative clinical experiences documenting shoulder pathology. A plethora of gross and histologic cadaveric studies, radiographic studies, and biomechanical studies now provides a sound foundation to understand how a minimally constrained articulation can balance mobility and stability. Alterations in any of the anatomic or biomechanical factors requisite for shoulder stability provide the pathogenesis for clinical instability.

The purpose of this chapter is to review the current anatomic and biomechanic factors that control glenohumeral joint motion and stability. Because anatomy and biomechanics are two interdependent disciplines, they will be discussed together as each of the relevant structures is reviewed. A deeper understanding of this relation will provide substantive data critical to classify and appreciate the pathophysiology of glenohumeral instability. More importantly, the application of anatomic and biomechanic principles provides a rational approach to the

treatment of glenohumeral instability for optimal functional restoration.

DEFINING THE PROBLEM

Laxity is asymptomatic, passive translation of the humeral head on the glenoid as determined by clinical examination and is unassociated with pain. Laxity is required for normal glenohumeral motion and may be affected by age,[62] gender, and congenital factors. In general, laxity changes with the position of the arm. At the extremes of rotation, the static restraints tighten and decrease laxity. Whether laxity is a risk factor for the development of clinical instability is a matter of debate.[184]

Instability is a pathologic condition that manifests as pain or discomfort in association with excessive translation of the humeral head on the glenoid fossa during active shoulder motion. Both clinical[48,62,73] and experimental studies[84,85,89,149,224,258,259,267] demonstrate a wide range of normal "play" in the glenohumeral joint; thus, it is the association with symptoms that clearly separates instability from excessive laxity. A spectrum of instability exists, representing increasing degrees of injury and dysfunction of the dynamic and static factors that function normally to contain the humeral head within the glenoid. Patients with multidirectional instability subluxate or dislocate in multiple directions, with concurrent reproduction of symptoms in at least two directions. Symptoms typically occur at midrange positions of glenohumeral motion, and often are associated with activities of daily living.[16,222] Recently, stability has been quantified as the force required to sublux the joint by a specified amount of translation. This has facilitated the study of the combined effects of muscle and capsular loads to joint stability and, therefore, represents an important advance in modeling the in vivo mechanical environment of the glenohumeral joint.[28,143,231]

ANATOMIC AND BIOMECHANIC CONSIDERATIONS

Anatomic control of glenohumeral joint stability can be divided into static (e.g., ligaments and tendons) and dynamic (e.g., muscular contraction) factors (Table 9-1). The cooperative role that these factors play is complex, and no single factor is responsible for glenohumeral joint stability. Similarly, no single lesion is responsible for clinical instability (Table 9-2). Contemporary approaches to the treatment of glenohumeral joint instability are directed at restoring normal anatomy and biomechanics, as opposed to simply constraining motion, as has been historically described.[46,64]

Establishing criteria for "normal" and "pathologic" conditions of the glenohumeral joint is often difficult owing

TABLE 9-1
FACTORS MAINTAINING JOINT STABILITY

Static Factors
Articular components
Articular version
Glenoid labrum
Negative intraarticular pressure
Adhesion–cohesion
Capsule and ligament
Rotator cuff
Dynamic Factors
Rotator cuff
Biceps brachii
Scapular rotators
Proprioception

TABLE 9-2
NORMAL AND ABNORMAL ANATOMY AND BIOMECHANICS

Stability Factor	Pathoanatomy
Glenoid version	Congenital: abnormal version; dysplasia Fracture causing abnormal version
Humeral version	Congenital: abnormal version; dysplasia Fracture/surgery causing abnormal version
Articular congruity	Congenital: dysplasia Acquired: fracture, Bankart lesion, osteoarthritis
Labrum	Bankart lesion "Fraying" secondary to laxity
Capsuloligamentous	Traumatic tear, cumulative microtrauma with plastic deformation Congenital laxity Loss of proprioceptive feedback
Negative intraarticular pressure	Capsular tear "Rotator interval" defect Lax capsule
Rotator cuff deficiency	Traumatic tear, cumulative microtrauma
Biceps	SLAP lesion Tendon rupture
Scapulothoracic motion	Dyskinesis: fatigue and weakness of serratus Long thoracic nerve palsy

SLAP, superior labrum from anterior to posterior.

to the considerable individual variation in capsuloligamentous anatomy[58,64,180,182,258,259] and in inherent shoulder laxity.[62,84,85,139] Dynamic factors (i.e., rotator cuff and biceps) are affected by their level of conditioning and strength. Scapulothoracic motion[263] is a more subtle, but equally important, dynamic factor helping to maintain shoulder stability. To add to this complex equation, an interaction between static capsuloligamentous factors and dynamic muscular factors, mediated through proprioception, has been postulated.[25,42,135,136,139,243,252]

Anatomic and biomechanic dysfunction leading to glenohumeral instability results from varying levels of applied stress (i.e., a single traumatic event vs. cumulative microtrauma), the relative risk of injury associated with an activity, the quality and integrity of the static stabilizers, and the strength and conditioning of the dynamic stabilizers. An individual's "susceptibility" for glenohumeral instability is dependent on these factors, each of which will be discussed in the following sections. The consequences of deficiency of any one component will be presented.

Static Factors

Articular Version

Both arthrographic and roentgenographic studies have characterized the relation between the humeral head and the glenoid surface of the scapula. With the arm hanging at the side in an adducted position, the scapula faces 30 degrees anteriorly on the chest wall and tilts 3 degrees upward relative to the transverse plane and 20 degrees forward relative to the sagittal plane (Fig. 9-1).[180] As described by Saha,[218] in 75% of persons the average glenoid orientation is in 7 degrees of retroversion, with 25% of persons having anteversion ranging from 2 to 10 degrees. Churchill[43] et al. evaluated 334 cadaveric scapulae and found that Caucasians have more retroversion than African Americans. Further, there is no difference in retroversion seen between males and females. Saha[218] and others[14] have observed that the glenoid has an average superior tilt of 5 degrees (Fig. 9-2). Scapular inclination may have a contributory role in controlling inferior stability.[259] However, the difficulty in interpreting these studies arises from a wide range of interindividual variability, the reproducibility of techniques used to measure these factors, and an unknown relative contribution to clinical instability.

Recently, the anatomy of the proximal humerus has been significantly clarified. Saha[218] was one of the first to radiographically show that the neck-shaft angle averages 130 to 140 degrees and retroversion averages 30 degrees relative to the transepicondylar axis of the distal humerus (see Fig. 9-2).

Walch et al.,[254] using a micron precision probe and a computer to render a three-dimensional image of 65 humeri, determined that the inclination of the articular surface varies between 114 and 147 degrees with an average value of 130 degrees. In this same study, the average humeral retroversion measured 17.9 degrees (range, –6.5 to 47.5 degrees).

Articular Conformity

The glenohumeral joint in the adult consists of the humeral head and glenoid surface of the scapula. Understanding the glenoid and humerus as separate, but interdependent, congruent structures is integral to appreciate how

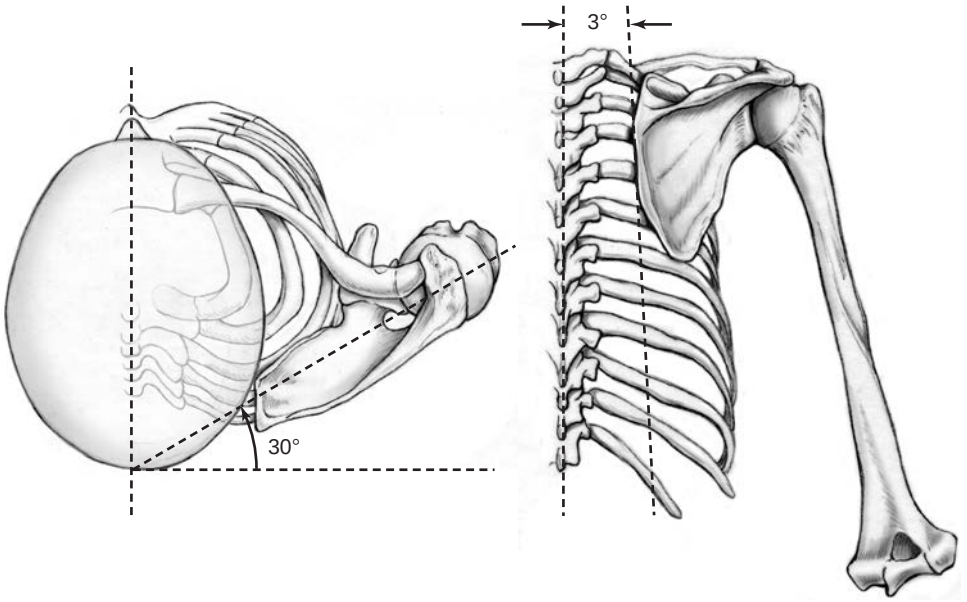

Figure 9-1 Scapular orientation on chest wall. **(Left)** 30 degrees anterior. **(Right)** 3 degrees upward. (Adapted from Warner JJP. The gross anatomy of the joint surfaces, ligaments, labrum and capsule. In: Matsen FA III, Fu FH, Hawkins RJ, eds. *The shoulder: a balance of mobility and stability.* Rosemont, IL: American Academy of Orthopaedic Surgeons, 1993:9.)

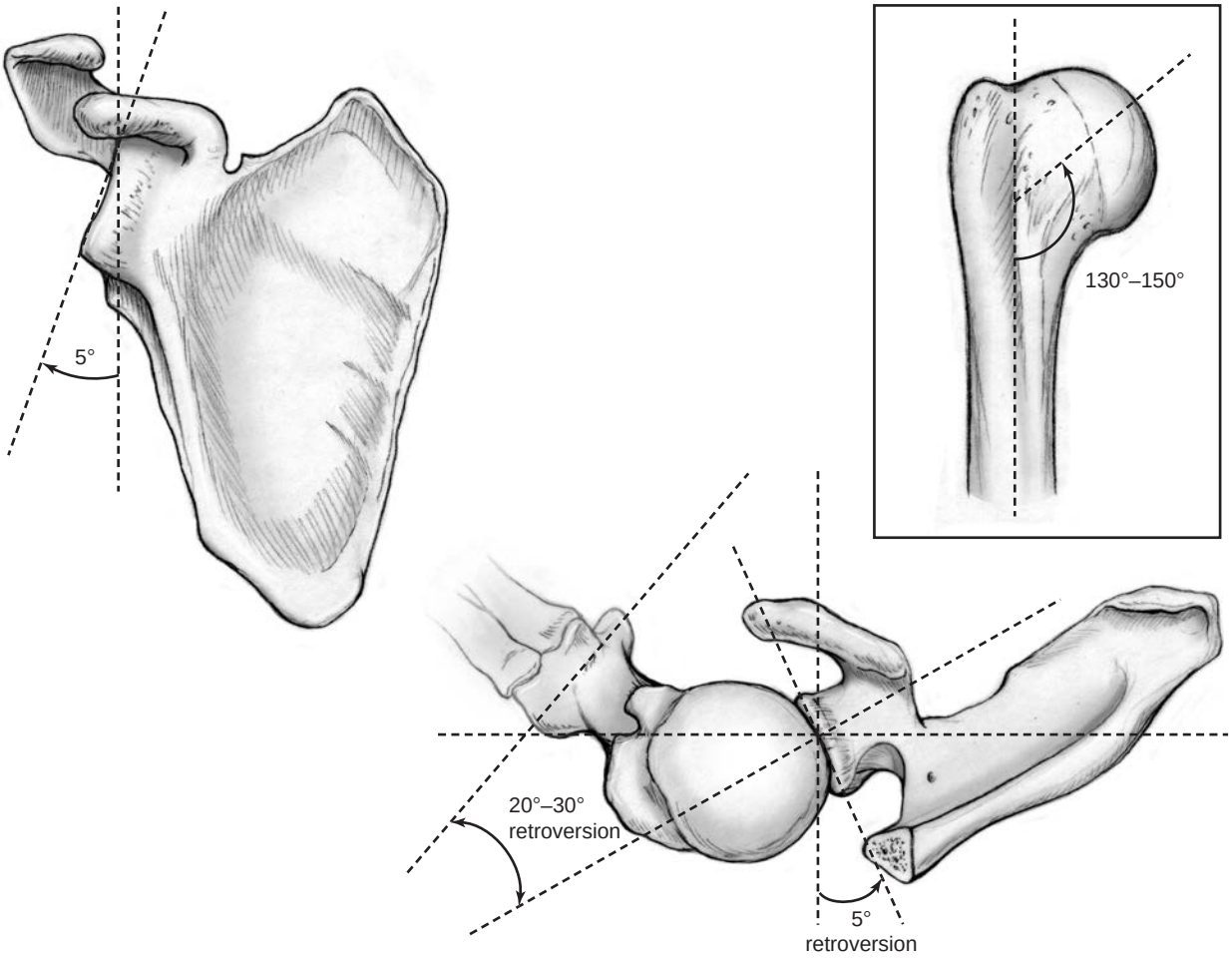

Figure 9-2 **(Left)** Superior tilt of the glenoid (see text). (From Warner JJP. The gross anatomy of the joint surfaces, ligaments, labrum and capsule. In: Matsen FA III, Fu FH, Hawkins RJ, eds. *The shoulder: a balance of mobility and stability.* Rosemont, IL: American Academy of Orthopaedic Surgeons, 1993:9, with permission.) **(Center and right)** Glenoid and humeral version, and neck-shaft angle of proximal humerus (see text). (Adapted from Warner JJP, Caborn DNM. Overview of shoulder instability. *Crit Rev Phys Rehabil Med* 1992;4:145–198.)

these two joint surfaces can maintain stability yet provide for a relatively large range of motion. Congruence can be defined as the difference in the radii of the humeral head and the glenoid articulating surfaces. The closer the difference is to 0, the more congruent is the joint.[23,229] This congruent articulation provides the foundation for the rotator cuff to establish a concavity–compression effect as it dynamically compresses the convex humeral head into the matched concavity of the glenoid.[140] Furthermore, as an extension of the glenoid, the labrum functions to increase the depth and surface area of the glenohumeral articulation, enhancing this effect.[95]

The glenoid surface is "pear-shaped," similar to an inverted comma, being approximately 20% narrower superiorly than inferiorly (Fig. 9-3). The average vertical and transverse dimensions are 35 and 25 mm, respectively.[43] In contrast, the larger humeral head has vertical and transverse dimensions averaging 48 and 45 mm, respectively.[180] Approximating a sphere, the humeral head has a surface area that is three times that of the glenoid.[229] In any position of rotation there is a surface area mismatch such that only 25% to 30% of the humeral head is in contact with the glenoid surface.[220] In other words, the glenoid's relatively smaller surface area is insufficient to cover the humeral head. This emphasizes the importance of the soft tissues and muscles surrounding the joint in providing stability during shoulder function.

Walch and Boileau[256] determined that the humeral head is comparable to a sphere in 90% of the 160 humeri they examined, with the articular surface constituting about one-third of the sphere. The diameter of the head was variable with an average of 43.2 mm (range, 36.5 to 51.7 mm) with an articular cartilage thickness on average of 15.2 mm (range, 12 to 18 mm). They demonstrated that the spherical humeral head sits with a frontal plane medial offset of 6.9 mm on average (range, 2.9 to 10.6 mm) and a

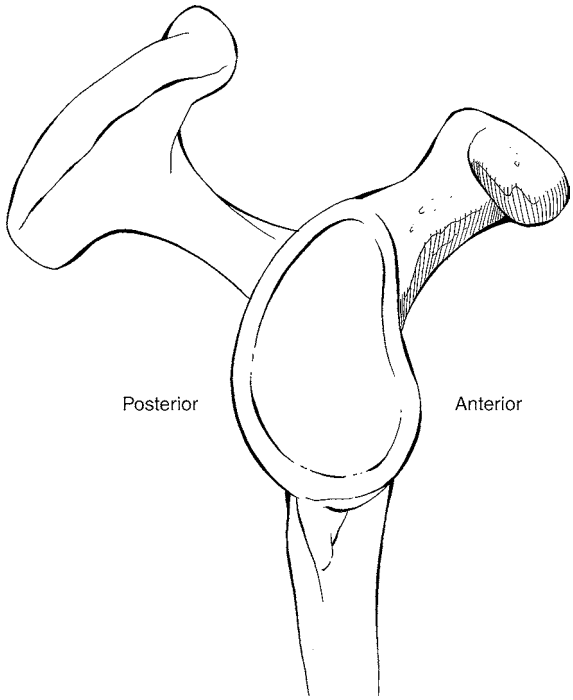

Figure 9-3 The "pear-shaped" face of the glenoid that articulates with the humeral head. (From Boardman ND III, Fu FH. Shoulder biomechanics. In: McGinty JB, Caspari RB, Jackson RW, Poehling GG, eds. *Operative arthroscopy.* Philadelphia: Lippincott-Raven, 1996:627, with permission.)

sagittal plane posterior offset of 2.6 mm (range, 0.8 to 6.1 mm). These parameters, however, may have more implications for shoulder arthroplasty design than for shoulder stability. Similarly, Iannotti et al.[100] reported that the humeral head approximates a sphere in the central articular areas and becomes slightly elliptical peripherally. Variations in these findings and their effect on the pathophysiology of shoulder instability are not clearly understood.

Conceptually, the glenohumeral joint has been compared to a "golfball sitting on a tee"[180] (Fig. 9-4). This analogy is based on historical beliefs sighting the relatively small area of the glenoid and its relative shallowness compared with the humeral head, allowing only a limited portion of the humeral head to contact the glenoid in any single shoulder position.[32,218,219] In fact, the articular surfaces of the humeral head and glenoid are almost perfectly matched with a congruence within 3 mm, with deviations from sphericity of less than 1%.[166,229] Additionally, the cartilage of the glenoid is thicker peripherally, and thus, plain radiographs tend to underestimate the relative concavity of the glenoid. This would imply that the glenohumeral joint would function similar to a ball-and-socket articulation as described by Kelkar et al.[118,119]

Although some coupled translation occurs at the extremes of glenohumeral rotation,[84,85,139] tracking of the geometric center of the cartilaginous articular surface with simulated muscle forces actually approximates ball-in-socket motion.[119] In the stable shoulder, external and internal rotation is associated with posterior and anterior humeral head translation, respectively. These relations may be altered in the unstable shoulder. McMahon et al.,[154] in an elegant model using a dynamic shoulder testing apparatus, measured muscle force values and tendon excursions across the glenohumeral joint during abduction in the scapular plane. They determined that humeral head translations on the glenoid were less than 2 mm under all testing conditions used and that the joint does behave kinematically as a "ball-and-socket" articulation during glenohumeral abduction. The importance of these findings is that articular incongruency is probably less of a predisposing factor for instability

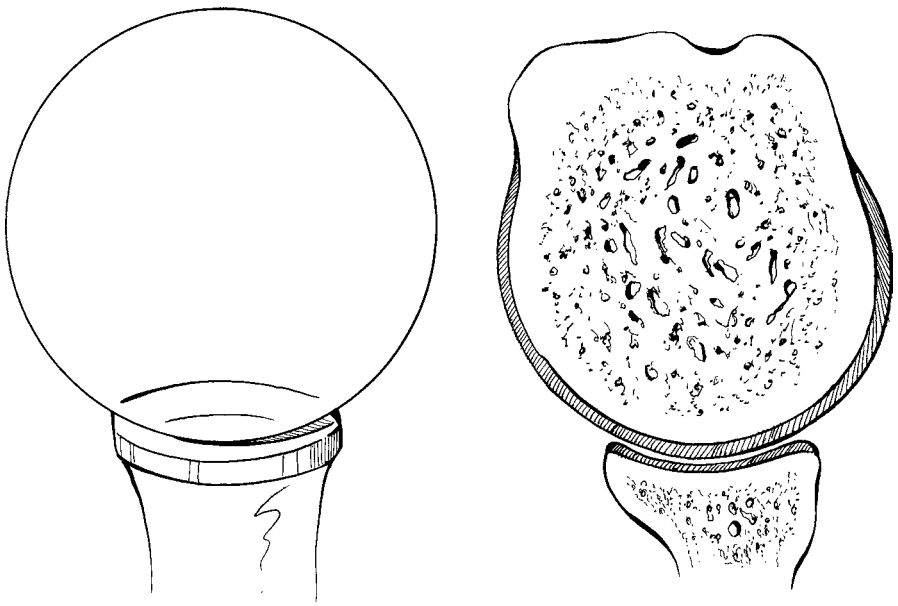

Figure 9-4 Analogy of the glenohumeral joint to a golf ball and tee. (From Boardman ND III, Fu FH. Shoulder biomechanics. In: McGinty JB, Caspari RB, Jackson RW, Poehling GG, eds. *Operative arthroscopy.* Philadelphia: Lippincott-Raven, 1996:627, with permission.)

than is surface area mismatch, as seen in glenoid dysplasia or glenoid fracture.[9,61,124,138,213,215] Additionally, the integrity of the soft tissues influences coupled translation, and this may also be a greater predisposing factor to instability than is articular incongruency.[35,84,94,95]

Glenoid Labrum

The relative lack of depth and surface area of the bony glenoid is compensated by the fibrous labrum acting to maintain normal glenohumeral biomechanics. As determined by Cooper et al.,[51] the labrum is a fibrous ring attaching to the glenoid articular cartilage through a narrow fibrocartilaginous transition zone. Above the glenoid equator, the labrum is relatively more mobile. In contrast, below the equator, the labrum is more consistently tightly attached to the glenoid articular cartilage. The tendon fibers of the long head of the biceps brachii blend with the superior labrum, and the inferior glenohumeral ligament (IGHL) blends into the inferior labrum. Cooper et al.[51] examined the vascular supply of the labrum and found that the superior and anterosuperior parts of the labrum were less vascular than the posterosuperior and inferior portions. Blood supply was limited to the periphery.

The labrum contributes to stability of the glenohumeral joint through several mechanisms. It acts as an anchor point for the capsuloligamentous structures.[51,164,204] Howell and Galinat[95] have shown how the labrum enhances stability by deepening the concavity of the glenoid socket to an average of 9 and 5 mm in the superoinferior and anteroposterior planes, respectively (Fig. 9-5). Loss of the labrum decreases the depth of the socket by 50% in either direction. Functionally, this acts as a "chock-block" preventing the head from slipping over the edge of the glenoid. Resection of the labrum reduces resistance to translation by 20%, and it is especially effective in doing so in combination with joint compression in the midrange of glenohumeral motion.[102,140] The labrum also acts to increase the surface area of contact, acting as a load-bearing structure similar to the function of the meniscus in the knee.[34,230] Unlike the meniscus, however, the labrum lacks the microscopic architecture to disperse hoop stress and, therefore, is unlikely to effectively bear load.

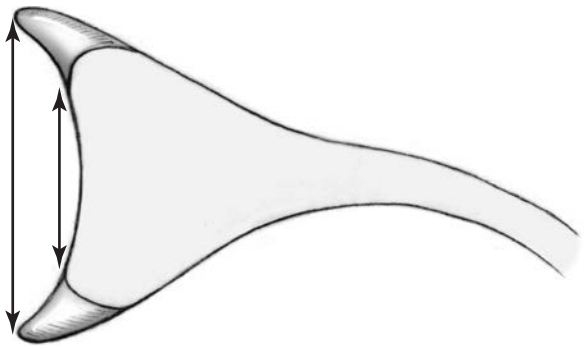

Figure 9-5 Glenoid labrum increases the surface area and depth of the glenoid socket. (Adapted from Warner JJP, Caborn DNM. Overview of shoulder instability. *Crit Rev Phys Rehabil Med* 1992;4:145–198.)

Vanderhooft et al.[250] and Bowen et al.[35] have shown that the labrum plays a significant stabilizing role during rotator cuff contraction, facilitating the concavity–compression mechanism as the humeral head is compressed into the glenoid. Lazarus et al.[130] have defined a *stability ratio,* a measure of the effectiveness of concavity–compression in the stabilization of the glenohumeral joint, as the ratio between the maximum dislocating force that can be stabilized in a given direction and the load compressing the head into the glenoid. They determined that by creating a chondral–labral defect, an 80% reduction in the height of the glenoid occurred, with a concomitant reduction in the stability ratio of 65% for translation in the direction of the defect.

A Bankart lesion represents a lesion of the labrum corresponding to the detachment of the anchoring point of the IGHL and middle glenohumeral ligament (MGHL) from the glenoid rim.[12,213] A Bankart lesion disrupts the concavity–compression effect during rotator cuff contraction, eliminates the "chock-block" effect, and decreases the depth of the socket by 50% with detachment of the capsuloligamentous structures. This lesion should not be confused with the normal anatomic variants of a sublabral sulcus underneath a cord-like MGHL, the Buford complex,[275] or a loosely attached labrum superiorly. Whether the Bankart lesion is the "essential lesion" leading to recurrent anterior instability, as suggested by several authors, is currently a topic of interest.[12,32,164] Isolated detachment of the labrum as a singular entity leading to recurrent instability was challenged early in the literature by Townley[245] and then by Speer et al.[232] Normal variations of the capsulolabral complex occur commonly.

The difference between acute and chronic shoulder instability is important clinically. In acute shoulder instability, the authors feel that the labrum is the "essential" lesion. In a classic article by Arciero et al.,[8] repair of the Bankart significantly reduced the recurrence rate of anterior shoulder dislocation. In patients with chronic anterior instability, it is the capsule and labrum combined, or capsulolabral tissue, that is the essential lesion.[148]

Pagnani et al.[190] have demonstrated the importance of the superior portion of the glenoid labrum. In a cadaver study, isolated lesions of the anterosuperior portion of the labrum did not have a significant effect on glenohumeral translation. However, complete lesions of the superior portion of the labrum associated with destabilization of the biceps insertion resulted in significant increases in anteroposterior and superoinferior glenohumeral translations in the lower and middle ranges of elevation. The implications of these findings are that destabilization of the glenoid insertions of the superior glenohumeral ligament (SGHL), MGHL, and biceps insertion may be associated with subtle increases in translation and may be

related to the symptoms present in patients who have isolated lesions of the superior labrum.[190,228]

Negative Intraarticular Pressure

In normal shoulders, a relative vacuum exists as a result of high osmotic pressure in the interstitial tissues, causing water to be drawn out of the glenohumeral joint.[147] As the articular surfaces are pulled apart, a suction effect develops to resist further displacement. The magnitude of this negative pressure has been shown to be about –42 cm of water in the adducted and relaxed shoulder. This increases to –82 cm of water during the application of a 25-N inferior force applied to the cadaver shoulder.[37] Negative intraarticular pressure becomes especially important when the rotator cuff is not contracting or when tension has not developed in the superior and coracohumeral ligaments during glenohumeral motion.

Pathologic conditions may include those that cause venting of the glenohumeral joint, leading to inferior subluxation, as has been shown experimentally by Warner et al.[257] and others.[127] Wuelker et al.[274] observed that venting of the joint increased displacement significantly in the anterior, posterior, and inferior planes. Anterior translation also increased by 55%[77] after capsular venting. This restraint becomes negligible, however, when the muscles contract with shoulder abduction or when the IGHL or superior capsular structures are under tension.[37,260] In a study by Helmig et al.,[91] venting of the capsule led to significant increases in anteroposterior translation and external rotation. The implications of their findings were that evaluation of shoulder stability in biomechanical investigations should be performed before violation of the negative intraarticular pressure mechanism occurs, or at the very least, measures should be corrected for this factor.

Thus, as a static restraint, negative intraarticular pressure appears to be important in limiting translation of the humeral head. Habermeyer et al.[82] noted that the presence of a Bankart lesion somehow eliminated the intraarticular seal to atmospheric pressure. A traumatic capsular rupture or an enlarged rotator interval capsular defect, possibly present at birth, presumably could lead to excessive glenohumeral translation, predisposing to instability.[49] Recently, Hashimoto et al.[87] have indicated that dynamic changes in intraarticular pressure can help differentiate patients with adhesive capsulitis, partial- and full-thickness rotator cuff tears, and instability. Practically, from a clinical perspective, reestablishing negative intraarticular pressure remains a theoretical concern and plays no role in the treatment of shoulder instability.

Adhesion–Cohesion

The glenohumeral joint contains less than 1 mm of synovial fluid that provides articular nourishment through diffusion and lubrication through several mechanisms (e.g., hydrodynamic, boundary, weeping, or boosted). Viscous and intermolecular forces help to create this adhesion–cohesion effect. Functionally, this is a stabilizing mechanism that permits sliding motion between the two joint surfaces while simultaneously limiting them from being pulled apart.[147] This is analogous to two glass plates separated by a thin film of water that slide easily over one another, but are difficult to separate. Negative intraarticular pressure and adhesive forces resulting from the presence of synovial fluid between the articular surfaces contribute static restraint, particularly when the capsule is lax and the muscles are relatively inactive.[101,127,145] Clinically, these factors probably play a minor role in maintaining glenohumeral stability and only at very low load levels.

Capsuloligamentous Structures

Few structures in the shoulder have received as much attention by investigators as the capsule and ligaments surrounding the glenohumeral joint. Traditionally, the ligaments of the capsule were described as discreet thickenings constituting the "glenohumeral ligaments."[58,180,183,221] Clinical observations at the time of surgery[4,32,70,164,169,192,213,215,217] or by cadaver shoulder dissections[12,45,58,64,95,126,133,174,182,213,217,246] Burkart and Debski[37a] have enhanced our understanding of these structures from an anatomic perspective. To obtain a concise appreciation of these structures, anatomic investigations must minimally distort the ligamentous relationships (Fig. 9-6). With increasing sophistication, the biomechanical function,[56,57,77,126,134,144,155,156,158,181,183,186,210,212,234,257,259] material properties,[21,29,56,83,117,134,144,155,156,158,207,210,234,244,257] and the interrelation of the rotator cuff and capsule have been described.[40,96,132,151,174,212,258] Synthesizing the available data into a cohesive algorithm applicable to the clinical setting of glenohumeral instability is a formidable task for most.

In a classic anatomic study, DePalma et al.[58] described the variability of the shoulder capsule, categorizing it into six basic types based on the pattern of the synovial recesses. Other anatomic studies have since confirmed and clarified the variable architecture of the glenohumeral ligaments.[64,164] Contemporary investigators suggest, as did DePalma et al.,[58] that some anatomic findings correlate with the risk of developing shoulder instability.[161,259] The basis for the functional roles of the capsular structures lies in their anatomic arrangement throughout the capsule. A significant advance in our knowledge came from Turkel et al.,[246] who confirmed by anatomic radiographic studies that different portions of the capsuloligamentous complex provided static stability that depends on arm position and the direction of the load applied to the proximal humerus.

The glenohumeral capsule is thin, less than 5 mm in thickness.[44] The glenohumeral ligaments function principally during rotation of the arm to reciprocally tighten and loosen, thus limiting translation and rotation in a

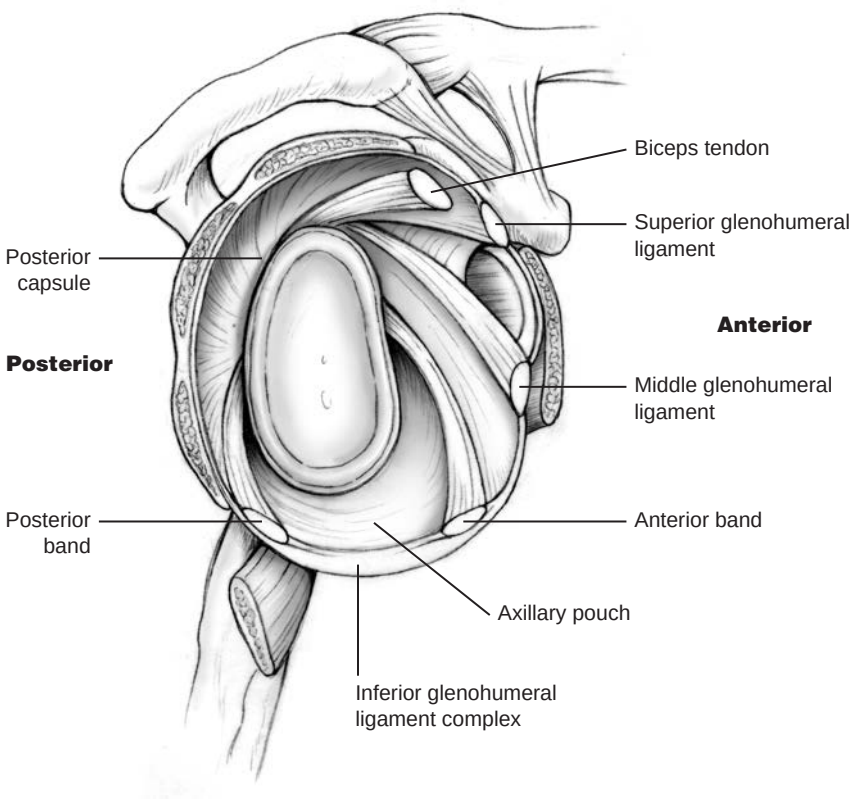

Figure 9-6 Capsuloligamentous anatomy viewed from the side with the anterior aspect to the right and the posterior aspect to the left. The humeral head has been removed, leaving the glenoid. The superior glenohumeral ligament and middle glenohumeral ligament are labeled. The inferior glenohumeral ligament complex consists of an anterior band, posterior band and interposed axillary pouch. The posterior capsule is the area above the posterior band. The biceps is also labeled. (Adapted from O'Brien SJ, Neves MC, Arnoczky SP, et al. The anatomy and histology of the inferior glenohumeral ligament complex of the shoulder. *Am J Sports Med* 1990;18: 449–456.)

load-sharing fashion.[261] In the midrange of rotation when these structures are relatively lax, stability is maintained primarily by the action of the rotator cuff and biceps through the concavity–compression effect across the glenohumeral joint.[85,140] The ligaments principally protect against instability when the joint is placed at the extremes of motion and become especially important when all other stabilizing mechanisms have been overwhelmed. [57,84,85,147,258]

Clinically, this becomes important during capsular reconstruction. Tensioning these structures in the midrange can potentially overtighten and constrain the joint, limiting rotation.[76,141,263] In the extreme case, this may lead to posterior humeral subluxation or arthritis.[18,22] The static role of each component of the capsuloligamentous structure is summarized in Table 9-3. In the sections that follow, each structure will be reviewed in terms of contemporary research describing the anatomy and biomechanics as well as a limited discussion on the relevant material properties.

Superior and Coracohumeral Ligaments

These structures are considered together because their anatomic courses are parallel and they constitute the reinforcing structures of the "rotator interval" region.[29,49,86,169,174,180,213] The rotator interval subtends a medially based triangular space, bordered superiorly by the anterior margin of the supraspinatus tendon, inferiorly by the superior border of the subscapularis tendon, medially by the base of the coracoid, and laterally by the long head of the biceps tendon and sulcus. The floor of the rotator interval is normally bridged by capsule. Occasionally, a complete opening within the tissue spanning the rotator interval is present and is described as a "rotator interval capsular defect" (Fig. 9-7).

The coracohumeral ligament (CHL) is a dense fibrous extraarticular structure originating on the lateral surface of the coracoid process as a broad (1 to 2 cm) and thin structure. It inserts into the greater and lesser tuberosities adjacent to the bicipital groove and becomes intermingled with the tendinous edges of the supraspinatus and subscapularis, respectively.[58,86] Cooper et al.[52] questioned the significance of the CHL, describing it as a capsular fold creating a "pup tent" of capsule within the rotator interval. Others argue that the CHL is a well-defined structure that prevents excessive inferior translation of the adducted humerus in either position of humeral rotation.[171,186] While the specific function of the CHL remains disputed, it is known to be geometrically more robust and mechanically stiffer than the SGHL.[30] It also provides stability to the biceps tendon.

The SGHL lies deep to the CHL, is variable in size, and is present in over 90% of cases.[58,180,183,217,224,259] Usually quite diminutive, it originates from the superior glenoid tubercle just inferior to the biceps tendon and runs parallel to the CHL as it inserts into the superior aspect of the lesser tuberosity just medial to the bicipital groove.

TABLE 9-3
FUNCTIONS OF THE LIGAMENTS

Author	SGHL	CHL	MGHL	IGHLC	Other
Turkel et al.[246]	Little role in anterior stability		Primary stabilizer for anterior stability at 45 degrees of ABD; limits ER in mid-ABD	Primary stabilizer for anterior instability in ABD	Subscapularis is secondary stabilizer at 45 degrees of ABD
Ovesen and Nielsen[185–187]	Secondary stabilizer to posterior instability	Secondary stabilizer to posterior instability; primary stabilizer against inferior instability in ABD	Important for anterior instability at 45 degrees of ABD	Posterior capsule plays role in anterior and posterior stability	
Schwartz et al.;[224] O'Brien et al.[181]	Little role in stability			1-degree and 2-degree stabilizers against anterior and posterior instability in ABD	
Basmajian and Bazant[14]	Primary restraint to inferior translation in ADD	Primary restraint to inferior translation in ADD			
Warren et al.[266]	Secondary restraint to posterior instability in ADD, flexed, IR				Posterior capsule is primary restraint to posterior translation
O'Connell et al.[183]	Primary restraint to ER in ADD		Secondary restraint to ER in ABD; primary restraint to anterior instability at 45 degrees of ABD		
Ferrari[64]	Primary restraint to ER in lower range of ABD		Important restraint to ER at 60 and 90 degrees of ABD		
Helmig et al.[90]		Primary restraint to inferior instability			
Harryman et al.[84]		Primary restraint to ER			
Warner et al.[255]	Primary restraint to inferior translation in ADD	Minimal role in inferior stability	Secondary stabilizer for inferior translation in ADD	Primary stabilizer to inferior translation in ABD and secondary stabilizer in ADD	

ABD, abduction; ADD, adduction; CHL, coracohumeral ligament; ER, external rotation; IGHLC, inferior glenohumeral ligament complex; IR, internal rotation; MGHL, middle glenohumeral ligament; SGHL, superior glenohumeral ligament.

Opinions vary on the specific functions of these two ligaments. Harryman et al.[86] characterized the relative biomechanical contribution of the rotator interval capsule to shoulder stability in cadaver specimens. A transverse incision in the rotator interval region including the capsule, CHL, and SGHL allowed statistically significant increases in humeral head translations in all planes tested. Imbrication of the rotator interval decreased inferior translation in adduction and posterior translation in flexion to less than the intact state. No attempt was made to isolate the role of specific capsular ligaments. Burkart and Debski[37a] performed a selective sectioning study of these ligaments and concluded that the SGHL is an important stabilizer in the anterior direction. The SGHL also limits external rotation of the adducted arm.[37a] Basmajian and Bazant,[14] using electromyographic and anatomic dissections, showed that the

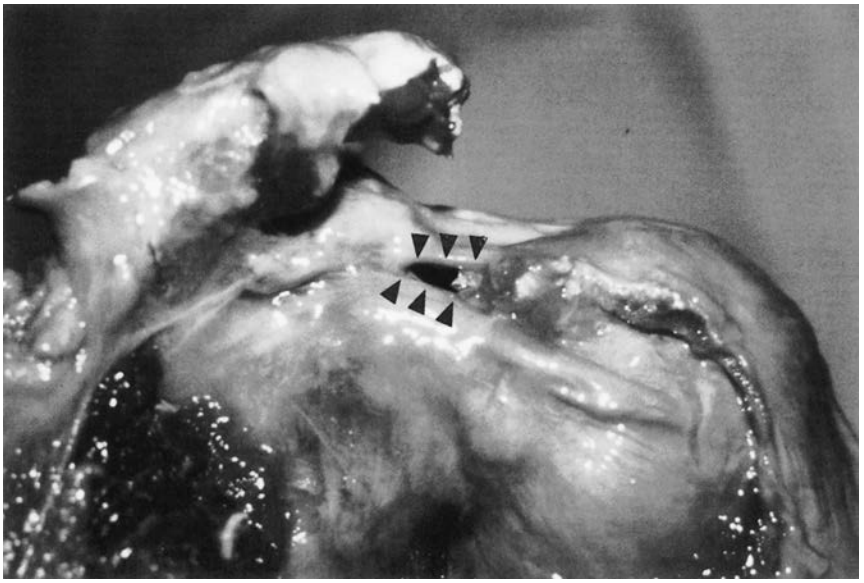

Figure 9-7 Gross anatomic specimen of a left shoulder demonstrating the opening within the rotator interval situated between the supraspinatus and subscapularis muscle tendons.

superior capsule and the CHL resisted downward displacement with the arm adducted, independent of load.

Patel et al.[193] described the CHL to consist of an anterior and posterior band originating at the coracoid and inserting into the lesser and greater tuberosities, respectively. During adduction and external rotation, the SGHL and anterior band of the CHL shortened from a maximally lengthened position. These changes were opposite those of the posterior band of the CHL that was maximally lengthened with adduction and internal rotation. Warner et al.[259] suggested that the SGHL resists inferior translation of the adducted shoulder and that the CHL is not important here. However, subsequent work by Boardman et al.[29] suggested that the CHL is the principal functional component of the capsule within the rotator interval. Observations by Lee et al.[133] suggest that the coracoacromial ligament has a role in static restraint of the glenohumeral joint as well. These authors propose that the coracoacromial ligament interacts with the CHL to prevent anterior and inferior translation, particularly between 0 and 30 degrees of abduction.

Despite varying opinions in the literature (see Table 9-3), the current consensus is that these two structures constrain the humeral head on the glenoid, limit inferior translation and external rotation when the arm is adducted, and limit posterior translation when the shoulder is in a position of forward flexion, adduction, and internal rotation. There has been renewed interest in this portion of the shoulder capsule because openings within the rotator interval have been associated with recurrent anteroinferior and multidirectional instability.[65,86,175,213] As suggested by several of these studies, addressing this pathology may be important in preventing recurrence. Conversely, contracture or scarring of this portion of the shoulder capsule has been associated with adhesive capsulitis.[86,171]

Middle Glenohumeral Ligament

As described by DePalma[58] and others,[180,182,224,258,259] the MGHL has the greatest variation in size and presence of all the ligaments of the shoulder. It is absent or poorly defined in 40% of individuals.[64,182] It originates from the supraglenoid tubercle and anterosuperior labrum, often along with the SGHL, and inserts just anterior to the lesser tuberosity, blending with the posterior aspect of the subscapularis tendon. Its variable morphology usually takes one of two forms: (a) sheet-like and confluent with the anterior band of the IGHL or (b) cord-like, with a foraminal separation between it and the anterior band of the IGHL. Moseley and Overgaard[164] reported that the MGHL originated from the scapular neck and formed an anterior pouch accommodating the humeral head in some patients with recurrent anterior instability.

It is generally believed that the MGHL functions as a passive restraint to both anterior and posterior translation of the humeral head when the arm is abducted in the range from 60 to 90 degrees in external rotation and limits inferior translation when the arm is adducted at the side. Those who are "MGHL dominant" individuals with a cord-like MGHL may be more dependent on this structure to provide a protective role against anterior instability.[161,259] Clinically, the MGHL may be detached from the anterior glenoid and constitutes the leading edge of a Bankart lesion, which typically includes the anterior band of the IGHL. However, the sublabral hole should not be confused with a detached labrum, for generally the labrum is more mobile above the equator of the glenoid.

Inferior Glenohumeral Ligament Complex

Originally described by DePalma,[58] several descriptions of the IGHL exist in the literature ranging from a triangular-shaped structure coursing from the labrum to the humeral neck, to one with well-defined thickenings at its leading edge.[182,244,246] Typically, it originates from the anteroinferior labrum or inferior half of the neck of the glenoid adjacent to the labrum and inserts just inferior to the MGHL at the humeral neck. Our current understanding has advanced to the point at which we now consider this structure to be quite developed, with very specific functions attributed to its individual components. O'Brien et al.[182] have defined this structure, through arthroscopic, gross, and histologic evaluation, as the inferior glenohumeral ligament complex (IGHLC) consisting of three components. They described discrete anterior and posterior bands (ligament) with an interposed thinner axillary pouch (see Fig. 9-6). The complex consists of three well-defined layers of collagen fibers extending from the glenoid to the humerus (inner and outer) and running circumferentially around the joint (middle). Ticker et al.[244] and Bigliani et al.[21] have recently challenged the presence of a discrete posterior band and found all regions of the IGHLC to be thicker near the glenoid than the humerus.

The IGHLC contributes to glenohumeral stability in several ways. Recently, Kuhn et al. found that the IGHLC is a restraint to external rotation of the arm in neutral and abducted positions.[126] O'Brien et al.[182] suggested that the IGHLC functions as a hammock to support the humeral head as it undergoes reciprocal tightening–loosening with abduction or rotation as the orientation of the complex changes. In adduction, it forms a dependent fold, acting as a secondary restraint limiting large inferior translations.[182,259] In abduction, however, this complex moves underneath the humeral head, becoming taut, in the fashion of a hammock, effectively limiting inferior translation. As the arm is internally rotated, the complex moves posteriorly, and as the arm is externally rotated, the complex moves anteriorly, forming a barrier to posterior and anterior dislocation, respectively (Fig. 9-8). Horizontal flexion and extension in abduction will also tighten the posterior or anterior components, respectively, thereby limiting anteroposterior translation.[224] Another biomechanical study by O'Brien et al.[181] verified that the primary anteroposterior

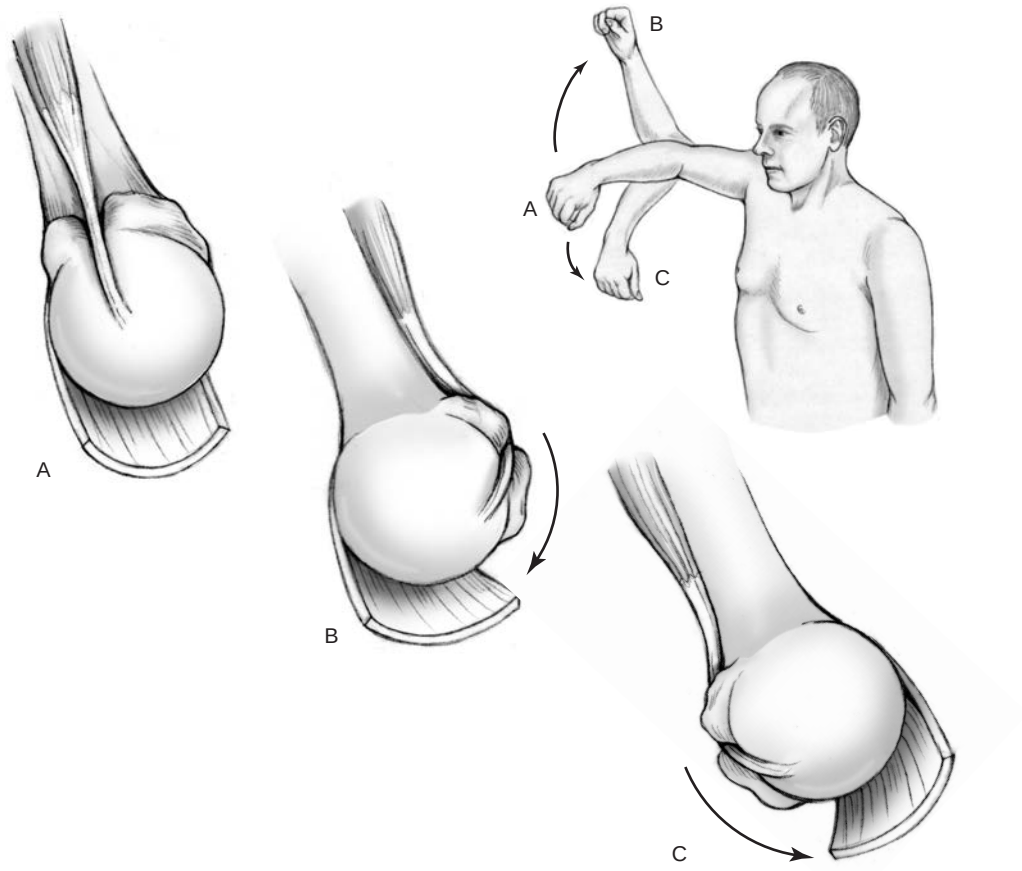

Figure 9-8 The "hammock"-like anatomy of the inferior glenohumeral ligament complex allows for reciprocal tightening of its anterior and posterior portions when the arm moves from neutral rotation in **(A)** abduction to external **(B)** and internal **(C)** rotation. (Adapted from Warner JJP, Caborn DNM. Overview of shoulder instability. *Grit Rev Phys Rehabil Med* 1992;4:145–198.)

stabilizer of the 90-degree abducted shoulder is the IGHL. The anterior band was the primary stabilizer in 30 degrees of horizontal extension and the posterior band at 30 degrees of horizontal flexion.

Injury to the IGHLC plays an integral role in the development of anterior instability. Surgical reconstruction directed at anatomic restoration of this part of the capsule has been advocated even in the earliest reports on the surgical management of shoulder instability.[12] Although all of the structures that define the shoulder capsule have at least a limited role, alterations of the IGHLC are believed by most to be a significant factor in the pathophysiology of anterior shoulder instability.

Posterior Capsule

This is the capsule extending from superior to the posterior band of the IGHLC to the intraarticular portion of the biceps tendon.[182] Other than the capsule found within the rotator interval, this is the thinnest region of the joint capsule.[49,182] There are no direct posterior ligamentous reinforcements. Its role is to limit posterior translation when the shoulder is forward-flexed, adducted, and internally rotated.[269] Clinically, this becomes relevant in patients who present with posterior instability. Unlike the other ligament structures about the glenohumeral capsule, the posterior capsule does not have a role in restraining external rotation.[5]

Material Properties

Because instability is often associated with failure of the static constraints (e.g., the capsuloligamentous structures), recent investigations have focused on the individual material properties and the modes of failure. The shoulder capsule is quite redundant, having a surface area two times that of the humeral head.[180] Material properties of the capsule refer to the intrinsic mechanical characteristics of its composition, molecular structure, and ultrastructure. Presumably anatomic variability (e.g., the SGHL and MGHL) may have clinical implications. In other words, a more robust ligament (e.g., IGHL) is presumably more tolerant of strain or force and would be expected to play a more significant role in helping to maintain glenohumeral stability.

Similar to other joint capsules in the body, the shoulder capsule is fibrous and rich in extracellular matrix. It is composed primarily of type I collagen, with lesser amounts of types II and III.[210] Debski et al.[56] quantified collagen fiber orientation in cadaveric capsule specimens and found that the collagen fibers of both the axillary pouch and the anterior band of the IGHL exhibited a random organization. Furthermore, there was no significant difference in fiber orientation seen in the bursal, middle, and articular portions of the axillary pouch. Malicky et al.[144] measured planar strains in the anteroinferior joint capsule and found considerable variability in maximum principal strains across specimens. The principal strain vectors were generally not aligned with the anterior band of the IGHL.[144] The results of these two recent studies strongly suggest that the IGHLC sustains loading in multiple directions rather than only along its length, as is the case with noncapsular ligaments.[259]

Reeves[207] determined that the average maximum tensile strength of the anteroinferior capsule measured in cadaver shoulders is 70 N (at least 20 kg), decreasing after age 50. Between the ages of 10 and 40, the anteroinferior labral insertion was the weakest portion of the whole complex, with more than two-thirds of the failures occurring there. Specimens in the fifth to seventh decades experienced capsular rupture and subscapularis tendon failure more frequently than failure at the labrum.[207] That the anteroinferior portion of the capsule fails first and capsular strength varies inversely with age has also been demonstrated by Kaltsas.[117] Hara et al.[83] evaluated the glenoid labrum and capsule and determined that the anteroinferior labrum close to the glenoid cartilage was weakest, rupturing with a mean force of 3.84 kg/5 mm.

The properties of the IGHL have been well described by Bigliani et al.,[21] who used tensile testing to analyze strength and failure modes in humerus–IGHLC–glenoid specimens. The region of the anterior band had the greatest thickness (average 2.8 mm), progressively decreasing in the axillary pouch (average 2.3 mm) and posterior capsular regions (average 1.7 mm). In contrast to O'Brien et al.,[182] no discrete posterior band was identified, and the axillary pouch was not the thickest region. Additionally, there were no significant differences in the resting length or width of these areas.[21]

Stress at failure of the anterior axillary pouch (average 5.5 MPa) was substantially lower than that described for the knee ligaments (estimated at 35 to 80 MPa198), emphasizing the importance of other stabilizing mechanisms in protecting the IGHLC from structural failure. Similar inferior strength characteristics have been described for the SGHL and CHL.[29] The superior band and anterior axillary pouch exhibit significant strain rate-dependent viscoelastic behavior. These effects were explained by compositional data determining that a proteoglycan content gradient exists, being greatest anterosuperiorly and least posteroinferiorly. Mechanically, this property leads to viscoelastic stiffening as the collagen fibers are "uncrimped" during tension.[21] This may also explain why the inferior glenohumeral ligament has the capacity to stretch considerably before ligament or insertion failure.[21] These investigators also determined that the predominant modes of failure were at the glenoid insertion, with slower strain rates as seen in the Bankart lesion, and in the midsubstance, with faster strain rates as seen with capsular laxity or stretching. This is explained by a nearly elastic behavior in the central region of the IGHL and principally viscoelastic behavior at the bony insertion.[21,244] These authors suggested

that viscoelastic behavior during tension and strain rate-dependent properties of the IGHL support its role as a humeral head stabilizer in the position of abduction and external rotation as force is rapidly applied. Subsequent tensile testing of the anterior band of the IGHLC in the apprehension position at substantially higher strain rates confirmed the viscoelastic property of increased failure stress that was previously noted.[133,155,156,158,234] Thus, functional adaptation may occur to stabilize the head during high-energy activities when other static or dynamic restraints are overwhelmed.

Morrey and Chao[163] have calculated that the anterior shear force in the position of apprehension is as high as 60 kg. To counteract these forces, contraction of the rotator cuff significantly reduces stress in the anterior capsule when the arm is in the maximally abducted and externally rotated position.[40] Thus, the dynamic restraints to stability function as a protective mechanism against structural failure of the static restraints. Although investigated to a lesser degree, the material properties of the restraints to posterior instability have been described. Weber and Caspari displaced the humeral head posteriorly in 90 degrees of flexion and full internal rotation, resulting in a horizontal split in the posterior capsule and posterior labral avulsion from the glenoid.[271] Because the results of these studies depend on a simulated mechanism of injury in the presence of an inactive rotator cuff, rigid interpretation and extrapolation to the pathoanatomy of instability is somewhat speculative.

Obligate Translations

A relatively new area of research interest is focused on understanding the relation between glenohumeral rotation and obligate translation caused by asymmetrical tightening–loosening of the capsuloligamentous structures. Tensile loading in either the anterior or superior structures is simultaneously accompanied by laxity in the posterior or inferior portion, respectively.[55,84,85,258] This is the so-called reciprocal load-sharing relationship of the capsule. Howell and Galinat[94] used axillary radiographs of patients to measure anteroposterior excursion during glenohumeral rotation. Except for maximal extension with external rotation, the humeral head remained centered on the glenoid. In normal subjects, the extended and externally rotated position caused the humeral head to translate posteriorly. In patients with anterior instability, posterior excursion did not occur. Taken a step further, Harryman et al.[85] monitored loads and translations with a magnetic-tracking device in cadaver specimens. Anterior translation occurred with flexion beyond 55 degrees, and posterior translation occurred with extension beyond 35 degrees. Interestingly, these authors found that surgical tightening of the posterior capsule resulted in increased anterior translation with flexion that occurred earlier in the arc of motion compared with normal specimens. Tightening of the rotator interval also increased obligate anterior translation with flexion. The effects of Bankart repair and overtightened inferior capsular shifts were also investigated by Janevic et al.[110] These procedures shifted the humeral head and joint contact posteriorly during loading with abduction, extension, and external rotation. The importance of these findings is that static restraints may function in positions other than the extremes of rotation. It is conceivable that unidirectional tightness, primary (e.g., overhead athlete) or iatrogenic (e.g., anterior capsulorrhaphy), could lead to instability in the opposite direction. Moreover, excessive translation in one direction may require damage to restraints on the same and opposite sides of the joint.[68] These concepts, while requiring further investigation, add an additional layer of complexity to the diagnosis and treatment of shoulder instability.

Rotator Cuff as a Static Stabilizer

Passive tension within the rotator cuff musculotendinous structures appears to have some static role in preventing glenohumeral translation. The "posterior mechanism of dislocation" occurs in older patients who sustain supraspinatus and infraspinatus tendon tears, with or without capsular injury, in association with anterior dislocation.[64,207] Rupture of the subscapularis has also been noted in patients with recurrent dislocations who are older than 35 years of age.[172] The subscapularis statically limits anterior translation in lower ranges of abduction with similar limitations to posterior translation found from the infraspinatus and teres minor.[186,187] Recently, the contribution of passive bulk tissues and the deltoid to static inferior glenohumeral stability was investigated by Motzkin et al.[165] This study determined that in both humeral adduction and abduction, passive bulk tissues (i.e., all tissues superficial to the deltoid) and the deltoid did not provide significant stability to the shoulder joint. Thus, the rotator cuff appears to be one of the few dynamic restraints that have a concomitant passive role in preventing glenohumeral instability.

Dynamic Factors

Clinical experience suggests that static stabilizers by themselves may not be as important in enhancing glenohumeral stability as that provided by the dynamic stabilizers or the relation between them. Experimentally, specimens dissected free of the rotator cuff and long head of the biceps tend to demonstrate at least some degree of inferior subluxation.[127,186] Active contraction of these structures contributes to the dynamic stabilization of the glenohumeral joint through two mechanisms: (a) joint compression (e.g., concavity–compression) resulting from synergistic and coordinated rotator cuff activity and (b) ligament dynamization through direct attachments to the rotator

cuff muscles. Augmenting these mechanisms are the long head of the biceps brachii, coordinated scapulothoracic rhythm, and proprioception providing feedback about extremity position and movement.

Joint Compression

Contraction of the rotator cuff and long head of the biceps brachii augments joint stability by enhancing the conforming fit and increasing the load needed to translate the humeral head through compression of the humeral head into the glenoid.[40,140,147] The rotator cuff muscle forces are ideally aligned for effective compression of the glenohumeral joint at all shoulder positions.[132] Lippitt et al.[140] quantified the magnitude of the tangential forces required to produce glenohumeral dislocation in the setting of applied joint-compressive loads of 50 and 100 N. Tangential forces were as high as 60% of the applied joint-compressive load. The stability of the joint was markedly reduced if a portion of the labrum was removed. Vahey et al.[249] introduced the concept of "scapulohumeral balance" to illustrate that glenoid geometry coupled with joint compression is a major stabilizing force. Bowen et al.[35] determined that a joint compression load of 111 N was sufficient to stabilize the glenohumeral joint in the face of a 50-N force, despite sectioning of three-fourths of the joint capsule. It has been suggested from the results of ligament-cutting studies and direct quantification of the efficiencies of the dynamic stabilizers that joint compression is a more important stabilizer to translation than are static capsular constraints.[27,35]

Poppen and Walker[200] showed that the joint reaction force was a maximum of 0.89 times body weight directed into the face of the glenoid at 90 degrees of abduction using a simplified two-dimensional cadaveric model with digitized radiographs. Also, the subscapularis had a greater mechanical advantage at lower abduction angles (i.e., 60 degrees), whereas the deltoid had a greater advantage at higher abduction positions. McKernan et al.[151] have validated these findings and attributed them to the anterior location of the subscapularis tendon in lower ranges of elevation, making it a more effective stabilizer against a given translation. This effect is reduced as the shoulder is elevated and the line action of the subscapularis moves superior to the joint.

All portions of the rotator cuff are probably important in enhancing stability, as was shown by Blasier et al.[26] In their biomechanical study, omission of tension in any one of the rotator cuff muscles led to a substantial reduction in anterior joint stability. Labriola et al. reported that all rotator cuff muscles contribute equally to anterior stability when the glenohumeral joint is in the anatomic position; at end range, the subscapularis is less important.[107,128] This is supported by an investigation by Wuelker et al., which found that a 50% decrease in the rotator cuff muscle forces resulted in nearly a 50% increase in anterior displacement of the humeral head in response to external loading at all glenohumeral joint positions.[278]

Rotator cuff tears result from either single traumatic or cumulative microtraumatic (i.e., overuse injuries) events. Because of age-related attrition, a dislocation in individuals older than the age of 40 is not uncommonly associated with a rotator cuff tear.[117,172,207] Rotator cuff tears result in superior translation of the humeral head during scapular plane abduction, and larger rotator cuff tears lead to increased displacement of the humeral head.[98] This demonstrates the importance of synchronous contraction of the entire cuff in maintaining containment of the humeral head in the glenoid.[200,267]

Increasing the joint compressive load appears to "center" the humeral head, reducing subsequent translation. This centering of the humeral head in the glenoid socket provides a stable fulcrum for elevation of the humerus.[67,97,240] Interestingly, the ability of isometric muscle contraction to "center" the humeral head is different in patients with traumatic instability compared to those with atraumatic instability. A recent evaluation of glenohumeral kinematics using magnetic resonance imaging revealed that such contraction led to recentering of the humeral head only in the patients with traumatic instability.[253]

In overhead athletes, for example, in whom the rotator cuff functions as an important decelerator to anterior translation, imbalanced muscle recruitment may play a role in those with more subtle forms of instability.[78,113] This has been validated by Warner et al.,[266] who demonstrated that patients with shoulder instability had altered rotator cuff strength patterns compared with normal controls. Asynchronous contraction of the rotator cuff, leading to voluntary instability, is an example in the extreme of the relative importance of the rotator cuff in enhancing dynamic stability of the glenohumeral joint.[214] Conversely, capsuloligamentous insufficiency could subject the rotator cuff to overuse, fatigue, and injury.

The importance of these findings is that rotator cuff–strengthening programs can improve the function of a weak or ineffective cuff by limiting translation of the humeral head on the glenoid during active shoulder motion.[39] Initial therapeutic approaches to shoulder instability, therefore, should emphasize strengthening, conditioning, and coordination of the rotator cuff as an integral part of the treatment program.

Ligament Dynamization

There appear to be direct connections between the rotator cuff tendons and the capsuloligamentous system.[45,64] Clark et al.[45] reported a complex anatomic relation between the tendons of the rotator cuff and the capsule adjacent to the humeral tuberosities. The joint capsule is adherent to the rotator cuff, except anterosuperiorly in the

rotator interval, found between the free margins of the supraspinatus tendon superiorly and subscapularis tendon inferiorly. Conceptually, active shoulder motion may "dynamize" the capsule and ligaments, thereby becoming a significant stabilizing factor in the midranges of rotation at which the ligaments and capsule are relatively lax. Warner et al.,[258] in a dynamic shoulder model, were able to define and document the orientation and interrelation between the glenohumeral ligaments during simulated rotator cuff contraction. Although this study clearly elucidated the effect of shoulder rotation on the orientation of the undisturbed and intact capsuloligamentous system, the dynamic effects of rotator cuff contraction upon the ligaments remains unclear.

Pagnani et al.[189] suggested that because the biceps inserts into the relatively mobile superior labrum, it is conceivable that tension would be transmitted by the labrum to the SGHL and MGHL to dynamize these static structures and indirectly enhance stability. A similar relation may exist owing to the proximity of the triceps to the medial aspect of the axillary pouch of the IGHLC.[45,51] Anterosuperiorly, the subscapularis and supraspinatus interconnect with the CHL, providing an additional site for dynamic interaction between static and dynamic restraints.

Active rotation may also have the effect of altering capsular tension, potentially providing a protective mechanism against failure. For example, coupled posterior humeral head translation with active external rotation may actually reduce anterior ligamentous strain.[85,94] McKernan et al.[152] and others[40,211] have shown in cadaveric experiments that contraction of the posterior rotator cuff muscles (i.e., infraspinatus and teres minor) and biceps tendon reduced IGHL strain in the late cocking phase of throwing. These dynamic factors may provide relative protection of the IGHLC or other anterior structures as they contribute to anterior stability by dynamically increasing the resistance to torsional forces in the position of apprehension. Recently, however, the role of the infraspinatus, as determined by electromyographic (EMG) analysis in patients with recurrent anterior instability, was not believed to be a critical component in providing anterior stability.[96] Clinically, that a stabilizing relation may exist between the capsule and musculature about the shoulder signifies the importance in reestablishing length–tension relations by either operative or nonoperative means in patients with shoulder instability.

Long Head of the Biceps Brachii

As the tendon of the long head of the biceps passes to its insertion in the supraglenoid tubercle, it occupies an intraarticular position. The relative importance as a dynamic stabilizer probably becomes significant when the rotator cuff or capsuloligamentous structures are overwhelmed. Several experimental studies have demonstrated the dynamic-stabilizing role of the long head of the biceps brachii for the glenohumeral joint.[3,78,103,106-108,121,162,189,203,211] Rodosky et al.[211] showed that, in the late cocking phase of throwing, contraction of the biceps tendon can significantly reduce anterior translation and increase torsional rigidity of the joint helping to resist external rotation. Additionally, strain in the IGHL was noted to increase after sectioning of the tendon.

Pagnani et al.[189] determined that the effect of the long head of the biceps is dependent on the shoulder position being greatest in middle and lower elevation angles. The biceps tended to stabilize the joint anteriorly when the arm was internally rotated and served as a posterior stabilizer when the humerus was externally rotated (Fig. 9-9). Itoi et al.[107] found that anteroposterior translation was significantly decreased with biceps loading, particularly with external rotation. Superoinferior translation was also reduced with simulated contraction of the biceps, which was believed to help center the humeral head on the glenoid, thereby stabilizing the fulcrum and allowing more efficient arm elevation. Levy et al.[137] emphasized through dynamic EMG analysis that elimination of elbow flexion or supination resulted in complete inactivity of the biceps brachii. Thus, the role of biceps function at the shoulder is either due to a passive mechanism or depends on tension developing in association with elbow and forearm activity.

Kim et al.[121] conducted a thorough EMG analysis of the biceps brachii muscle in patients with anterior instability. The voltage of the biceps muscle was significantly greater in the unstable shoulder compared to the opposite arm in all positions of the arm. Moreover, activity increased in abduction and external rotation of the unstable shoulder; there was no change in activity in the stable shoulder placed in this position. These findings imply a secondary stabilizing function of the biceps muscle, which compensates for failed primary static restraints.[121]

These concepts may help explain why the biceps tendon or superior labrum may demonstrate lesions in throwers[3,228] and why it is occasionally found to be hypertrophied in the rotator cuff–deficient patient.[146,238] Extreme external rotation loads the long head of the biceps tendon, which predisposes the throwing athlete to biceps or biceps–labrum complex injuries.[126,162,203] Clinically, this suggests that nonoperative treatment of instability or rotator cuff deficiency should be directed at rehabilitation of the biceps brachii in addition to the rotator cuff muscles.

Scapular Rotators

Until recently, scapulothoracic motion has been relatively ignored as an important dynamic factor maintaining stability of the glenohumeral joint. The scapular rotators include the following muscles: trapezius, rhomboids, latissimus dorsi, serratus anterior, and levator scapulae. Codman[47] first introduced the concept of "scapulohumeral

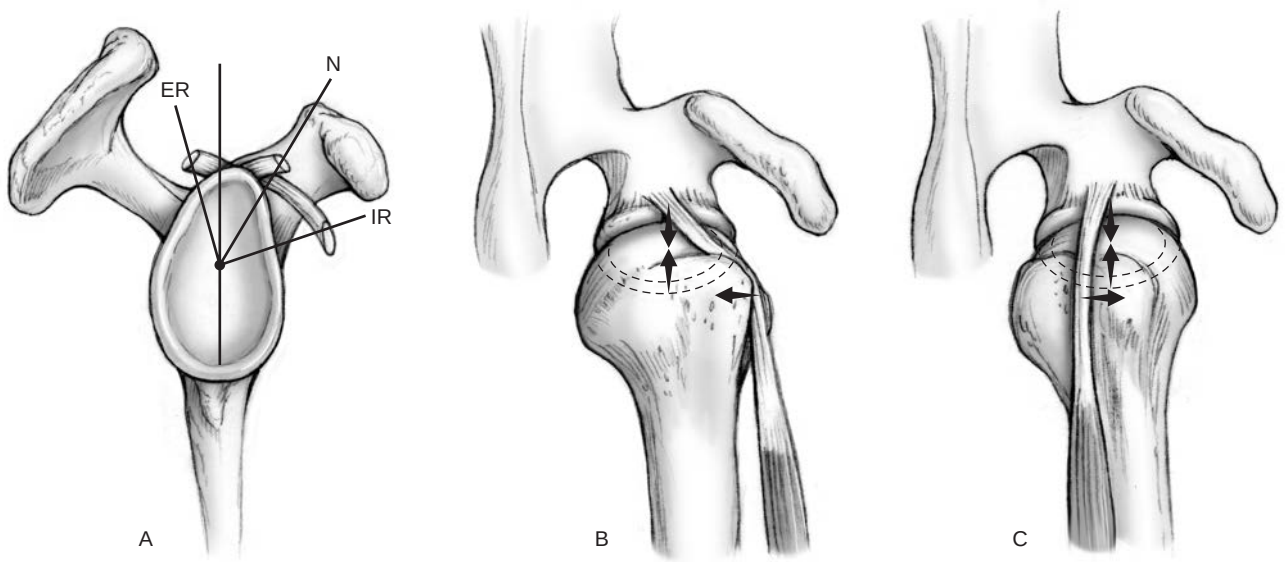

Figure 9-9 Diagrammatic representation of forces created with simulated contraction of long head biceps brachii. **(A)** Rotation of humerus changes orientation of biceps tendon relative to the joint. In neutral rotation (*N*) tendon generally occupies a slightly anterior position. With internal rotation (*IR*) the tendon lies anterior to joint. In contrast, the tendon occupies a slightly posterior position with external rotation (*ER*). **(B)** With internal rotation of humerus, the biceps appears to generate joint compressive forces (*paired arrows*) and posteriorly directed force (*single arrow*), which restrain glenohumeral translation. **(C)** With external rotation of the humerus, anteriorly directed force (*single arrow*) appears to accompany joint compressive forces (*paired arrows*). (Adapted from Pagnani M, Deng X-H, Warren R, Torzilli P, O'Brien S. Role of the long head of the biceps brachii in glenohumeral stability: a biomechanical study in cadavera. *J Shoulder Elbow Surg* 1996;5:255–262.)

rhythm," which has now been recognized by others to be an important contributor to joint stability.[14,105,218,265] Even though somewhat variable, the normal scapulohumeral rhythm motion relation is two of glenohumeral rotation for every one of scapulothoracic rotation during scapular plane abduction.[200,201] Clinical and radiographic studies have documented abnormal scapulothoracic motion in patients with shoulder instability.[188,265] EMG analysis of the scapulothoracic musculature has demonstrated fatigue of the serratus anterior and trapezius with repetitive overhead activities, leading to poor scapulothoracic control.[78,178] McMahon et al. demonstrated that patients with glenohumeral instability have decreased serratus anterior activity during abduction, scaption, and forward flexion.[157]

The scapular rotators function to provide a stable platform beneath the humeral head during shoulder motion (Fig. 9-10). These muscles allow the glenoid to adjust to changes in arm position. The scapular inclination angle is a significant factor preventing inferior translation of the adducted shoulder.[105] For example, the scapula normally rotates upward (i.e., protraction) in synchrony with arm elevation as the serratus anterior contracts. Thus, clinically, scapulothoracic weakness or dysfunction is associated with varying degrees of scapular winging, which is often found in patients with shoulder instability.[265] Warner et al.[265] have hypothesized that scapulothoracic dysfunction may be a cause of "nonoutlet" impingement, as the advancing greater tuberosity is unable to avoid impingement on the coracoacromial arch during forward flexion. Presently, however, it is unclear if scapulothoracic dysfunction is a cause or product of shoulder instability. Despite these unanswered questions, nonsurgical management of shoulder instability must include rehabilitation of the scapular rotators.

The roles of the deltoid and pectoralis major muscles about the glenohumeral joint have been evaluated in the last several years. Kido et al.[120] evaluated the stabilizing function of the anterior, middle, and posterior deltoid muscle in normal shoulders and in shoulders with instability. In normal shoulders, tension on the middle deltoid reduced the amount of anterior translation substantially. When the joint capsule was vented, or when there was a simulated Bankart lesion, loading each of the three segments of the deltoid muscle decreased anterior displacement. The authors concluded that the deltoid muscle is an anterior stabilizer of the glenohumeral joint with the arm in the position of apprehension, and that this function may become more important in the unstable shoulder.[120]

Lee and An[131] similarly found that deltoid muscle activity increases glenohumeral joint stability. However, this

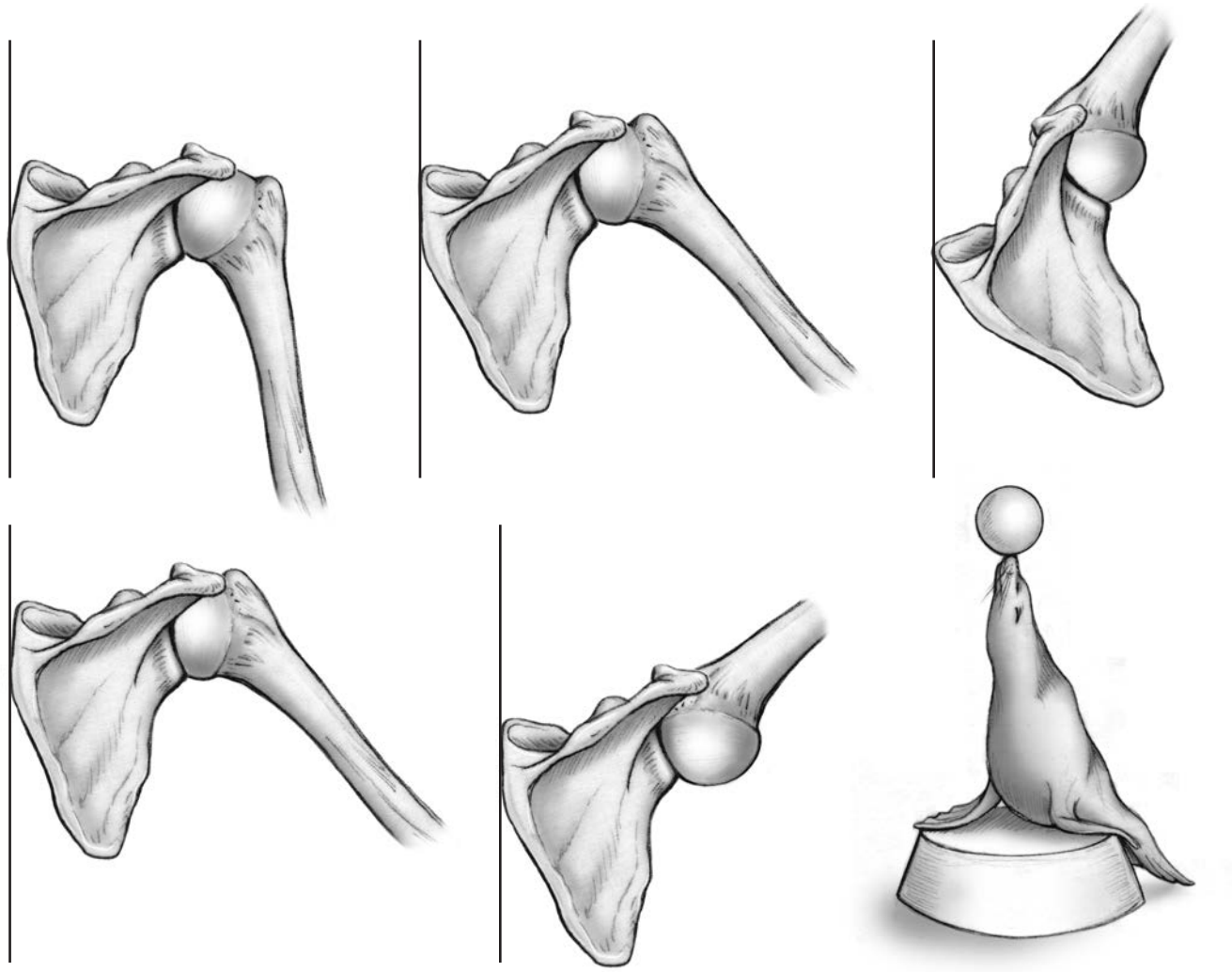

Figure 9-10 **(Top row)** Normal scapulothoracic rotation positions the glenoid underneath the humeral head so that it acts as a stable platform. **(Bottom row)** Failure of proper scapulothoracic motion results in loss of the stable glenoid platform underneath the humeral head. This is analogous to a seal balancing a ball on its nose. (Adapted from Warner JJP, Caborn DNM. Overview of shoulder instability. *Crit Rev Phys Rehabil Med* 1992;4:145–198.)

effect was most pronounced at 60 degrees of glenohumeral abduction in the scapular plane. Conversely, deltoid activity decreased glenohumeral stability when the glenohumeral joint was abducted to 60 degrees in the coronal plane. The role of the pectoralis muscle on glenohumeral joint stability is less clear, though Arciero and Cruser[7] reported a case of traumatic glenohumeral dislocation and pectoralis tendon rupture while bench pressing. They theorized that eccentric loading of the pectoralis led to both injuries. Sinha et al.[227] reported an irreducible glenohumeral dislocation that was successfully reduced only after paralysis of the pectoralis major with botulinum A toxin. Management of glenohumeral joint stability may be altered to consider these muscles as their role about the glenohumeral joint becomes clearer.

Proprioception

The perception of joint position and joint motion is termed *proprioception*. Proprioceptive interaction between ligaments and muscles may mediate a protective mechanism against capsular failure and instability.[25,42,123,135] Murakami et al.[167] described what were thought to be mechanoreceptors in the transition zone between the labrum and capsule in primates. Since then, others[112,251,252] have described similar findings in the capsule and ligaments of the glenohumeral joint.

Mechanoreceptors are specialized nerve endings (e.g., pacinian corpuscles, Ruffini endings, and Golgi tendon-like endings) that transduce mechanical deformation into electric signals that transmit information about joint

position and motion.[79,80] Vangsness et al.[252] evaluated the capsuloligamentous structures for the presence of these mechanoreceptors: Low-threshold, slow-adapting Ruffini afferents were most abundant overall, except in the glenohumeral ligaments where low-threshold, rapid-adapting pacinian-type afferents were more numerous. No mechanoreceptors were observed in the subacromial bursa or glenoid labrum.

Lephart et al.[135] and Warner et al.[264] have hypothesized that the capsuloligamentous structures may contribute to stability by providing an afferent feedback for reflex muscular contraction of the rotator cuff and biceps. It is plausible, as these authors discuss, that as these receptors respond to tension changes in the capsule during rotation, active stabilization may occur through reflex arcs from the capsule to the surrounding rotator cuff, allowing selective contraction of the rotator cuff and biceps muscles in response to changes in acceleration.

Several studies have found decreased proprioception in shoulders with instability.[13,25,135,136,168,243] Capsuloligamentous disruption combined with proprioceptive deficits contribute to functional instability.[136,243] Barden et al.[13] measured hand position error in patients with multidirectional instability (MDI) and compared this to patients without instability. The subjects with MDI had significantly greater hand position error than the control group. Interestingly, there was no difference between hand position error in the symptomatic arm and the contralateral arm in patients with MDI. The authors inferred that patients' capacity to use proprioception to refine upper-extremity movement is reduced in MDI.[13] Blasier et al.[25] reported similar findings.

With use of a specialized proprioception testing device, Lephart et al.[135] evaluated subjects with and without traumatic anterior instability preoperatively and postoperatively after arthroscopic or open Bankart repairs. In normal shoulders, the threshold to detect passive motion (TTDPM) averaged 1.5 to 2.2. In those with instability, TTDPM was 2.8. These differences were statistically significant. Postoperatively, patients' TTDPM was no different from normal. One criticism of this study was that the speeds at which patients were tested were much slower than the speeds that occur with overhead sports. Ito et al.[109] has stated that traumatic dislocation may, in fact, occur sooner than the response time of the rotator cuff stretch reflex. Thus, the relative importance of this proposed mechanism may be more significant in lower-energy situations when the rotator cuff or biceps has time to react to relative changes in capsular tension. Furthermore, it is postulated that proprioception may be a way to protect the capsuloligamentous structures from failure owing to repetitive microtrauma, leading to excessive translation or instability.[135,264]

Whether inherent deficits in proprioception predispose a patient to glenohumeral instability or instability reduces proprioceptive capacity remains unclear. It is interesting to find that surgical procedures that retension the capsuloligamentous structures improve glenohumeral joint proprioception.[135,202,243] One long-term follow-up study reported improvements in joint position sense for at least 5 years postoperatively; position sense may be comparable to normal, healthy shoulders.[202]

PATHOANATOMY OF SHOULDER INSTABILITY

In addition to the "essential lesion" (i.e., labral detachment), recurrent instability has been attributed to several pathologic entities. As indicated in Table 9-2, each of the factors already discussed plays a role in the pathogenesis of shoulder instability. Several authors have cited attenuation of the capsule and capsular ligaments[21,159,164,199,245,246] with associated histopathologic changes.[135,150,198,210,252] Impression fracture of the humeral head (Hill-Sachs or reverse Hill-Sachs lesion),[50,93,153] attenuation of the subscapularis tendon,[59,71,236] capsular rupture,[115,206,208] and humeral avulsion of the glenohumeral ligaments are other documented causes of anterior instability.[10,31,223,235,237,268,276] A thorough history is important to ascertain the mechanism of injury. A single violent trauma will focus the injury on a specific anatomic region (e.g., Bankart lesion or capsular damage). On the other hand, repetitive microtrauma may cause more subtle capsular stretch, emphasizing the pathoanatomic continuum of capsular injury. Clinically, appreciation of the pathoanatomy of shoulder instability is important because surgical intervention is ultimately directed at anatomic and biomechanic restoration.

Bankart Lesion

The most common form of shoulder instability is recurrent anterior subluxation or dislocation resulting from trauma. Perthes[195] and Bankart[12] (e.g., Perthes-Bankart lesion) originally described the detachment of the capsulolabral complex from the glenoid rim and scapular neck as the "essential lesion" leading to recurrent anterior dislocation. This has been challenged by Speer et al.,[232] who found that simulation of the Bankart lesion in cadaveric cutting studies resulted in only minimal increases in anterior translation. Baker et al.[11] established a classification system based on arthroscopic findings of initial anterior shoulder dislocations. Sixty-two percent had evidence of a Bankart or equivalent lesion, with all of these patients demonstrating gross instability with examination under anesthesia. Thirteen percent were stable on examination and demonstrated no evidence of labral detachment. Taylor and Arciero[237] studied first-time patients with traumatic anterior shoulder dislocations and determined arthroscopically that 97% had evidence of isolated detachment of the capsuloligamentous complex from the glenoid rim and neck, without evidence

of intracapsular injury. Others have noted a similarly high incidence of Bankart lesions at the time of surgery.[239]

Despite experimental and clinical evidence of increased anterior translation of the humeral head on the glenoid caused by a Bankart lesion, most patients present with recurrent anterior instability with additional pathology that may have developed or advanced over time. Additionally, plastic deformation or capsular injury in patients with first-time dislocations may not be appreciated by macroscopic evaluation because it may represent microscopic ultrastructural failure not visible to the naked eye. It is now believed that recurrent complete dislocation requires an additional pathoanatomic component (e.g., capsular plastic deformation or stretch).[12,21,213,232]

Recognition of this concept is clinically relevant. Isolated arthroscopic Bankart repair is technically challenging and with earlier reports associated with higher failure rates, possibly because of associated pathology, poor patient selection, and minimal scar formation.[6,53,129,160,233,254] Moreover, reduction in anterior translation, which is the goal of the procedure, is only significant with larger imbrication (5 mm) of the capsule. Larger imbrication leads to a more severe limitation of external rotation.[177] Conversely, open Bankart procedures that address "only" the labral detachment may create enough capsular scarring to prevent recurrence. Thus, the choice of operative procedure will depend on the patient's history, examination under anesthesia, arthroscopic anatomy, and appreciation for capsular injury. These tenants become more complex in the patient with multidirectional instability in whom capsular laxity is the dominant pathology, and Bankart lesions are less frequently found.

Capsular Injury

Traumatic Intrasubstance Injury

Clinical observations indicate that capsular injury is commonly associated with traumatic anterior shoulder dislocation. Capsular injury resulting from traumatic anterior shoulder dislocation was recognized as early as the 13th century. Reeves demonstrated capsular rupture by arthrography in 55% of the anterior dislocations he treated.[206,208] Symeonides[236] observed that 15% of his patients treated for anterior dislocation had both labral detachment and anterior capsular ruptures. Johnson[115] observed that 54% of his patients at the time of arthroscopy for anterior dislocation had torn glenohumeral ligaments. Conversely, others have shown that only minimal irrecoverable elongation of the inferior glenohumeral ligament occurs after traumatic unidirectional dislocations.[155,156,158,234]

Experimentally, Bigliani et al.[21] measured the stress–strain data at failure of the IGHLC in bone–IGHL–bone preparations and concluded that before failure, significant plastic deformation (e.g., strain) occurred. The implications of these findings are that laxity of the IGHLC leading to instability is not only a congenital finding, but that it may be acquired through submaximal trauma (single or repetitive) without causing rupture or detachment. When the anterior shear force overcomes the capsular tensile strength or when the rotator cuff fatigues or cannot effectively contract (e.g., rotator cuff tears), the ligaments may fail on an ultrastructural level.[135,198,252] Rodeo et al.[210] provided evidence of ultrastructural changes in the joint capsules of unstable shoulders. In joint capsules from patients with instability, there are increases in the amount of stable and reducible cross-links (the latter of which is abundant in remodeling tissue) as well as the mean collagen fibril diameter (which correlates positively with tissue strength) compared to patients with stable shoulders.[210] Others have shown histologic changes in shoulder capsules of patients with traumatic instability, such as a denuded synovial layer, subsynovial edema, increased cellularity, and increased vascularity.[150]

Similarly, age-related attrition of the rotator cuff tissues is greater than in capsular tissues such that anterior dislocation commonly results in a rotator cuff tear, potentially leading to capsular injury in older patients.[207] Gamulin et al.[71] evaluated the histomorphometry of the subscapularis muscle in 52 patients operated on for recurrent traumatic anterior shoulder dislocation. They observed interstitial fibrosis within the subscapularis muscle and modifications in the ratio of fiber types that are characteristic of disuse atrophy. Clinically, capsular injury leading to laxity and labral detachment can be found to coexist, supporting concomitant capsulorrhaphy and Bankart repair in the surgical management of instability. Bigliani et al.[19] and Altchek and Dines[2] have advocated addressing capsular pathology as part of the surgical treatment of recurrent anterior shoulder instability.

Humeral Avulsion

First described in 1942 by Nicola,[173] avulsion of the capsule from the humerus can occur with forceful hyperabduction. Disruption of the lateral capsule from the humeral neck is probably rare, but has been reported in two cases by Bach et al.[10] and in one case by Taylor and Arciero[237] in association with anterior dislocation. Wolf et al.[276] has termed this a "HAGL lesion" representing humeral avulsion of the glenohumeral ligament. Appreciation for this variant of capsular injury at the time of arthroscopy can be difficult, though these authors advocate searching for it in patients with traumatic anterior instability who show no signs of a Bankart lesion.[276]

The HAGL lesion appears as a thickened, rolled edge of capsular defect, typically found in the inferior pouch of the shoulder below the level of the subscapularis muscle.[31] Associated glenohumeral abnormalities are common, most often in the form of rotator cuff tears; greater than 90% of these tears involve the subscapularis muscle.[10,31,223,268,276] Only 20% of HAGL lesions may be viewed radiographically.[10]

If it is visualized it likely represents a bony HAGL (BHAGL) lesion, which may mimic a bony Bankart lesion.[179] The BHAGL lesion, first described by Bach et al.,[10] is a HAGL lesion associated with bony avulsion of the humeral neck. Oberlander et al.[179] recommended evaluating such lesions with axillary or West Point views of the glenohumeral joint. In either of these views the BHAGL is seen superimposed on the proximal humerus, whereas the bony Bankart lesion is seen along the inferior half of the glenoid cavity. Stoller[235] described the J sign of the HAGL lesion on magnetic resonance arthrography, whereby the axillary pouch changes from a fluid-distended U-shaped structure to a J-shaped structure. This is attributed to inferior displacement of the anterior band of the inferior glenohumeral ligament.[235] Humeral avulsion of the glenohumeral ligament should be repaired anatomically at the time of surgical reconstruction.

Repetitive Injury

The overhead athlete (e.g., pitchers, throwers, swimmers, volleyball players, tennis players, water polo players, and javelin throwers) represents a special category of patients with complaints relating to instability. These patients subject their shoulder to repetitive stresses that potentially lead to microtrauma not readily appreciated at the time of arthroscopy. In a novel study, Pollock et al.[199] evaluated the response of the IGHL to a range of cyclic deformations and different levels of strain in an attempt to identify mechanical microdamage caused by repetitive loading. They found a significant decrease in the residual strain magnitude of the IGHL after cyclic loading compared to baseline. It was concluded that the cumulative effect of repetitive subfailure strain causes irreversible stretching of the IGHL. This may contribute to the development of shoulder instability.[199] Malicky et al.[142] also identified irreversible changes in strain magnitude of the anteroinferior capsule after 16 mm of humeral translation.

Repetitive rotational motion of the glenohumeral joint may also contribute to instability. Remia et al.[209] reported on an experimental model of multidirectional instability of the glenohumeral joint and found that application of internal and external rotational stretches to the capsule causes increased translation in all directions, without capsular disruption. Likewise, Mihata et al.[159] found increased shoulder laxity after nondestructive stretching of 30% beyond maximal humeral external rotation. This was attributed to a significant lengthening of the anterior band of the IGHL. Repetitive injury may be the cause of acquired laxity, as seen in gymnasts, and may present as multidirectional instability rather than pure unidirectional instability. These patients are often confused as having isolated subacromial impingement and inappropriately treated as such.[69,241] It is now believed that "subtle glenohumeral instability" may be associated with secondary subacromial impingement, and capsular laxity is the primary pathology that should be addressed.[242]

Capsular Laxity

Intrinsic Capsular Laxity

Capsular laxity is a prerequisite to allow a large range of glenohumeral motion. The degree of laxity varies among individuals, and attempts at correlating the extent and direction of laxity under anesthesia can be confusing, for the overlap between normal laxity and clinical instability is difficult to ascertain.[48,62,73,85,86,184,248,266] Interestingly, shoulders of asymptomatic patients can exhibit a range of rotational or translational motion comparable to that seen in patients diagnosed with symptomatic instability.[85,89,89] In the preadolescent shoulder joint, more than two-thirds may be asymptomatically subluxated on examination.[62] That asymptomatic subluxation or even dislocation may occur in the "normal" shoulder at the time of anesthesia was also appreciated by O'Driscoll and Evans[184] and Warner et al.[262] Thus, it is unclear if constitutional laxity is a risk factor for clinical instability of the shoulder joint. With a proper history of the mechanism and symptoms, correlation with drawer testing under anesthesia can be useful if one considers the effect of arm position on different portions of the capsule.

Inherited Disorders of Collagen

Inherited disorders of collagen are relatively rare, but present an unusual challenge in the management of glenohumeral instability. The collagen disorder most associated with shoulder instability is Ehlers-Danlos syndrome (EDS), which is characterized by increased laxity, problems with wound healing, and vascular anomalies. Several subtypes of EDS have been identified based on which type and which synthetic step of collagen is aberrant. EDS I is inherited in an autosomal dominant pattern and is most commonly associated with hyperlaxity. EDS II is a milder form of EDS I. The other subtypes of EDS predominantly affect the blood vessels. A survey of 42 patients with EDS revealed that they had experienced a combined 214 shoulder procedures. The indications were pain, instability, poor range of motion, or a combination of these.[89] Another case report describes a 19-year-old female with EDS and bilateral multidirectional shoulder instability who underwent multiple operations to achieve stability, all of which were ineffective.[149] Thus, shoulder instability with EDS remains a difficult area to manage even with contemporary techniques.

Humeral and Glenoid Bone Loss

Humeral Bone Loss

Articular abnormalities of the humeral head can disrupt the anatomic relation of the glenohumeral joint, predisposing to recurrent instability. In an evaluation of radiographs from 160 patients with chronic anterior shoulder instability, Edwards et al.[61] identified humeral impaction

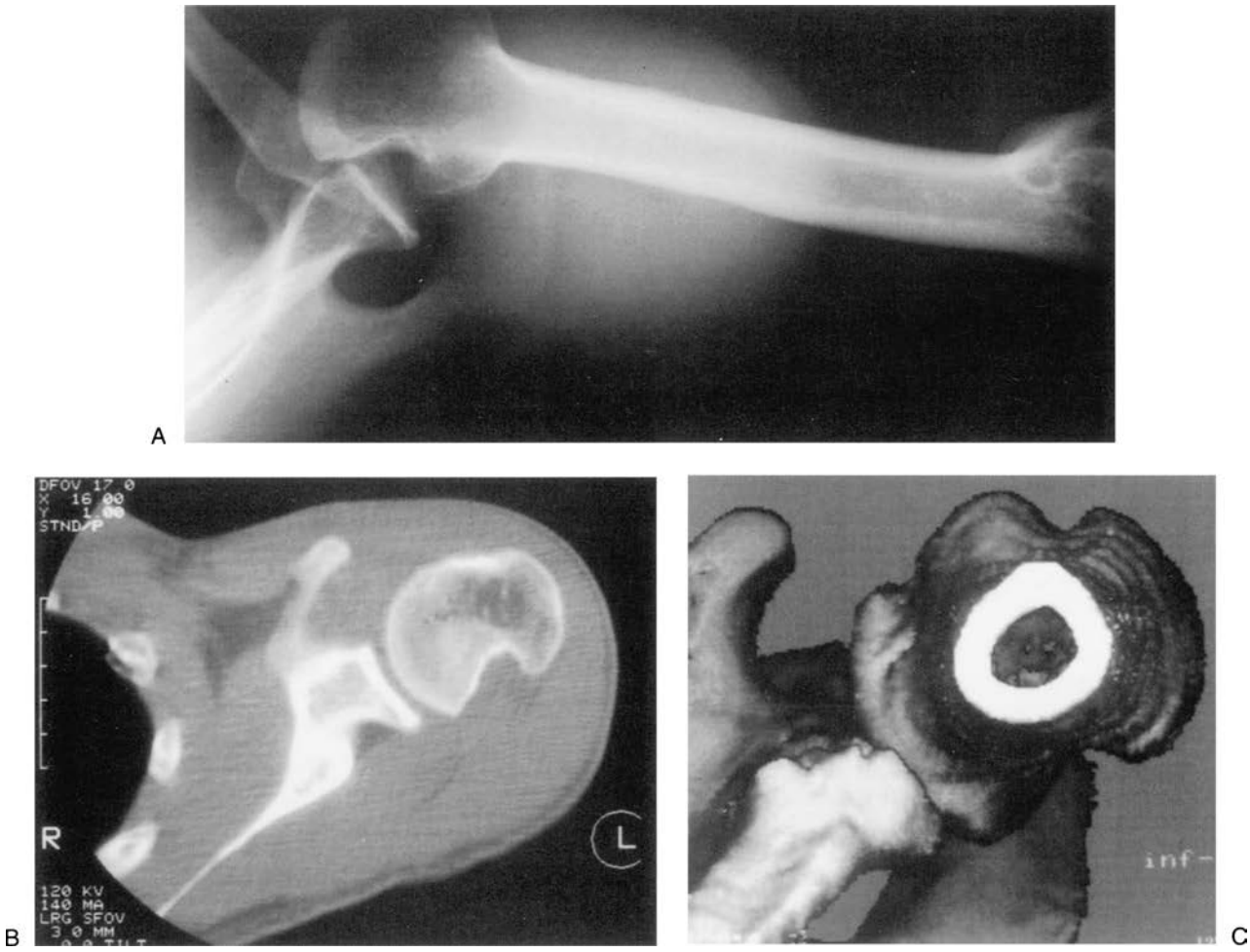

Figure 9-11 **(A)** Anterior dislocation in a patient with a large Hill-Sachs lesion (more than 30% of the humeral head). **(B)** Computed tomographic (CT) scan in the same patient. **(C)** A three-dimensional CT reconstruction also shows a large Bankart lesion in addition to the large Hill-Sachs lesion.

fracture in 117 shoulders (73%). A large Hill-Sachs or reverse Hill-Sachs impression fracture (Fig. 9-11) on the posterolateral or anterolateral margin of the humeral head, respectively, is created when the humeral head dislocates over the anterior or posterior glenoid rim.[41,194,213,216] This lesion is present in more than 80% of anterior dislocations and 25% of anterior subluxations.[41,194] Hill-Sachs lesions have been noted at the time of arthroscopy in patients with recurrent anterior instability up to 100% of the time.[41,176,237] Werner et al.[273] reported a prevalence of Hill-Sachs lesions approaching 60% in patients with atraumatic instability that did not respond to conservative therapy. The small Hill-Sachs lesions are not usually thought to be a major contributor to recurrent anterior instability.[41] The incidence with posterior instability is unknown.

The Hill-Sachs lesion is larger with dislocations of longer duration, recurrent dislocations, and inferior displacement of the humeral head.[66,92] In most instances this lesion is relatively small and plays little role in ongoing shoulder instability or its surgical management. Relatively small lesions may be prevented from coming into contact with the anterior glenoid rim simply by performing a more generous anterior capsulorrhaphy (Fig. 9-12). Caution is warranted with this practice because excessively tight anterior repairs may be associated with the development of late arthrosis.[18,22] However, when the Hill-Sachs lesion involves more than 30% of the humeral articular surface, it may contribute to recurrent anterior instability, even with capsular repair.[215,216]

The mechanism for this persistent instability is that with increasing external rotation, the lesion slips over the anterior glenoid (i.e., as in the original injury) and sits in an anteromedial position outside of the glenoid cavity. Surgical treatment of these defects involves filling the defect with allograft bone (Fig. 9-13),[74,114] muscle tendon transfer,[50,66,153] (e.g., infraspinatus or subscapularis), or humeral head replacement in older individuals. Alternatively, the lesion can be rotated out of contact with the glenoid with proximal humeral osteotomy.[270] Decision making is predicated on the ability to perform an anatomic

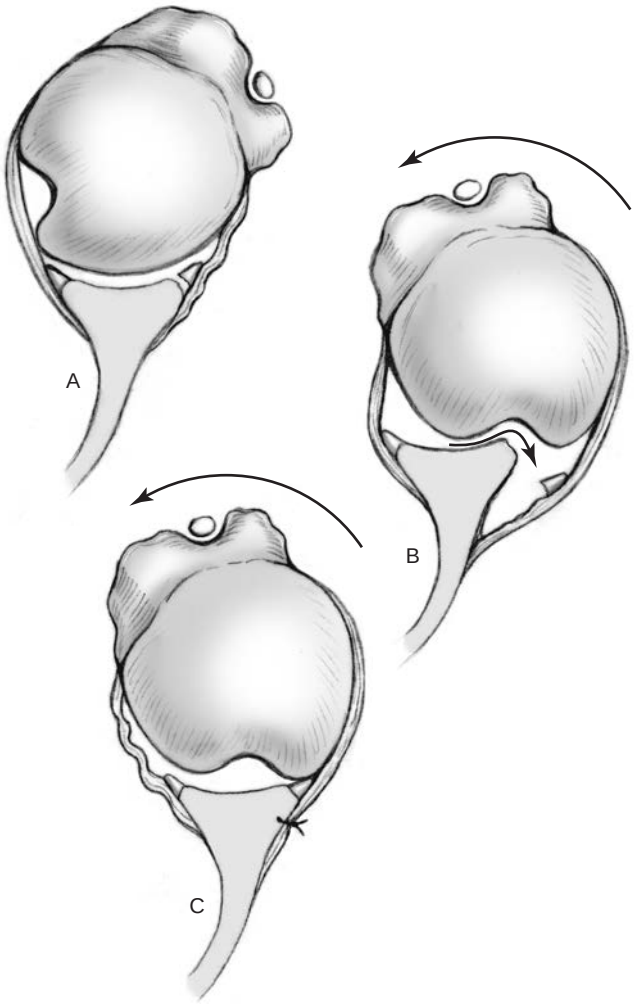

Figure 9-12 Role of the Hill-Sachs lesion in anterior shoulder instability. **(A)** With the arm in internal rotation, the Hill-Sachs lesion is not in contact with the glenoid. **(B)** With external rotation, the humeral head translates anteriorly because of the incompetent anterior capsular mechanism. This allows the humeral head to dislocate through the Hill-Sachs lesion. **(C)** An adequate Bankart repair keeps the Hill-Sachs lesion contained on the glenoid, unless it is greater than 30% of the humeral articular surface. (Adapted from Warner JJP, Schulte KR, Imhoff AB. Current concepts in shoulder instability. In: Stauffer RN, Erlich MG, Kostuik JP, Fu FF. *Advances in operative orthopaedics*, vol. 3. Philadelphia: Mosby-Year Book, 1995;217–247.)

reconstruction of the anteroinferior structures with an appropriate capsular plication followed by reassessment for the ability to engage the Hill-Sachs lesion in various positions of rotation with a simultaneously applied anterior force to the proximal humerus.

Glenoid Bone Loss

Glenoid bone loss has been implicated as a predisposing factor for recurrent instability after surgical repair.[24,104,247] Burkhart et al.[38] observed that glenoids with enough bone loss to convert the normally pear-shaped glenoid to an inverted pear configuration are at particular risk for redislocation after surgical repair. Recent work by Gupta and Lee[81] offers a cause for posterior erosion of the glenoid. They evaluated glenoid–humerus contact forces in 12 overhead activity positions and found that there is a significant increase in contact pressure between the humeral head and posterior glenoid when the humerus is horizontally abducted to 70 degrees. They concluded that repetitive overhead activities may load the glenohumeral joint asymmetrically and lead to posterior glenoid erosion.[81]

Bony lesions of the anterior or posterior glenoid rim have also been described and are believed by some to be important enough to be formally reconstructed during open capsulorrhaphy. Edwards et al. found osseous lesions of the glenoid in 126 of 160 (78%) patients with chronic anterior glenohumeral instability.[61] These were seen on the glenoid profile view. These lesions are either due to an osseous Bankart or wear related to repeated instability. Pavlov et al.[194] described an osseous Bankart lesion of the anterior glenoid in 15% of patients with recurrent anterior dislocation and in approximately 50% of patients with recurrent anterior subluxation. Gerber[75] has advocated intraarticular iliac bone graft to formally reconstruct the glenoid cavity before capsular repair to restore normal anatomy of the glenoid. Bigliani et al.[24] believes that compromise of 25% or more of the glenoid surface warrants bony reconstruction. Burkhart et al.[38] recommend a coracoid process transfer (i.e., Latarjet procedure) when a bony Bankart lesion narrows the inferior half of the glenoid to a width that is less than that of the superior half of the glenoid (i.e., the inverted-pear configuration).

Defects smaller than 20% can be rendered extraarticular by repairing the capsule and labrum back to the edge of the intact glenoid. Larger fragments can be mobilized and fixed through traditional means.[33] Unlike the Hill-Sachs lesion, there are few data available to suggest which glenoid defects require repair, débridement, or neglect. Itoi et al.[104] investigated the effect of glenoid defect size on anteroinferior stability after Bankart repair. These authors found that with the arm in the position of apprehension, the size of the osseous defect does not affect stability of the arm. However, increasing the size of the osseous defect reduces stability when the arm is placed in abduction and internal rotation.[104] The overall aim of any reconstructive procedure directed at larger defects is to deepen the socket and support the capsule.

Articular Version Abnormalities

Clinically, excessive glenoid retroversion is thought to be a contributing factor to posterior instability and may infrequently be due to a variant of glenoid dysplasia. In most cases, however, excessive version is acquired from eccentric articular surface wear. Magnetic resonance imaging reveals that shoulders with posteroinferior instability have greater

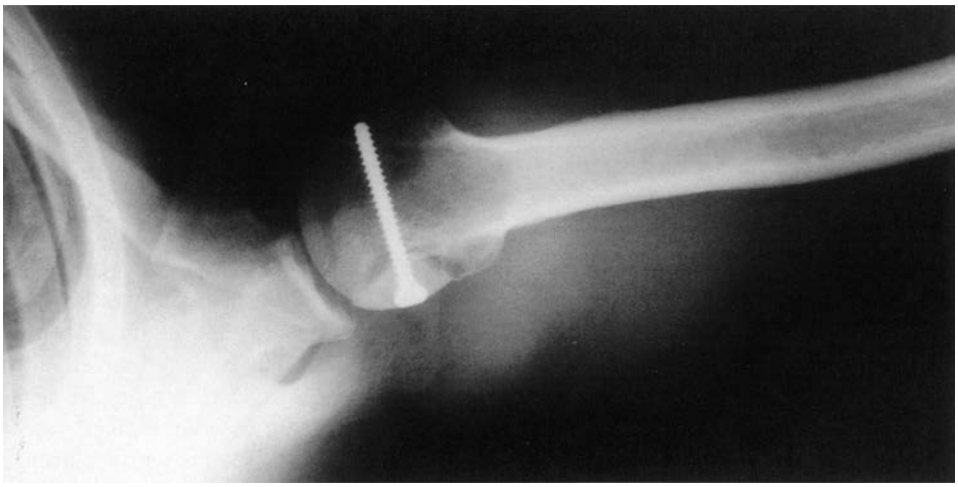

Figure 9-13 This large Hill-Sachs defect was treated with a Bankart repair and osteochondral allograft reconstruction of the humeral head.

retroversion of both the osseous and chondrolabral portion of the glenoid, and there is loss of height of the posterior portion of the labrum.[122] These features lead to loss in chondrolabral containment of the glenohumeral joint in patients with posteroinferior instability of the shoulder.[122]

Some surgeons recommend glenoid osteotomy in addition to soft tissue procedures.[36,125,225] Several other authors[125,205] have reported varying degrees of normal glenoid and humeral articular version, indicating that further study is needed to support a relation between the development of instability and bony alignment. Glenoid osteotomy and rotational humeral osteotomies, seemingly reasonable treatment options in the presence of articular version abnormalities, have been associated with the development of glenohumeral arthritis.[99,116] Currently, in North America, humeral rotational osteotomy or glenoid osteotomy is not commonly practiced, perhaps reflecting the unclear relation between these factors and clinical instability.

PUTTING IT ALL TOGETHER

Successful management of shoulder instability requires a thorough knowledge of all factors responsible for stability in addition to those pathologic factors contributing to instability. Shoulder instability may be viewed as any condition in which the balance of the various stabilizing structures is disrupted, leading to increased joint translation and the development of clinical symptoms.[89,191] Because the large spherical head of the humerus articulates with a relatively small and shallow glenoid, the glenohumeral joint requires several mechanisms to maintain stability while providing for a large range of motion. Static and dynamic stability is provided by the combined effects of the capsuloligamentous structures and rotator cuff and biceps. In the midranges of rotation, where the capsuloligamentous structures are lax, most joint stability is through the dynamic action of the rotator cuff and biceps tendons through concavity–compression of the humeral head within the glenoid socket. The ligamentous structures, which are primarily capsular thickenings, function only at the extreme positions of rotation, preventing excessive rotation of the humeral head on the glenoid. Contraction of the muscles around the shoulder may act secondarily by protecting the relatively weak ligamentous structures from being overwhelmed from excessive tension. Because interpretations of the literature are often confusing, this section is an effort to synthesize the findings already discussed.

The labrum provides an attachment site for the glenohumeral ligaments and the tendon of the long head of the biceps. Its principal function is to increase the depth of the glenoid socket and to act as a chock block in preventing the head from rolling over the anterior edge of the glenoid. Recently, however, the role of the labrum in preventing translation or instability has been challenged. The Bankart lesion, by its anatomic definition, implies dysfunction of the IGHLC, and possibly the SGHL and MGHL. Thus, virtually all labral lesions, especially those below the glenoid equator, are thought to be associated with glenohumeral instability. However, plastic deformation, capsular rupture, abnormal laxity, periosteal stripping, or any combination of these lesions may also be associated with complete dislocation, with or without the Bankart lesion. Thus, one of the goals of reconstructive surgery for glenohumeral instability is to anatomically reconstruct both the labral and capsular deficiency independent of cause (e.g., genetic predisposition or extrinsic forces).

The role of the capsule and ligaments in preventing instability is quite complex and depends on shoulder position and the direction of the applied force. Generally, the anterior capsule becomes more important during extension and the posterior capsule during flexion. Extremes of internal and external rotation have the effect of winding up the capsular structures, leading to joint compression and increased stability owing to tension developing in the relevant structures. In general, the inferior capsular structures

are most functional near full elevation and the superior capsular structures near full adduction.

The IGHLC is the primary static check against anterior, posterior, and inferior translation between 45 and 90 degrees of glenohumeral elevation. The SGHL and MGHL limit anteroposterior and inferior translation in the middle and lower ranges of elevation as the arm approaches the adducted position. Experimentally, posterior translation in the flexed, adducted, and internally rotated position may require disruption of the anterosuperior capsule (including the SGHL) in addition to the posterior structures. Although controversial, the SGHL, CHL, and IGHL probably function together to limit inferior translation of the adducted shoulder and act as secondary restraints against posterior translation. Clinically, these structures are addressed during either arthroscopic or open Bankart repair or capsulorrhaphy. However, simply overtightening the capsule to limit the end ranges of motion to achieve stability may lead to pathologic limitation of shoulder motion and late arthrosis.

The rotator interval region between the subscapularis and supraspinatus may be associated with abnormal translation, especially inferior translation of the adducted arm and, possibly, anteroposterior translation. Contraction of the rotator cuff and long head of the biceps brachii affects both static and dynamic factors that enhance stability. Primarily, they act in concert to increase compression across the glenohumeral joint, increasing the loads required to translate the humeral head. These factors are especially important in the midranges of motion where the capsuloligamentous structures are more lax. The long head of the biceps brachii is a significant secondary stabilizer when the capsuloligamentous structures begin to fail. The scapulothoracic stabilizers help accurately time and position the glenoid beneath the humeral head. Dysfunction in any of these stabilizers can lead to subsequent instability as residual stabilizing mechanisms become overwhelmed. Furthermore, proprioceptive mechanisms help to coordinate and time this system and can be restored after instability surgery.

Finally, the effects of abnormal articular surfaces, articular version, negative intraarticular pressure, and adhesion–cohesion, either in part or in combination, can lead to or worsen shoulder instability. By themselves, however, they may play only a small role in the pathogenesis of shoulder instability. Rarely is bone loss significant enough to warrant surgical correction. Unfortunately, clinical data are lacking for most of these factors, and an algorithmic approach to their treatment is currently evolving as experimental models improve.

CLASSIFICATION

The importance of a classification system for shoulder instability is best appreciated from observations of treatment failures resulting from improper matching of a surgical procedure with the appropriate pathology. Careful classification improves our ability to tailor individualized treatment programs for patients with glenohumeral instability. Although other classification systems exist for shoulder instability,[197] a system based on four factors is commonly employed: the degree of instability, the frequency of occurrence, direction, and cause of the instability (Table 9-4).

TABLE 9-4
SHOULDER INSTABILITY CLASSIFICATION

I. Degree
- A. Dislocation
- B. Subluxation
- C. Subtle

II. Frequency
- A. Acute (primary)
- B. Chronic
  - 1. Recurrent
  - 2. Fixed

III. Etiology
- A. Traumatic (macrotrauma)
- B. Atraumatic
  - 1. Voluntary (muscular)
  - 2. Involuntary (positional)
- C. Acquired (microtrauma)
- D. Congenital
- E. Neuromuscular (Erb's palsy, cerebral palsy, seizures)

IV. Direction
- A. Unidirectional
  - 1. Anterior
  - 2. Posterior
  - 3. Inferior
- B. Bidirectional
  - 1. Anteroinferior
  - 2. Posteroinferior
- C. Multidirectional

The degree of instability is proportional to the level of injury to the capsulolabral structures. *Dislocation* is defined as complete separation of the articular surfaces, often requiring a reduction maneuver to restore joint alignment. *Subluxation* is symptomatic instability without complete dislocation of the articular surfaces. These patients may complain of only pain without an appreciation for actual instability.

Subtle degrees of instability may be due to microtrauma, which may occur from overuse, as seen with repetitive overhead throwing. As in those with subluxation, some of these patients present with pain, with no knowledge of underlying instability. Additionally, patients with multidirectional or posterior instability may have associated tendinitis and pain without a sense of actual shoulder instability.[72,217,226] The avid overhead athlete may also present with pain in the posterior aspect (e.g., internal impingement of the posterior rotator cuff on the posterosuperior glenoid) of the shoulder

during the late cocking or early acceleration phase of throwing. Impingement, when present, is due to altered biomechanics and is a secondary phenomenon, rather than true mechanical impingement from the coracoacromial arch.[1,94,255] These can be difficult diagnostic and therapeutic problems.

The frequency of instability is described as acute or chronic. The temporal delineation between acute and chronic is not well defined in the literature, and those definitions that do exist may not have direct clinical relevance. However, for descriptive purposes, an *acute episode* of glenohumeral instability generally refers to the primary dislocation and is defined as one in which the patient is seen in the acute period (within several hours or even a few days) of the injury. These injuries may or may not need to be manually reduced, for occasionally they will reduce spontaneously. *Chronic instability* is in reference to either recurrent episodes of acute instability (a.k.a. recurrent instability) or dislocations that remain displaced for greater periods of time. The latter type may be more appropriately termed fixed or locked dislocations. Most commonly, this is seen in a missed or neglected posterior dislocation.[88]

The cause of instability may be categorized as traumatic, atraumatic, microtraumatic, congenital, or neuromuscular. As our understanding of the pathophysiology of shoulder instability has evolved, we now recognize that simplifying cause into atraumatic or traumatic is somewhat limiting. Thomas and Matsen[239] originally introduced the acronyms TUBS and AMBRI to help us think about the cause and treatment of most patients who have shoulder instability. The TUBS variety of instability describes a patient with macro*t*raumatic *u*nidirectional instability associated with a *B*ankart lesion that typically responds well to *s*urgery. The AMBRI variety of instability describes a patient with *a*traumatic *m*ultidirectional instability that is *b*ilateral and often responds to *r*ehabilitation; rarely, this type of instability requires an *i*nferior capsular shift.

Patients with this atraumatic instability may demonstrate the ability to voluntarily dislocate their shoulders. By selective muscle contraction and relaxation, these patients can position their shoulder to result in subluxation or dislocation. Most commonly, this is seen in cases of posterior and multidirectional instability, but pure anterior instability can also be produced. Rowe et al.[214] have observed that voluntary subluxation can be associated with emotional and psychiatric disorders of secondary gain. In general, this category of voluntary instability has a high rate of recurrence after surgical stabilization if the underlying psychopathology is not addressed.[169,214] A subtype of voluntary instability includes those who have an unconscious behavioral tic leading to selective muscular contraction. This form of voluntary instability may respond best to biofeedback techniques.[15]

Alternatively, dislocation may occur voluntarily with underlying involuntary instability, as with activities or even during sleep owing to instability that is positional. Although patients can voluntarily reproduce disabling instability just by positioning their arm, they prefer not to do so. Most commonly, these patients may have involuntary posterior instability that can be demonstrated by positioning the arm into flexion, adduction, and internal rotation. These patients often adapt by avoiding positions of risk where the shoulder might dislocate.[226] This positional type of involuntary instability, unlike voluntary instability caused by psychiatric factors or a behavior muscular tic, may respond well to surgical stabilization.[20,70]

Neer[170] recognized acquired instability that results from repetitive microtrauma (overuse) to the glenohumeral joint. These patients often provide a history of being an avid overhead athlete (e.g., baseball, swimming, tennis, and such) as they subject their anterior and inferior capsuloligamentous structures to repetitive injury and stretch causing symptomatic instability. These patients are not uncommonly found to have a preexistent constitutional congenital hyperlaxity.[60,62,184] Conceivably, these patients excel at their sport because of this excessive laxity and may develop symptomatic instability through subsequent trauma. Finally, neurologic disorders can lead to instability including stroke, Erb's palsy, and seizures, which can cause both anterior and posterior instability. Thus, rather than a discrete cause, there remains a spectrum of instability, with traumatic and atraumatic mechanisms occupying the extremes. This is reflected by the variety of findings observed at the time of surgery that may include any combination of a Bankart lesion, capsular laxity, or capsular rupture.

The direction of instability can be anterior, posterior, inferior, or any combination of these. Unidirectional instability occurs in only one of these directions. Multidirectional instability as in the AMBRI variety may demonstrate all three directions of instability in addition to generalized ligamentous laxity. The principal direction of instability in both the TUBS and AMBRI varieties of instability is usually anterior. However, the presence of inferior instability is the hallmark of the diagnosis of multidirectional instability. To add to already confusing nomenclature, Pollock and Bigliani[196] and Bigliani et al.[19] have described patients with an intermediate degree of instability, who demonstrate an inferior component in addition to an anterior or posterior component as bidirectional (e.g., anteroinferior or posteroinferior). This type of instability is more common in overhead athletes who expose their anterior and inferior capsular restraints to repetitive microtrauma, leading to plastic deformation and stretch. An additional subtlety is that patients with posterior instability often exhibit smaller degrees of inferior and even anterior instability.

Recognition of global capsular laxity and instability in more than one direction that is due to either of the extremes of causation (e.g., traumatic or atraumatic) is critical for determining appropriate surgical management so as not to exacerbate the instability in the direction left

unaddressed.[169] Often, it is the primary direction of instability that causes most of the patients' symptoms and is, therefore, most commonly addressed surgically. However, procedures that treat anterior capsular laxity by Bankart repair or capsular plication may not adequately manage the associated components of inferior and/or posterior instability. In the extreme, asymmetrical tightening during capsulorrhaphy can lead to a fixed subluxation in the opposite direction.[17,141] Thus, establishing the principal direction of the instability and acknowledging the lesser components by the time of surgical intervention is critical to obtain a successful outcome.

CONCLUSIONS

The aims of this chapter were to review the anatomy, biomechanics, and pathophysiology of shoulder instability. An understanding of what is "normal" provides a foundation for diagnosing and treating what is considered to be pathologic. Because current research endeavors have focused on the basic science of shoulder instability, we no longer have to rely on an anecdotal and qualitative account of the associated pathology noted at the time of treatment. We now have an organized and quantitative approach to the treatment of shoulder instability. Anatomic studies have provided abundant information on the macro- and ultrastructure of the static and dynamic restraints to stability. Experiments examining the biomechanics of shoulder instability have helped clarify the effects of articular version, the labrum, negative intraarticular pressure, the material properties and limits of function of the capsuloligamentous complex, and the dynamic interaction between static and dynamic restraints. There are still several unanswered questions. As technology is advancing, we must continue to evaluate how older and newer techniques correct anatomic and biomechanic abnormalities leading to glenohumeral instability. Newer forms of "heat therapy" and arthroscopic techniques are exciting means to perform less invasive surgery. However, meticulous analysis will be required to determine their value. Our current understanding of anatomy and biomechanics should greatly facilitate this goal.

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10 Biomechanics and Pathologic Lesions in the Overhead Athlete

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INTRODUCTION

The goal in treating overhead athletes is not simply to eliminate their pain, but also to return them to play. Today, knowledge of shoulder anatomy, pathoanatomy, and biomechanics has dramatically improved treatment of injuries in the overhead athlete. Return to play is probable after treatment of numerous, common shoulder maladies.

To accommodate a large range of motion, there is less bony stability at the shoulder than other diarthrodial joints. Simple shoulder motion necessitates the coordinated actions at four separate articulations. The demands placed on the shoulder during rigorous overhead athletics are appreciable. Injury to the soft tissues that guide and limit motion can cause significant loss of function. Less common in the general population, this is a frequent finding in overhead athletes participating in activities that stress soft tissues near their physiologic limits.

In this chapter, the biomechanics and pathologic lesions of the shoulder in the overhead athlete are described. The stages of the throwing motion, including electromyographic (EMG) data, are described for both the normal and the injured shoulder. Baseball pitching is used as an example, but swimming, football, and tennis are also described. Then, patterns of shoulder injury and management principles in the overhead athlete are reviewed. Lastly we will detail postoperative care including rehabilitation of the overhead athlete and indications for return to play.

BIOMECHANICS OF THE OVERHEAD MOTION

Role of Arm Position

Clinical evaluation of the overhead athlete requires knowledge of normal mechanics of the sport and is best assessed during the athletic endeavor. The most important aspect of proper mechanics is that the motion is natural. This applies to all players, whether in baseball (when the ball is thrown from the outfield or the pitching mound), swimming, football, tennis, or any of the other activities that require an overhead motion.

Baseball is a good example of our needing to know the biomechanics and pathologic lesions common in the overhead athlete. Injuries to the shoulder account for most injuries to adult baseball players,[2] and most of these injuries occur during the throwing motion. A professional baseball pitcher may play 30 to 40 games in a season, throwing as many as 150 pitches in a game, many of these at high velocity.[72] Biomechanics change and improve with level of play from youth to professional. While amateur pitchers may not play as many games in a season, they still try to throw the ball as fast as possible while maintaining accuracy. It is not surprising that shoulder pain is a frequent complaint among baseball pitchers of all skill levels.

When an overhead athlete complains of shoulder pain, one should obtain the relationship of the shoulder pain to sports activity and arm position. A baseball pitcher who complains of shoulder pain during the cocking stage of the throwing motion may have anterior shoulder instability. The pitcher who has pain during the follow-through stage may have posterior shoulder instability. For best chances in correct diagnosis and treatment of shoulder injury in the overhead athlete, one should know the specific biomechanics of the athlete's sport and the specific activities that result in arm positions that make the shoulder vulnerable to injury.

Overhead throwing is a component of many sports. Caring for athletes participating in all of these different sports can be a challenge. Fortunately, the biomechanics of different overhead athletes are more similar than they are different. Of all overhead athletes, the complex biomechanics of throwing a baseball has been most studied.[14,29,32,33,47,48,50,72] Therefore, in the following section we will describe the biomechanics of baseball first. Because of differences that warrant special descriptions of other overhead athletic endeavors, we will also describe the biomechanics of football, swimming, and tennis.

Sport-Specific Biomechanics

Baseball

Stages of the Pitching and Throwing Motion, Including Electromyographic Data

Baseball throwing is a total body activity. It is an elaborate, synchronous progression of body movements that starts in the legs and trunk, proceeds to the upper extremities, and concludes in the rapid propulsion of the ball. The effectiveness of a baseball throw is determined by various factors, including velocity, accuracy, spin production, and endurance. Synchrony of muscular contractions and neurologic control throughout the body are essential to produce an effective throwing motion. Effectiveness also necessitates repetitive performance at a level that maximally stresses the physiologic limits of the shoulder. There is a delicate balance between mobility and stability of the joints of the upper extremities while throwing, and maintenance of this fragile balance is paramount. Small aberrations in the mechanisms that control stability have a significant and cumulative effect on upper extremity function and increase the risk of crossing the fine line between maximal throwing effectiveness and injury.

The mechanics of baseball throwing seem to differ slightly between player positions, but, in essence, the motions are quite similar. When an outfielder throws the ball, he or she first takes a step backward and then lands on the opposite forefoot with the knee slightly flexed. A pitcher should do the same. When the pitcher overthrows to get more speed on the ball, he tends to hyperextend the knee and land on the heel.[82] This places sudden, large forces on the shoulder during the cocking stage of throwing. Additionally, when the pitcher starts to fatigue, the elbow begins to drop and he or she increases the lordosis in the spine. This is how a pitcher with poor mechanics from back pain may later develop shoulder problems. For a specific athlete, any deviation from the normal, natural motion may be indicative of injury.

Most throwing studies have concentrated on the pitcher, because the motion is more constant, the collection of EMG data is easier, and pitchers frequently injure their arms. From a virtual standstill, a professional pitcher will accelerate a 142-g baseball to a release velocity of more than 90 miles per hour in just 50 msec. Tremendous tensile, compressive, and rotational forces must be created and dissipated in the shoulder. A detailed description of the throwing motion will help clarify how this is done.

The Normal Shoulder. The baseball pitch is divided into five stages (Fig. 10-1): windup, early cocking stage, late cocking stage, short propulsive stage of acceleration, and follow-through or deceleration stage, which can also be divided into early and late.

At stance, the pitcher stands facing the batter with the shoulders parallel to the rubber. The pivot foot (right for right-handers) is positioned on the rubber.

In the windup stage, the body mechanics are quite individual. In general, windup begins with the stride foot (left for right-handers) coiling backward, away from home plate, and the arms swinging overhead. At this time, the position of the fingers on the ball is finalized while screened by the glove. The pivot foot rotates on the rubber as weight is transferred to it. This stage ends with the ball

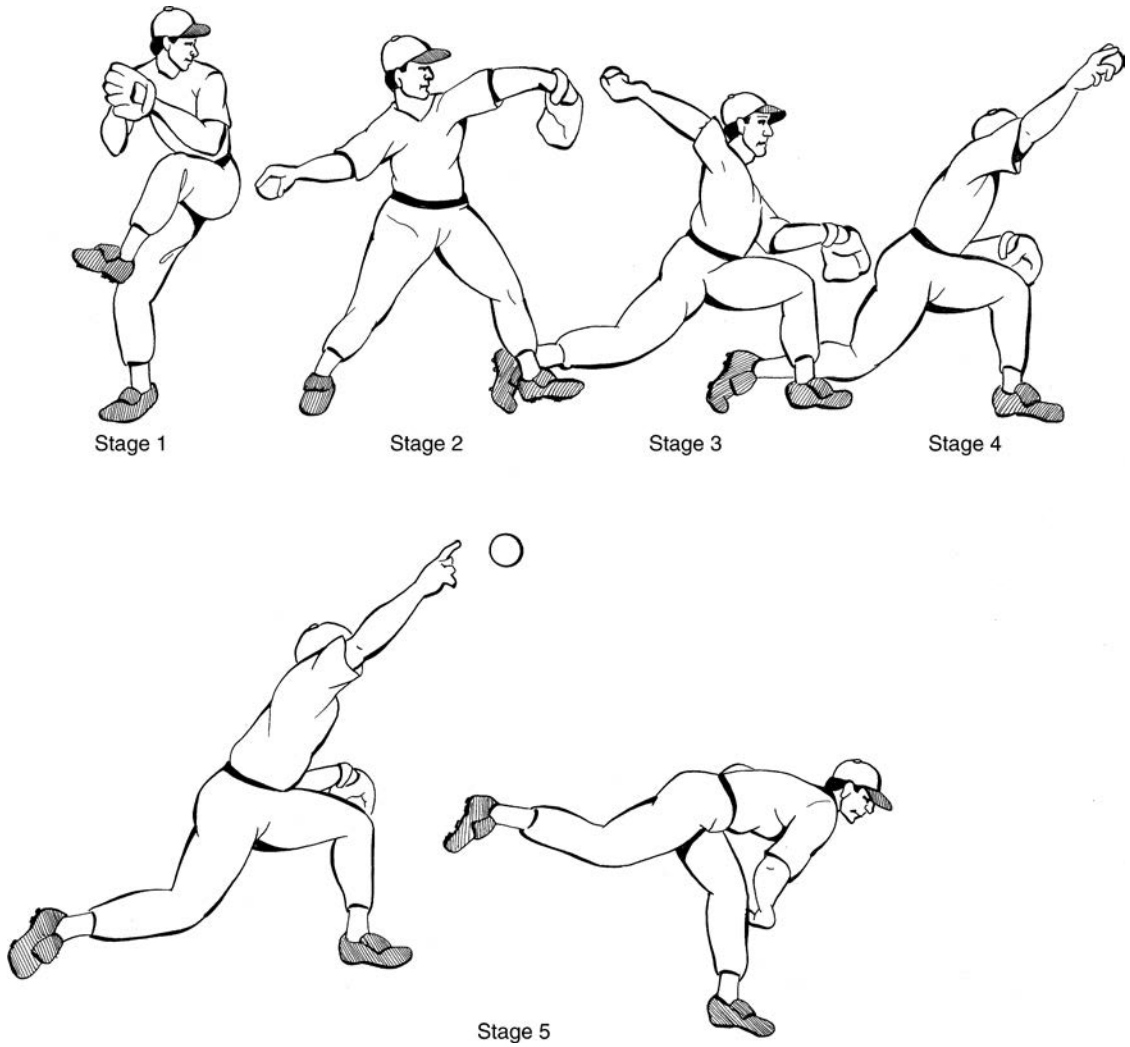


Figure 10-1 The five phases of the baseball pitch.

leaving the glove hand and the body balanced on the pivot foot. The EMG activity of the shoulder girdle and upper extremities is low during the windup, which reflects a lack of critical events in these anatomic locations related to performance or to injury potential. Instead, there is critical activity in the trunk and lower extremities during the windup. Common to all pitchers during the windup is a stable base to initiate the pitch. Good stance limb stability results from an upright and balanced trunk to minimize anterior-to-posterior sway of the body.

Cocking is divided into early and late stages. It begins when the ball leaves the glove. Early cocking starts with hip extension of the pivot leg that propels forward the stride leg, nondominant upper extremity, and trunk. The gluteus maximus of the pivot leg is important in providing this propulsion. This critical step in pitching is known as "leading with the pelvis." It is important that the pitcher strides to prevent early external rotation of hip. This "opening" of the pelvis and trunk pivot the body when it should be propelled forward. Temporarily, the dominant upper extremity lags behind the rest of the body. The trapezius and the serratus anterior muscles form a force couple to upwardly rotate and protract the scapula. This scapula motion is essential to place the glenoid in a stable position for the abducting and rotating humeral head. If the scapula is not positioned correctly, impingement can occur.[22] The deltoid and supraspinatus muscles act in synergy to abduct the humerus. Saha[81] described the glenohumeral muscles as drivers and steerers. In this case, the deltoid is the driver of the motion, and the supraspinatus is the steerer that fine-tunes the position of the humeral head in the glenoid. The remainder of the rotator cuff muscles have less activity during this phase,[22] indicating the importance of the supraspinatus in functioning with the deltoid in humeral abduction during this stage. The hand should be on top of the ball to prevent early external rotation of the shoulder and supination of the forearm.

Late cocking begins with rapid forward motion of the trunk.[29] The dominant shoulder rotates forward. Excessive horizontal abduction of this shoulder (e.g., "hyperangulation") is minimized by keeping the lead arm closed, in front

of the body. Abduction of the humerus is maintained and external rotation increases from 46 to 170 degrees.[25] Muscle forces are needed to overcome the inertia and gravity that act on the shoulder in horizontal abduction, external rotation, and adduction. Static and dynamic restraints combine to stabilize against these forces. In this position, the primary static anterior stabilizer of the glenohumeral joint is the anterior band of the inferior glenohumeral ligaments.[88] Although the supraspinatus and the deltoid activity diminish as the humerus ceases to abduct, the other rotator cuff muscles increase in activity to stabilize the humeral head.[22] The subscapularis acts as a barrier to anterior translation, together with the pectoralis major and the latissimus dorsi. These muscles act as a dynamic sling to augment the inferior glenohumeral ligament.[22] The posterior rotator cuff muscles are also quite active during late cocking as the infraspinatus and teres minor actively externally rotate the humerus. Their posterior placement also enables them to act as checkreins to anterior subluxation. Activity of the trunk and lower extremities is critical throughout pitching. Keeping the stride foot directed toward home plate minimizes over- or under-rotation of the body during the late cocking phase.

Although there is no further abduction of the shoulder during late cocking, the scapulothoracic muscles continue to be active to produce a stable platform for the humeral head and to enhance maximal humeral external rotation.[22] The middle portion of the trapezius, the rhomboids, and the levator scapulae are all key in providing scapular stabilization. The serratus anterior is also important in opposing retraction to stabilize the scapula. Sometimes symptoms of anterior instability of the glenohumeral joint are demonstrated during this stage because of an imbalance of the scapula-stabilizing mechanisms.[47] The cocking phase of pitching ends when the stride foot contacts the ground. It is important that the dominant arm is in a good throwing position at the end of cocking. Also, the lead shoulder should remain closed until the stride foot lands.

The acceleration stage begins with maximal shoulder external rotation and terminates with ball release. The humerus internally rotates approximately 100 degrees in one-half second.[25] The humeral internal rotation torque is 14,000 in.-lb, with an angular velocity of 6,100 degrees per second.[29,33] The acceleration of the arm is coincident with the deceleration of the rest of the body, producing efficient transfer of energy to the upper extremity and ball.[29] A large glenohumeral joint compressive force (860 N), which has a stabilizing effect, also occurs. Synchronous muscular contraction about the glenohumeral joint balances the requirements of stabilization and rapid motion. A stable scapula is needed, and all of the scapular muscles have high activity in providing this function.

Shoulder angular velocity during acceleration is transferred from the trunk, with augmentation by the latissimus dorsi and the pectoralis major. The latissimus dorsi has even higher activity than the pectoralis major,[22] and it is anatomically positioned to generate large torques.[6] These two muscles are important in actively contributing to ball velocity, evidenced by a clinical study that reported they are the only two muscles to have a positive correlation between peak torque in isokinetic testing and pitching velocity.[3] The subscapularis, especially the upper portion, also has very high activity during the acceleration stage and functions with the pectoralis major and latissimus dorsi.[22] Whereas the pectoralis major and the latissimus dorsi are the primary propellers of the arm, the subscapularis functions as a steering muscle to position the humeral head precisely in the glenoid. This coordinated function of the subscapularis with the latissimus dorsi has been observed in other overhead athletics as well.[74,75] The teres minor activity is also high, with the muscle acting as a checkrein to anterior instability. Athletes may note symptoms during this stage of throwing, the most typical problem being anterior instability.

Follow-through is after ball release and can also be divided into early and late stages. Initially, the trunk and dominant lower extremity rotate forward. The shoulder continues to adduct and internally rotate to 30 degrees. Kinetic energy not transferred to the ball must be absorbed by the decelerating arm and body. Deceleration is estimated to be 500,000 degrees per second^3 at the shoulder, with an external rotation torque of approximately 15,000 in.-lb at the humerus.[29,72] In general, deceleration of the upper extremity is accomplished by simultaneous contraction of opposing muscles around the shoulder.[22] The trapezius, serratus anterior, and rhomboids all demonstrate high or very high activity. The deltoid is active, especially the posterior and middle portions, which are positioned to oppose the motion of the upper extremity. The teres minor has the highest activity of all the glenohumeral muscles, continuing to provide a posterior stabilizing checkrein. Injury to the posterior glenohumeral joint stabilizers will commonly become apparent during this stage. Late follow-through is a noncritical stage, with all of the shoulder muscles exhibiting decreasing activity.[22] All of the kinetic energy has been dissipated, and the trunk is beginning to extend, allowing the pitcher to field the position.

In summary, the throwing motion requires the rapid transmission of immense forces throughout the shoulder. This has been estimated to be 27,000 in.-lb, which is four times that in the leg during a soccer kick[29]; this puts the shoulder at great risk for injury.

The Injured Shoulder. Alterations from these normal muscle activities occur in the injured shoulder. In athletes with anterior glenohumeral joint instability, Glousman and coworkers[32] found that elite pitchers with anterior joint instability had decreased serratus anterior EMG activity in all stages of the pitch when compared with normal. There were, in fact, significant differences between these two

groups in the EMG activity of all the muscles tested, except the middle deltoid. The biceps and supraspinatus muscles had more activity in the unstable shoulders. This was thought to be a compensatory mechanism to help stabilize the humeral head against the glenoid fossa. In addition, the infraspinatus showed increased activity during the late cocking phase of the pitching motion in those with instability.

Alterations from normal serratus anterior muscle activities are meaningful. Because of the rapidity of upper extremity motions, observation of scapula motion is nearly impossible during these overhead activities. However, during simple shoulder motions such as abduction, EMG activity in the serratus anterior muscle in the shoulder with anterior joint instability is diminished when compared to normal.[62] These findings indicate an abnormality in the coordinated rotation of the scapula on the thorax, termed *scapulothoracic rhythm*. Known primarily as a protractor that prevents winging of the scapula, the serratus anterior muscle is also important in the scapulothoracic rhythm during shoulder elevation.[5] Normal scapulothoracic rhythm has been extensively studied,[23,28,42,76] and these studies indicate that the normal scapulothoracic rhythm has been altered, possibly resulting in a scapular lag.

Football

The football throwing motion is similar to the baseball throw except that there is no windup.[52] Thus, there are four sequential phases (Fig. 10-2): early cocking, late cocking, acceleration, and follow-through. Early cocking is initiated at rear foot plant and continues to maximal shoulder abduction and internal rotation. Late cocking starts at maximal shoulder abduction and internal rotation and ends with maximal shoulder external rotation. The acceleration phase began with maximal shoulder external rotation and ends with ball release. Finally, the follow-through starts at ball release and ends with maximal horizontal adduction of the shoulder.

The rotator cuff muscles have the high levels of EMG activity throughout the football throw. Activity in the supraspinatus and infraspinatus muscles is similar. Both have moderate activity in early cocking, late cocking, and acceleration and high activity during follow-through. Activity in the subscapularis differs from the supraspinatus and infraspinatus muscles during the football throw. It is minimally active during early cocking and moderately active during late cocking but has high activity in both the acceleration and follow-through phases.

All three heads of the deltoid muscle are minimally to moderately active throughout all four phases. The anterior deltoid muscle is minimally active during early cocking and moderately active during late cocking, acceleration, and follow-through. The middle deltoid is minimally active during the first three phases and moderately active during follow-through. The posterior deltoid is minimally active during early cocking, late cocking, and acceleration, and moderately active during follow-through.

The pectoralis major muscle is minimally active during early cocking, moderately active during late cocking, and highly active during acceleration and follow-through. The latissimus dorsi muscle is minimally active during early and late cocking, moderately active during acceleration, and

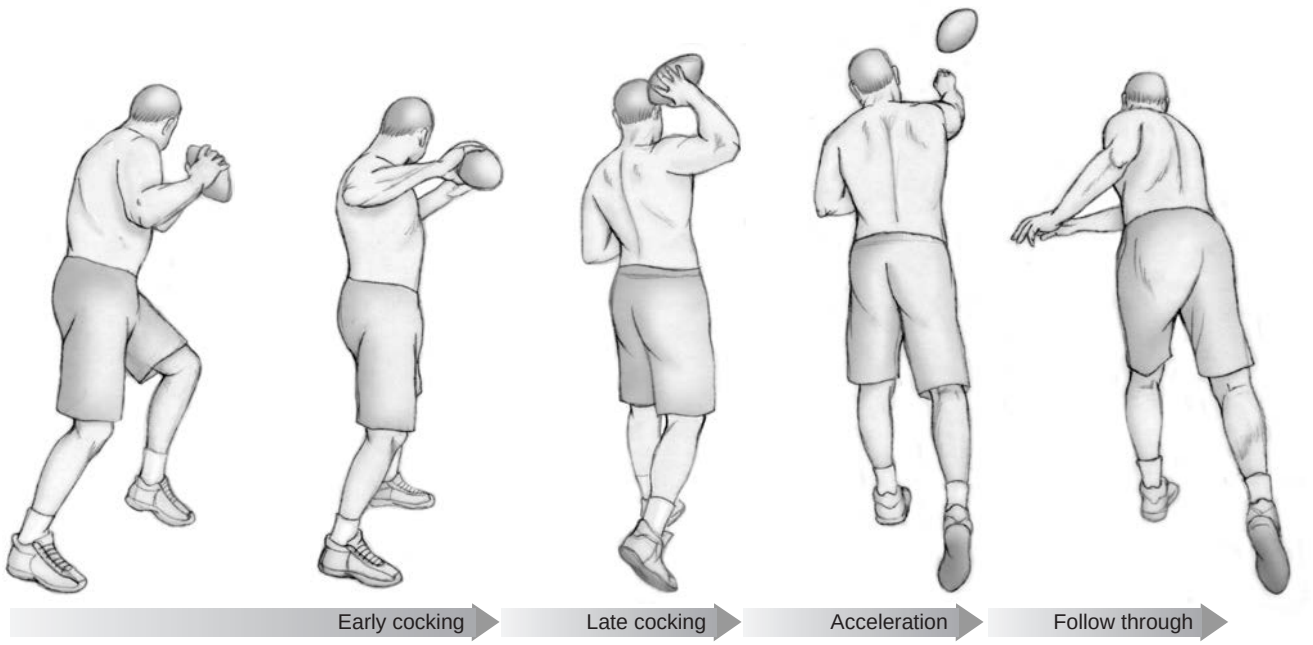


Figure 10-2 The five phases of the football throw.

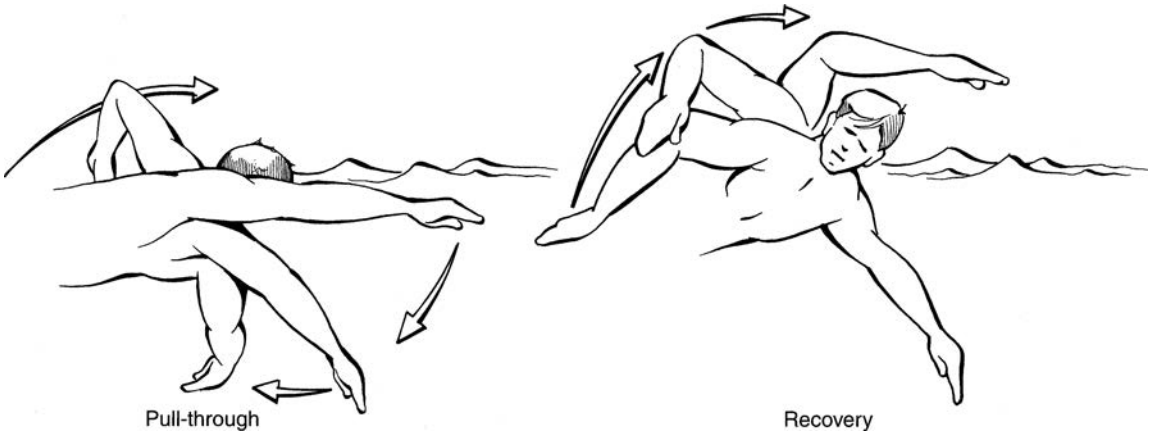


Figure 10-3 The pull-through phase of the freestyle stroke can be subdivided into hand entry, mid–pull-through, and end of pull-through. The recovery phase can be subdivided into elbow lift, midrecovery, and hand entry.

highly active during follow-through. Lastly, the biceps brachii muscle is minimally active throughout all four phases.

Swimming

The swimming strokes can be divided into the pull-through and recovery phases.[78] In the freestyle stroke the pull-through phase is subdivided into hand entry, mid–pull-through, and end of pull-through (Fig. 10-3). During hand entry, the shoulder is internally rotated and abducted, and the body roll begins. In mid–pull-through the shoulder is at 90 degrees abduction and neutral rotation. Body roll reaches a maximum of 40 to 60 degrees from horizontal. With the end of pull-through, the shoulder is internally rotated and fully adducted as the body returns to horizontal. The recovery phase is subdivided into elbow lift, midrecovery, and hand entry. In elbow lift, the shoulder begins to abduct and rotate externally. The body roll begins in the opposite direction from pull-through. In midrecovery, the shoulder is abducted to 90 degrees and externally rotated beyond neutral. Body roll reaches a maximum of 40 to 60 degrees in the opposite direction. Breathing occurs by turning the head to the side. In hand entry, the shoulder is externally rotated and maximally abducted, and the body is returned to neutral roll.

In the backstroke at hand entry, the shoulder is externally rotated and abducted as body roll begins from the neutral position. In mid–pull-through, the shoulder is abducted 90 degrees in neutral rotation with maximum body roll. At the end of pull-through, the shoulder is internally rotated and adducted, and body roll is horizontal. In the recovery phase of the backstroke, there is hand lift, rather than elbow lift. In hand lift, the shoulder begins with abduction and external rotation, and the body roll allows the arm to clear the water. In midrecovery, the shoulder is 90 degrees abducted and body roll is maximum.

In the butterfly stroke, the pull-through phase is the same as in the freestyle, but there is absence of body roll in all stages. To avoid shoulder flexion or extension, the hands are spread apart at the mid–pull-through stage. The recovery phase is again similar to that in the freestyle, with an absence of body roll. Body lift allows both arms to clear the water. Shoulder flexion and extension do not occur.

EMG activity indicates that the rotator cuff is important during swimming.[75,78] The supraspinatus, infraspinatus, and middle deltoid activities are predominant in the recovery phase. They abduct and externally rotate the extremity in preparation for a new pull-through. This position, similar to the cocking phase of throwing, places the shoulder at risk for subacromial impingement.[38] The serratus anterior also has an important function during recovery. It allows the acromion to rotate clear of the abducting humerus and provides a stable glenoid base on which the humeral head can rotate. The serratus anterior works at nearly maximal levels to accomplish this. Significantly decreased EMG activity of the serratus anterior muscle was found in swimmers with shoulder pain. During the pulling stage of the freestyle stroke there was significantly less activity in the serratus anterior in subjects with a painful shoulder when compared with normal.[83] The serratus anterior demonstrates similar findings during the pull-through stage of the butterfly stroke in subjects with a painful shoulder.[73] If this muscle becomes fatigued during the course of a number of cycles, scapular rotation may not coincide with humeral abduction. As a result, impingement syndrome may occur.

The biceps brachii muscle exhibited erratic activity during all of the strokes and functioned primarily at the elbow, which is similar to its role in pitching. The latissimus dorsi and pectoralis major were propulsive muscles, with a resulting action similar to that of the acceleration phase of throwing.

In summary, particular attention must be paid to conditioning the rotator cuff and serratus anterior muscles in an

effort to decrease the common problem of swimmer's shoulder impingement syndrome. Exercises must concentrate specifically on increasing the endurance of the serratus anterior muscle.

Tennis

The tennis serve can be divided into the same stages of complex muscle activity as a baseball pitch. The deltoid muscle function is low during cocking compared with pitching because trunk rotation contributes to shoulder abduction. The acceleration and follow-through stages in the tennis serve demonstrate muscle patterns and activity that are similar to those observed in throwing a baseball. Because the motions for serving the tennis ball are similar to those for pitching, tennis players may benefit from the same conditioning program as that outlined for pitchers. Likewise, emphasis should be placed on rehabilitating the rotator cuff and serratus anterior muscles.

The ground strokes, both forehand and backhand, can be divided into three stages (Fig. 10-4). In stage I, racquet preparation begins with shoulder turn and ends with the initiation of weight transfer to the front foot. Stage II is acceleration and begins with weight transfer to the front foot accompanied by forward racquet movement and culminates at ball impact. Stage III is follow-through and begins at ball impact and ends with completion of the stroke. The forehand ground stroke reveals a relatively passive windup sequence. Trunk rotation provides some of the force for shoulder motion. In follow-through, there is a marked decrease in activity among the accelerating muscles and a concomitant increase in the external rotators responsible for deceleration. The backhand ground stroke is similar in concept, but opposite in muscle activity, to the forehand. Follow-through demonstrates deceleration with increased activity of the internal rotators.

SPORT-SPECIFIC PATTERNS OF SHOULDER INJURY

Biomechanics of Pathology in the Overhead Athlete

Study of shoulder biomechanics has been invaluable in our understanding of pathogenesis,[61,86] pathoanatomy,[11,54,56,63,88] diagnosis,[37,64] and treatment [12,30] of common pathologic lesions. Recently, cadaveric models that simulate injury in the overhead athlete's shoulder were developed to improve our understanding of the athlete's shoulder.

Cadaveric Models of the Overhead Athlete without Muscle Forces (Fig. 10-5)[34]

First, nondestructive stretching of the anterior capsule was simulated while the humeral head was constrained in the glenoid. This resulted in a significant increase in external rotation of the shoulder. Next, posterior capsular contracture was simulated and resulted in decreased internal rotation. There was also a significant increase in anterior translation after nondestructive capsular stretching. The humeral head also was noted to have changed position during testing. Rotation of the humerus from neutral to maximum external rotation caused a posterior–inferior shift of the humeral head. This did not change significantly after anterior capsular stretching. Following a simulated posterior

Figure 10-4 The ground strokes of the tennis stroke, both forehand and backhand, can be divided into three stages: racquet preparation, acceleration, and follow-through.

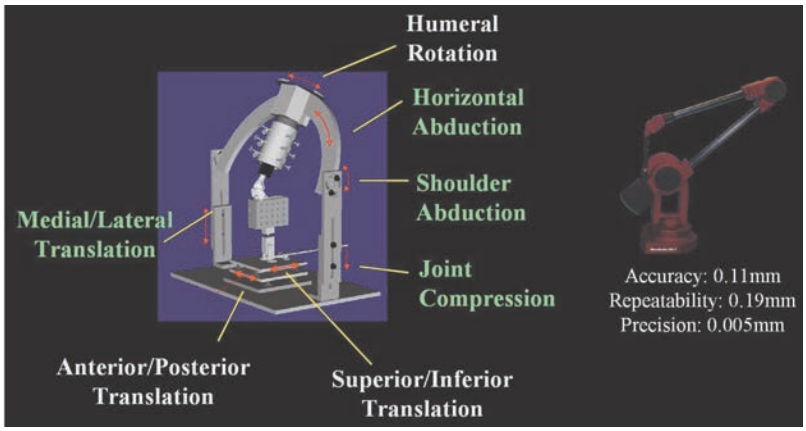


Figure 10-5 Cadaveric models of the overhead athlete without muscle forces.

capsular contracture, there was a significant superior shift of the humeral head at maximum external humeral rotation when compared to the stretched condition (2.0 ± 0.6 mm, $p = 0.013$). A posterior capsular contracture with decreased internal rotation would not allow the humerus to externally rotate into its normal posterior–inferior position in the cocking phase of throwing. Instead, the humeral head is forced posterior–superior (Fig. 10-6), which may result in a superior labrum from anterior to posterior (SLAP) lesion by a peel-back mechanism of humeral head impingement on the posterosuperior glenoid. (See the section on "SLAP lesions" in "Common Injuries in the Overhead Athlete" in this chapter for explanation of the proposed effect of posteroinferior capsular contracture as a cause of shoulder injury in the overhead athlete.)

Cadaveric Models of the Overhead Athlete with Muscle Forces

In a second cadaveric shoulder model for overhead athletes,[55] the shoulder muscles were simulated. Changes in glenohumeral kinematics and joint reactive forces were then studied through a range of motion. Cadaveric shoulders were tested in 90 degrees of shoulder abduction in the scapular plane using a custom shoulder jig with a six degree–of–freedom load cell and a microscribe digitizing system. The muscles were loaded with a pulley system used to approximate the muscle force vector toward the center of the muscle belly. The deltoid, infraspinatus, and teres minor were each loaded as were the supraspinatus, subscapularis, pectoralis major, and latissimus dorsi. Glenohumeral joint forces and the path of humeral head center were measured under three conditions; the final condition simulated the shoulder of an overhead athlete. These included an intact condition, a condition of nondestructive stretching of the anterior capsule, and a condition where posterior capsular contracture was simulated. The humeral rotational range of motion was measured from maximum external rotation to maximum internal rotation for each condition and the glenohumeral joint forces and the path of the humeral head center were quantified.

Nondestructive stretching of the anterior capsule in this cadaveric model resulted in external rotation increasing an average increase of 13 degrees, while internal rotation increased 1 degree. Internal rotation was decreased an average of 9 degrees in the condition where posterior capsular contracture was simulated and external rotation decreased 5 degrees. There was also a shift in the path of the humeral

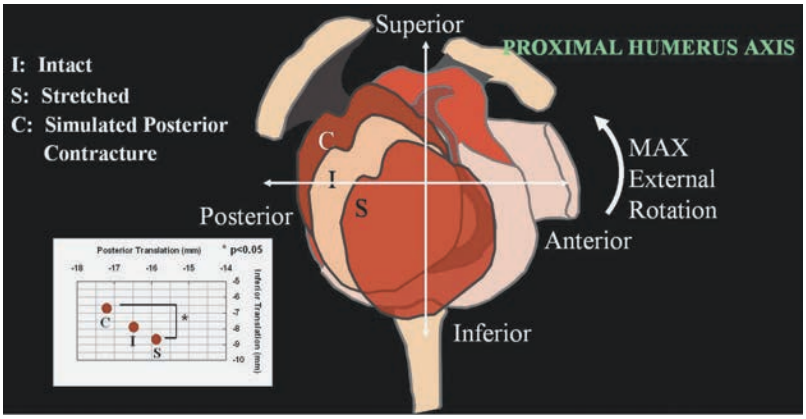


Figure 10-6 A posterior capsular contracture with decreased internal rotation does not allow the humerus to externally rotate into its normal posterior–inferior position in the cocking phase of throwing. Instead, the humeral head is forced posterosuperior, which may explain the cause of SLAP lesions in overhead athletes.

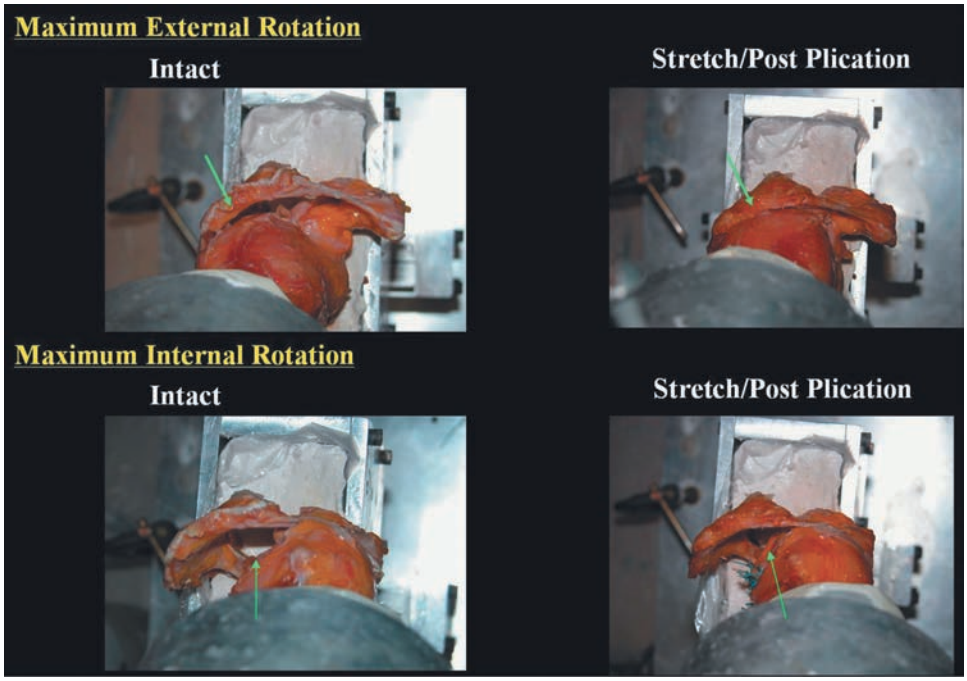


Figure 10-7 Glenohumeral joint forces and the path of the humeral head center were measured under three conditions with the final condition simulating an overhead athlete's shoulder. The path of the humeral head center with respect to the glenoid only exhibited statistically significant differences at extreme ranges of motion.

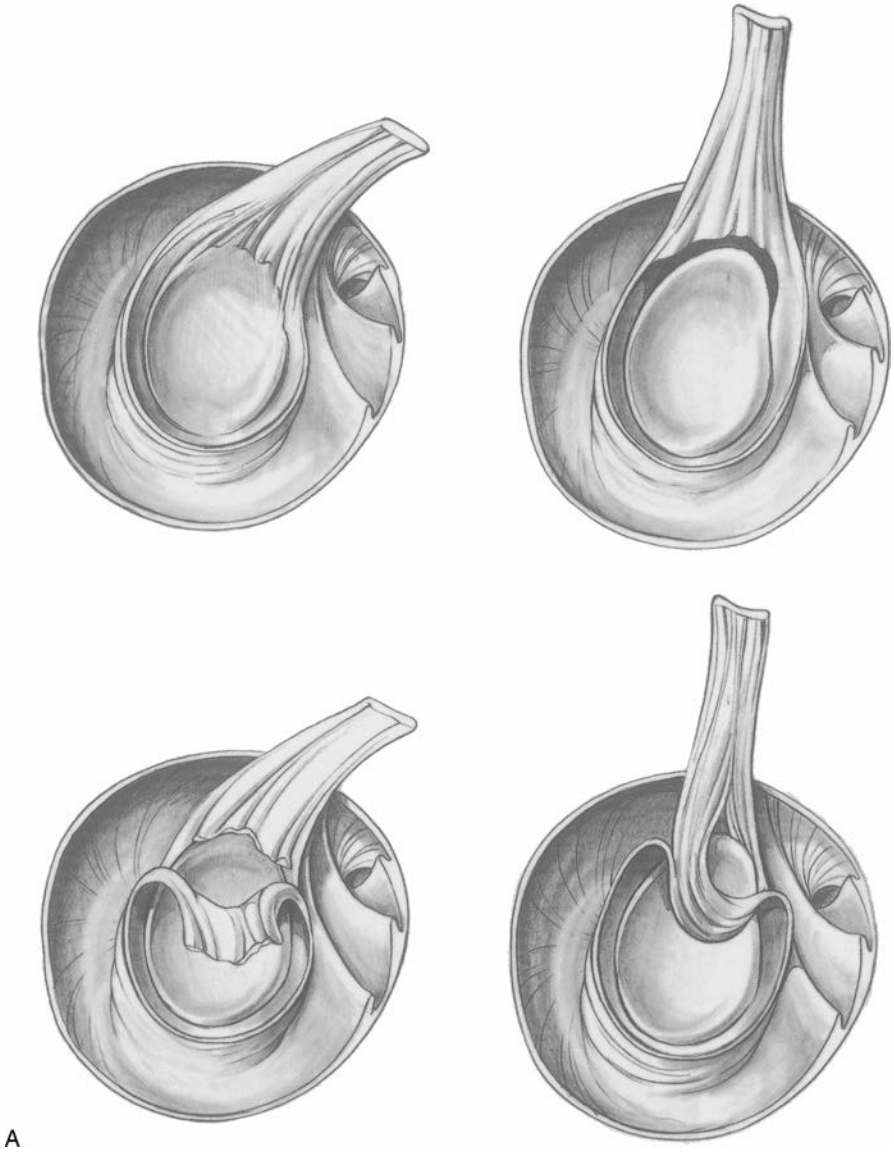
head center with respect to the glenoid near the limit of external rotation (Fig. 10-7). Compared to the intact condition, at maximum external rotation the humeral head was more inferior in the condition of nondestructive stretching of the anterior capsule and was more superior in the condition where posterior capsular contracture was simulated. With internal rotation of the abducted shoulder and in the condition where posterior capsular contracture was simulated, the humeral head was displaced inferiorly compared to the intact and stretched state.

In the anterior-to-posterior directions there were significant differences in the condition where posterior capsular contracture was simulated, but only at 15 degrees of external rotation. Then, the humeral head center was more anterior compared to the intact condition. It was also more anterior than that in the condition of nondestructive stretching of the anterior capsule. There were no statistically significant differences of the joint forces in comparison of the three conditions.

When comparing the path of the humeral head center from external rotation to internal rotation, capsuloligamentous effects were found throughout the range of motion but reached statistical significance only at the extremes where the static stabilizers make their greatest contribution. The concavity compression stability effect[56] afforded by the muscular forces was well demonstrated with this cadaveric model. In this study, the path of the humeral head center did not vary greatly and the forces about the glenohumeral joint did not vary significantly after differing conditions that simulated the shoulder of the overhead athlete. While this model does not simulate the varying muscles forces, both eccentric and concentric, through the throwing motion, it demonstrates that the muscles have a strong influence on glenohumeral joint stability. Also, impingement of the humeral head on the superior glenoid may occur secondary to the shoulder muscles functioning to maintain glenohumeral joint balance and congruency.

SLAP Lesion

In overhead athletes, lesions involving the superior labrum and the biceps anchor cause shoulder pain and instability.[84] The long head of the biceps tendon has long been thought to have a role in preventing superior translation of the humeral head.[24,31] Experimental work has subsequently demonstrated that it stabilizes the humeral head on the glenoid in both the anterior-to-posterior direction[44,69,79] and the superior-to-inferior direction.[45,69] Superior translation of the humeral head has also been demonstrated in vivo in individuals after isolated rupture of the long head of the biceps.[91] Both the long head and the short head of the biceps contribute to anterior stability, and this stabilizing effect is more significant when there is joint instability from a Bankart lesion.[45] Thus, the origin of the biceps is one of the structures that act in synchrony to stabilize the humeral head on the glenoid.



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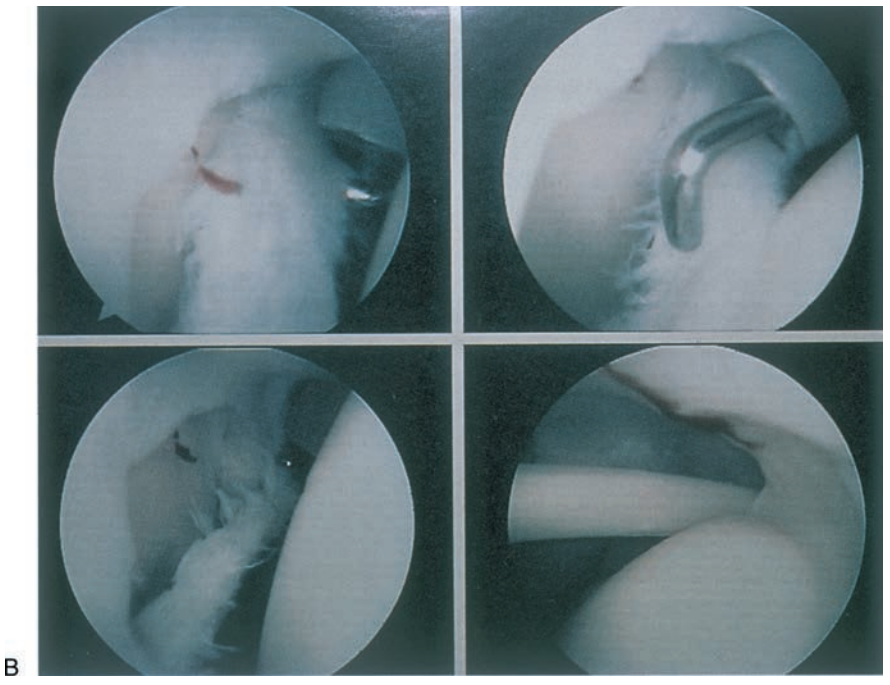

B

Figure 10-8 **(A)** Line drawing of the SLAP lesion types. **(B)** Arthroscopic photograph of a type III SLAP lesion. **(C, D)** Arthroscopic photograph of a type IV SLAP lesion. (B and C courtesy of JP Iannotti.)

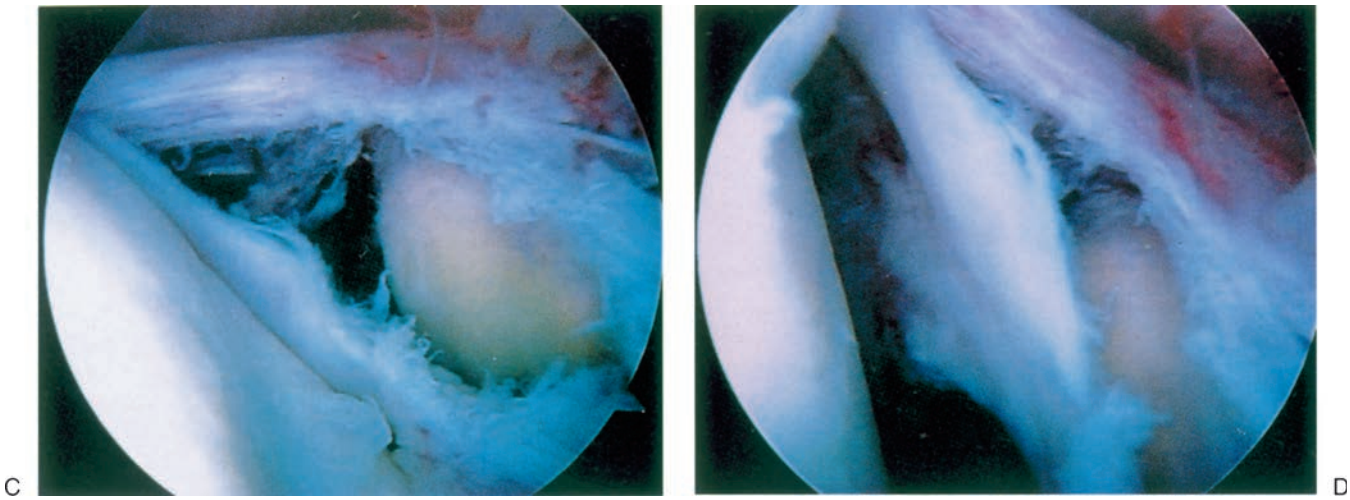

Figure 10-8 (*continued*)

Snyder and coworkers[85] described lesions of the biceps origin and the superior glenoid labrum, which they termed SLAP lesions as an acronym for superior labrum anterior and posterior. They postulated that these lesions were the result of superior subluxation of the joint. There may be a history of minimal trauma in older individuals, for there appears to be a propensity for degenerative lesions of the labrum and biceps tendon in this location.[77] These types of lesions were later ascribed to injury of the superior glenoid rim by the traction of the biceps tendon[10] as the elbow was decelerated during the follow-through phase of throwing.[1] SLAP lesions may also result from external rotation of the humeral head in the late cocking phase of throwing. Pannosian and coworkers[71] reported on the effects of type II SLAP lesions on glenohumeral rotation and translation in six cadaveric shoulders before and after repair. Glenohumeral translations with the application of 15N and 20N in the anterior, posterior, superior, and inferior directions were measured with the glenohumeral joint in 60 degrees abduction and 90 degrees external rotation. Data were recorded for intact shoulders, shoulders with arthroscopy portals, shoulders with arthroscopically created anterior type II SLAP lesions, shoulders with arthroscopically created anterior and posterior type II SLAP lesions, and shoulders having undergone arthroscopic repair. With the introduction of a SLAP lesion, significant increases in total range of motion, external rotation, internal rotation, anterior–posterior translation, and inferior translation were observed. Following arthroscopic repair, total range of motion, internal rotation, external rotation, and glenohumeral translations significantly decreased. These findings suggest that type II SLAP lesions cause significant glenohumeral instability and are similar to results found in other studies.[16,68] This study also found that type II SLAP lesions can be effectively treated with current arthroscopic techniques, which is a finding different from a prior study that found abnormal translations to be only partially restored after repair.[16] These differences in results may have been due to differences in repair techniques and warrant further investigation into the efficacy of repair of type II SLAP lesions.

Originally, five types of SLAP lesions were reported (Fig. 10-8). A simple degenerative fraying of the superior labrum is a type I lesion. The peripheral edge of the labrum remains firmly attached to the glenoid, and the biceps tendon attachment on the supraglenoid tubercle of the glenoid is intact. A type II lesion is a tear of the superior labrum and the biceps tendon attachment from the glenoid (Fig. 10-9). This lesion may be confused with an

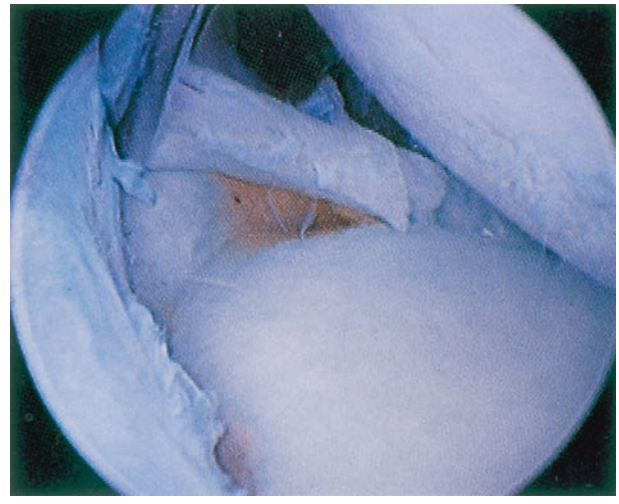

Figure 10-9 Arthroscopic view from the posterior portal of a joint that demonstrates a type II SLAP lesion.

anatomic variant where the labrum superiorly is meniscoid with a free edge.[20] Complete detachment of the labrum with exposure of bare bone from the superior glenoid neck is abnormal. Types I and II are most common, together accounting for over three-quarters of all SLAP lesions.[7,53] A bucket-handle tear of the labrum with preservation of the biceps anchor is a type III lesion (Fig. 10-10). In these cases the edge of the labrum is torn and can be displaced into the joint, whereas the peripheral portion of the labrum remains firmly attached to the glenoid. Additionally, the biceps tendon attachment is intact. When the biceps tendon is split with a portion remaining attached to the supraglenoid tubercle, the lesion is a type IV. This lesion is similar to the type III lesion except that a portion of the biceps tendon attachment is also involved. A type V lesion is any combination of these.

The effects of simulated type II SLAP lesions were studied by McMahon and coworkers[60] to determine if severity of the lesion affected glenohumeral joint translations. A robotic/universal force sensor (USF) testing system was used to simulate "load and shift tests" by applying an anterior or posterior load of 50N to each shoulder. The "apprehension test" for anterior instability was simulated by applying an anterior load of 50N with an external rotation torque of 3 Nm at 30 and 60 degrees of abduction. This loading protocol was repeated after creating two type II SLAP lesions of different severity. In the first, the superior labrum and the biceps anchor were subperiosteally elevated from the glenoid bone (SLAP-II-1), and in the second the biceps anchor was completely detached (SLAP-II-2). At 30 degrees of abduction, anterior translation of the vented joint from anterior loading significantly increased with the SLAP-II-2 lesion and compared to the SLAP-II-1 lesion. Increases in anterior translations at 60 degrees of abduction were not significantly different. Inferior translation also resulted from anterior loading. At 30 degrees of abduction, the inferior translation was greater for the type II SLAP lesions, regardless of severity, compared to the vented joint. No significant increases in anterior translation occurred in response to the combined loading condition. Glenohumeral translation was increased, regardless of severity, after simulation of type II SLAP lesions. During stabilizing surgical interventions, passive stabilizers that are injured in the type II SLAP lesion should be considered as well as dynamic activity in the tendon of the long head of the biceps brachii.

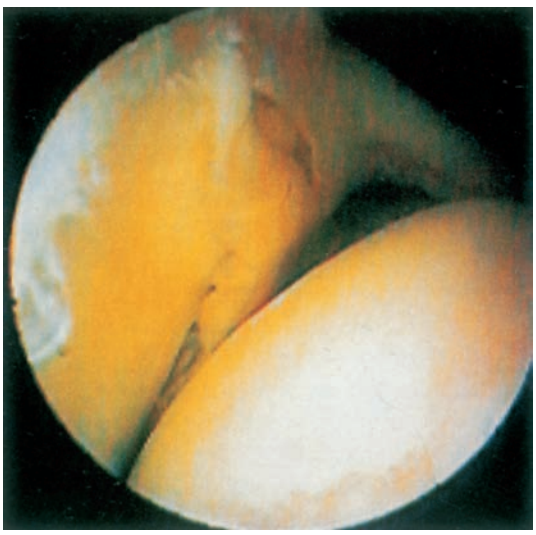

Figure 10-10 Arthroscopic view from the posterior portal of a joint that demonstrates a type III SLAP lesion.

Common Injuries in the Overhead Athlete

SLAP Lesion

Diagnosis of SLAP lesions is often difficult from the athlete's physical examination alone. The active compression test or O'Brien test[67] requires the standing patient to forward flex the affected arm to 90 degrees, with the elbow in full extension. The patient then adducts the arm 10 to 15 degrees medial to the sagittal plane of the body and internally rotates so the thumb points downward. The examiner, standing behind the patient, applies a downward force to the arm and the patient resists the downward motion. With the arm in the same position, the forearm is then fully supinated and the maneuver is repeated. The test is considered positive if pain is elicited during the first step and is then reduced or eliminated with the second step. Sometimes there is a painful click inside the shoulder. This test will also be positive if the patient has acromioclavicular joint pathology, but the pain will be localized on top of the shoulder. Jobe's relocation test[36] is also often positive in patients with a labral lesion. With the patient supine and the muscles relaxed, the shoulder is placed into the position of anterior apprehension by abducting to 90 degrees and then gently externally rotating the shoulder to the limit of motion (Fig. 10-11A). A gentle anterior-directed force may also be applied to the humeral head, resulting in posterosuperior shoulder pain. The relocation test is performed by reproducing this position and applying a posteriorly directed force to the humeral head (Fig. 10-11B) and the pain resolves.

Both the active compression and the relocation tests correlate with tears of the labrum.[35] However, problems remain in distinguishing a SLAP tear from other shoulder lesions. This results partly from SLAP lesions occurring concurrently with other shoulder pathology; isolated SLAP lesions are uncommon.[53] Also, the clinical findings associated with the different types of SLAP lesions overlap with the findings of other shoulder pathology. Overhead athletes are often able to compensate and compete successfully despite their shoulder injuries. This may result in a dilemma of treatment; is the shoulder pain coming from the SLAP lesion or from some other shoulder injury? Also, the underlying cause of SLAP lesions in overhead athletes

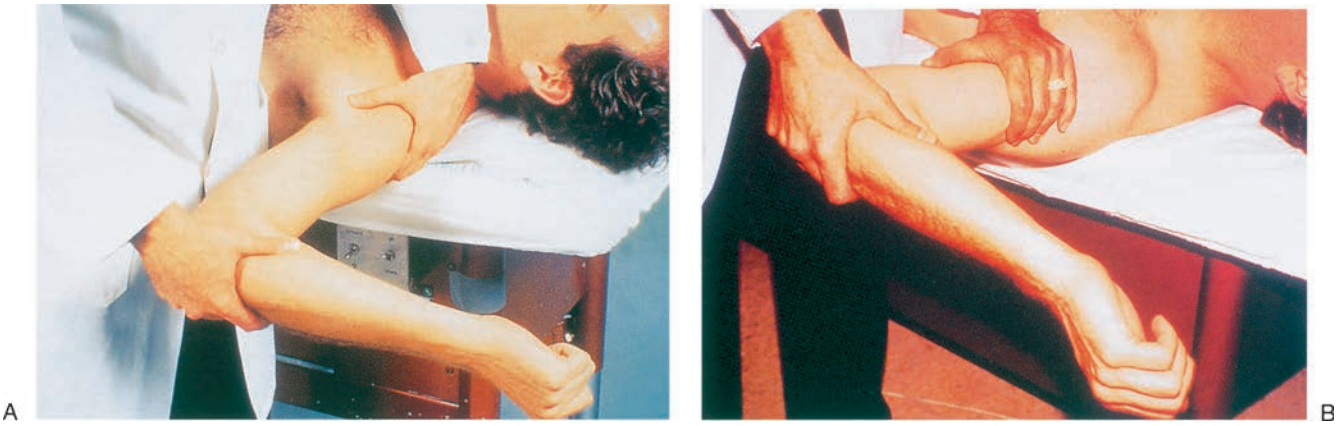

Figure 10-11 **(A)** With the patient supine and the muscles relaxed, the shoulder is placed into the position of apprehension by abducting to 90 degrees and then gently externally rotating the shoulder to the limit of motion. The overhead athlete with subtle instability feels pain as the tuberosity or the rotator cuff impinges against the posterior–superior glenoid rim. A gentle anterior directed force may be applied to the humeral head and the signs become more obvious. **(B)** The relocation test is performed by reproducing this position and applying a posteriorly directed force to the humeral head.

has been postulated to be a separate shoulder injury, namely posteroinferior capsular contracture.[17] Because the capsule changes in position with movement of the shoulder, the capsule is posteroinferior only with the arm at the side. In the shoulder of the overhead athlete and specifically the baseball pitcher, this capsular region ends up being anteroinferior in the cocking position of abduction and external rotation. When the contracture is severe, it forces the humeral head posterosuperior. The result is a SLAP lesion by a peel-back mechanism of humeral head impingement on the posterosuperior glenoid.

We recommend treatment of SLAP lesions based on mechanical symptoms and symptoms of instability. Fraying or tearing of the superior labrum, the biceps tendon, or both may result in mechanical symptoms of catching or clicking in the shoulder. Typical of types I and III SLAP lesions, these lesions may be débrided. Many individuals with a SLAP lesion, however, also have glenohumeral joint instability. In this case, the avulsed superior labrum and biceps tendon anchor should be fixed to glenoid bone. Types II and IV SLAP lesions are examples. Sometimes there is loose soft tissue that should be débrided as well. And, if there is minimal increase in joint translations, repair of the SLAP lesion alone is sufficient. With meaningful symptoms of instability, however, an anterior glenohumeral stabilization procedure should also be performed.

Instability Associated with Overuse Capsular Laxity

Anterior Instability

Repetitive shoulder motion in the overhead athlete may lead to tears of the anteroinferior labrum and anterior subluxation of the shoulder, leading to progressive instability. The glenoid labrum is a fibrocartilaginous rim around the glenoid fossa that deepens the socket and provides stability for the humeral head. It also is a connection for the surrounding capsuloligamentous structures. Glenoid labrum tears may also occur from acute trauma.

The load-and-shift test can also be used to confirm the diagnosis of anterior instability. For this test, the examiner applies a compressive load to the relaxed glenohumeral joint and an attempt is made to translate the humeral head. This test should be done in a number of different abduction positions simulating the cocking position of throwing and graded into one of three types. In a grade 1 load and shift the humeral head translates to the rim of the glenoid but not over it, which is a normal finding for testing in the anterior direction. For the test to be grade 2 the humeral head should translate over the rim of the glenoid and reduce spontaneously, whereas for the grade 3 test, the humeral head does not reduce and instead locks on the anterior glenoid rim. Only grade 2 and 3 load-and-shift tests are abnormal for testing in the anterior direction and in our experience are uncommonly found in overhead athletes with anterior instability associated with overuse capsular laxity.

Simple translational testing can also be helpful. The patient should be in a supine position and the shoulder muscles relaxed. Then the humeral head is grasped and an attempt made to translate it anteriorly. As with the load-and-shift test it is best to compare with the contralateral shoulder and examine the translation in several shoulder positions of abduction.

We always perform an examination under anesthesia on the shoulder of overhead athletes who we plan to treat surgically for glenohumeral joint instability, but we rarely are able to detect abnormalities; the instability

may be so subtle that it goes undetected. In our experience, valuable information can be gleaned from a variation of the simple translation testing of the shoulder when done with the athlete under anesthesia. With the shoulder abducted and in neutral rotation, the amount of anterior translation is assessed. With increasing external rotation of the shoulder, the amount of translation decreases and then becomes zero. At this point the position of the shoulder is noted. The test is then repeated with the contralateral shoulder. In the shoulder with instability, there is considerably more external rotation of the shoulder before anterior translation halts.

Arthroscopic findings, such as a classic Bankart lesion or a Hill-Sachs lesion, are unusual. Findings may include stretching of the anterior band of the inferior glenohumeral ligament, labral fraying, and mild labral separation. Chondral damage, seen as a defect in the posterior humeral head articular cartilage, may also be present. Rotator cuff damage is usually on the articular surface of the supraspinatus tendon. Conservative care emphasizes strengthening and conditioning of the rotator cuff and scapular muscles. Return to sport should be gradual, with careful attention to symptoms. Initially after return to overhead throwing, if pain occurs it should be mild in intensity and resolve within a few hours of cessation of the activity. Within a short time, pain should be absent during the overhead throwing activity. If the pain persists, the duration or vigor of the activity must be diminished. Most individuals will improve with such a program. If after 6 months of rehabilitation the symptoms continue, operative repair of the anterior capsuloligamentous structures is indicated. Isolated acromioplasty is not warranted and can lead to increased anterior instability.

Posterior Instability

Individuals with posterior instability of the glenohumeral joint can be divided into two distinct groups: those with traumatic posterior dislocation or subluxation and those with posterior subluxation from repetitive microtrauma. The traumatic posterior instability is usually the result of a fall on the outstretched upper extremity with a normal shoulder. Athletes very rarely suffer a posterior shoulder dislocation. Recurrent posterior subluxation is more typical in overhead athletes and results from chronic, insidious microtrauma to the posterior capsule, which subsequently becomes attenuated. For example, if a pitcher overthrows, does not warm up properly, or has poor mechanics that lead to a limited follow-through, then the posterior capsule can be injured. Repeated, daily injuries during participation in practice and games lead to chronic subluxation. However, chronic subluxation can also result from a single traumatic episode that results in subluxation with repeated use in overhead athletics. A football quarterback who is tackled and falls on his outstretched hand may feel a pulling sensation in the posterior shoulder. There may or may not be appreciation of joint instability. When the acute pain has subsided, there may be posterior shoulder pain with throwing, and velocity may be diminished.

It is common for overhead athletes with recurrent, posterior subluxation to complain of pain during the offending sporting activity. Pain may be absent at other times, and specific complaints of joint instability are unusual. On physical examination there is no asymmetry of the shoulders and range of motion is normal, as is strength. The pain of subluxation is sometimes felt when the shoulder is placed in the position of 90 degrees of forward flexion, horizontally adducted across the body, and internally rotated. Suspected posterior joint instability can be further evaluated by having the patient lie supine with the shoulder over the edge of the examination table, in the same position as described earlier, while a posterior force is applied to the shoulder. Although meaningful when positive, apprehension of posterior instability is rare. Accompanying symptoms of pain or apprehension are very important, for asymptomatic posterior subluxation may represent normal laxity and may not indicate pathologic instability. The athlete may be able to reproduce his or her symptoms by voluntarily subluxating the shoulder posteriorly with the shoulder in the forward-flexed and internally rotated position. These athletes are usually not psychologically disturbed and do not use their shoulders for secondary gain. They can be treated the same as the athlete who is unable to voluntarily subluxate the joint.

The load-and-shift test described previously in the assessment of anterior shoulder instability may also be helpful in assessing posterior instability. It is important to compare the contralateral shoulder because over half of shoulders in asymptomatic athletes have a grade 2 load and shift when tested in the posterior direction.[59]

Rehabilitation of the shoulder muscles, including the rotator cuff and the scapular muscles, is generally helpful in reducing symptoms of posterior instability. Strengthening of the infraspinatus, the teres minor, and the posterior deltoid muscles should be specifically addressed and biofeedback to rehabilitate the posterior shoulder muscles may have a place. When rehabilitation is maintained for at least 6 months, about 70% of individuals with posterior instability will improve symptomatically, and many athletes will be able to return to their sport.[40] When instability is recalcitrant to this rehabilitation program, we recommend a posterior capsulorrhaphy because the capsule is usually redundant. An osteochondral lesion (the reverse Hill-Sachs lesion) or a capsulolabral avulsion (the reverse Bankart lesion) is usually not seen. Generalized ligamentous laxity associated with posterior joint instability is a particular therapeutic problem best treated with an aggressive rehabilitation program; surgery is not recommended.

Internal Glenoid Impingement

The pitcher with internal impingement commonly complains of pain and decreased effectiveness. Pitch control is usually maintained, but velocity is diminished. Pain is located in the posterosuperior shoulder and is associated with throwing. After throwing is completed, the pain diminishes and then resolves after sufficient rest until throwing resumes.

The rotator cuff can be pinched against the posterosuperior glenoid rim with the shoulder abducted to 90 degrees and then maximally externally rotated.[70] With the glenohumeral joints fixed in this position, the greater tuberosity forces the rotator cuff against the glenoid rim (Fig. 10-12).[46,90] A subtle increase in anterior glenohumeral translation can also cause internal impingement.[21] Normally, both static and dynamic stabilizers act to prevent anterior instability, but in the apprehension position the normal distance from the rotator cuff to the posterosuperior rim of the glenoid is small so that little tolerance exists.

During the normal baseball pitch, the humerus stays in the plane of the scapula during the cocking and acceleration phases. Fatigue of the shoulder musculature from repetitive throwing may result in a lag of the humerus, posterior to the plane of the scapula during these phases; internal impingement results. There may also be damage to the static stabilizers, such as stretching of the anterior band of the inferior glenohumeral ligament. As the humeral head translates anteriorly with the shoulder in the apprehension position, more of the rotator cuff would contact the glenoid rim. This increased anterior translation then aggravates the internal impingement.

The shoulder relocation test is the best test for internal impingement. Reproducing the anterior apprehension position produces posterosuperior shoulder pain as the tuberosity or the rotator cuff impinges against the posterosuperior glenoid rim. Then, during the relocation test the distance between the rotator cuff and the posterosuperior rim of the glenoid increases from the posteriorly directed force being applied. In the athlete with anterior instability, this keeps the joint located, so the pain resolves. If continuous maximal external rotation is applied throughout the test, the shoulder can usually be externally rotated further.

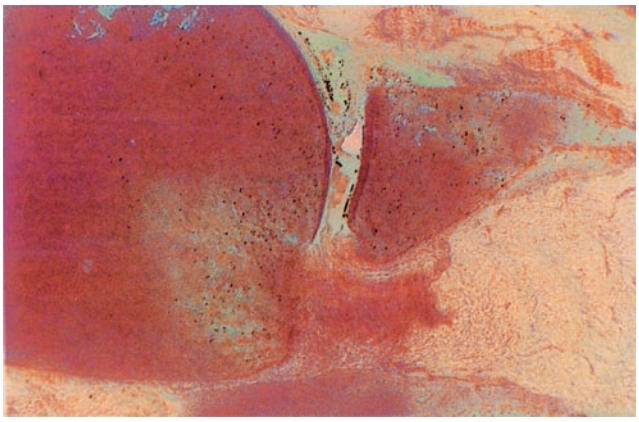

Figure 10-12 Cadaveric section of the glenohumeral joint with the shoulder put into the apprehension position of abduction and maximal external rotation; the rotator cuff pinches against the posterior superior glenoid rim.

When necessary, arthroscopic evaluation of the glenohumeral joint reveals characteristic lesions. There is fraying of the supraspinatus tendon in a location slightly more posterior to that seen with classic impingement. Fraying of the posterosuperior glenoid rim is also seen. At arthroscopy the intraarticular effects of the relocation test can be directly visualized. With the arthroscope in the posterior portal, the shoulder is put into the apprehension position of abduction and maximal external rotation, and the internal impingement can be seen as the rotator cuff abuts against the posterior superior glenoid rim (Fig. 10-13A). Then, with a posteriorly directed force applied to the humeral head, this is relieved (Fig. 10-13B). This can also be visualized with magnetic resonance imaging (MRI) (Fig. 10-14).

In the normal glenohumeral joint, the humeral head translates slightly posteriorly when the shoulder is abducted and externally rotated.[39] This normal posterior translation helps to minimize abnormal contact of the rotator cuff on the posterosuperior glenoid. Individuals with anterior joint instability, by contrast, did not demonstrate movement in the posterior direction, and in some cases the humeral head translated anteriorly. This abnormal translation increases internal glenoid impingement. During the first part of the relocation test in the joint with instability, the anterior translation of the humeral head results in rotator cuff tendon compression between the greater tuberosity and the posterosuperior glenoid rim. The application of the posteriorly directed humeral force then relocates the humeral head to its normal posterior position and the rotator cuff tendon does not contact the glenoid rim.[21]

Because internal impingement is usually a secondary problem, treatment must be directed toward the underlying causes, which are excessive humeral angulation, subtle anterior glenohumeral joint instability, and fatigue. Muscular development of the dynamic restraints and correction of problems with the throwing mechanics are indicated. Physical therapy aimed at strengthening and conditioning the shoulder muscles, including the rotator cuff, deltoid, and scapular muscles, is instituted. It is important to include the scapular muscles in the rehabilitation efforts to restore the normal scapulothoracic rhythm. Both concentric and eccentric strengthening and conditioning exercises are included. Although posterior capsular stretching may also be beneficial, care is taken not to stretch the static restraints to anterior translation. Additionally, cardiovascular and general muscular conditioning is incorporated in the rehabilitation program to minimize fatigue. Generally there will be meaningful improvement

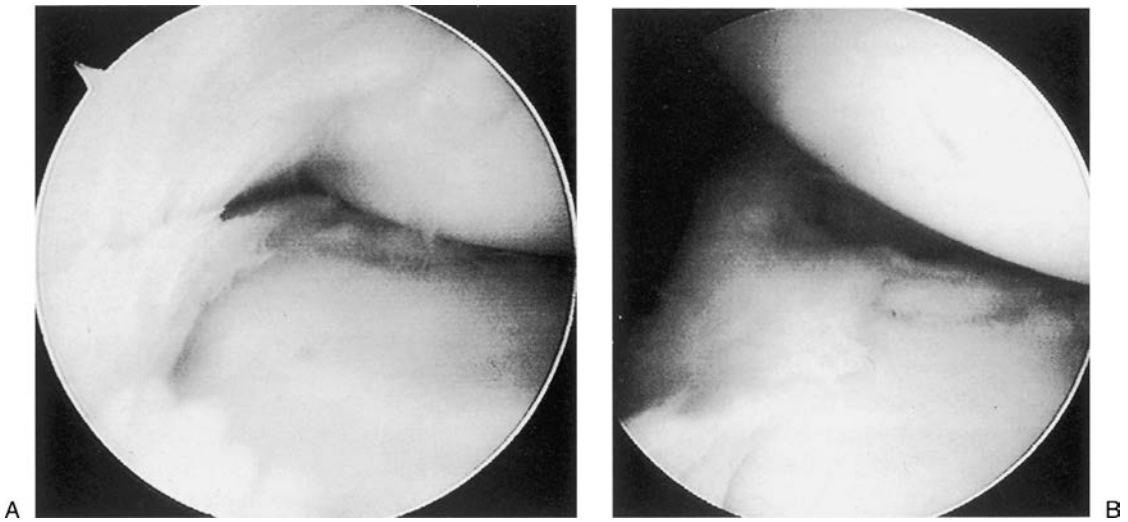

Figure 10-13 **(A)** View of the relocation test from inside the joint demonstrating internal impingement. With the arthroscope in the posterior portal, the shoulder is put into the position of abduction and maximal external rotation. The rotator cuff pinches against the posterior superior glenoid rim. **(B)** With a posteriorly directed force applied to it, the humeral head returns to a normal position, and the greater tuberosity is posterior to the glenoid rim.

in symptoms within 3 months, and throwing can resume with return to the prior level of competition after 6 months of treatment. Most overhead athletes (greater than 80%) can be successfully treated without surgery.

When anterior translation is markedly increased, these efforts may prove to be insufficient to allow the overhead athlete to return to pain-free activities. If the individual is intent on returning to overhead athletics, surgery is then indicated. In our experience, arthroscopic débridement of the rotator cuff and posterior superior glenoid fraying is unsuccessful if there is meaningful anterior joint instability. Only those individuals who would have responded successfully to the rehabilitation program have successful results with débridement alone. If there is meaningful anterior instability, operative repair of the anterior capsuloligamentous structures is required. Rehabilitation is then instituted, with expectation to return to the prior level of competition after 12 months.

Bennett Lesion

The Bennett lesion is an ossification or calcification of the posterior inferior glenoid found in overhead athletes, most commonly baseball players. Generally, the overhead athlete complains of pain while throwing and physical examination reveals tenderness in the posterior inferior glenoid region.

In 1941, Bennett[9] described a posteroinferior glenoid lesion, similar to an arthritic osteophyte, that he thought

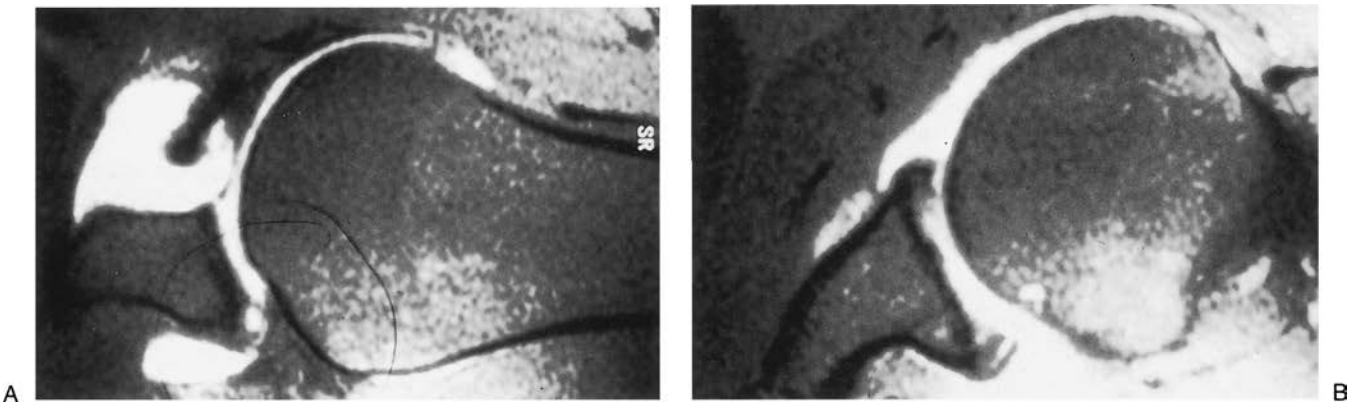

Figure 10-14 **(A)** Magnetic resonance imaging (MRI) of a shoulder positioned in the apprehension position demonstrating internal glenoid impingement. **(B)** MRI of a normal shoulder positioned in the apprehension position. The rotator cuff does not impinge on the posterior superior glenoid rim.

was the result of a traction injury of the long head of the triceps brachii. Pain accompanied the lesion. This was thought to be because of proximity to the axillary nerve and subsequent irritation. Radiographic analysis of the lesion necessitated direction of the x-ray beam 5 degrees cephalad, with the shoulder abducted and externally rotated. Initially, treatment of the Bennett lesion was resection from a posterior approach, but later Bennett stated that this was not necessary.[8] Ferrari and coworkers[26] used computed tomography–arthrography in seven elite pitchers to demonstrate that the Bennett lesion was extraarticular. Six had posterior labral tears. The Bennett lesion was described as a posteroinferior ossification associated with posterior labral injury.[26] Treatment included a period of rest followed by a rehabilitation program and treatment with nonsteroidal antiinflammatory drugs for 3 to 4 months. If this failed, arthroscopic débridement of associated labral lesions may be indicated.

Suprascapular Nerve Injury

Chronic neurologic injuries about the shoulder can present in overhead athletes of all ages. The neurologic examination should include complete sensory evaluation, testing of reflex arcs, observation for atrophy, and strength testing.

Cervical spine pain can be confused with shoulder pathology. These athletes may initially complain of shoulder pain, but neck pain is usually present on careful questioning. Physical examination and cervical spine radiographs enable correct localization of the pathology. A herniated nucleus pulposus can cause nerve root compression in the neck, with pain radiating down the arm in the distribution of the affected nerve.

The suprascapular nerve originates from the upper trunk of the brachial plexus to innervate the supraspinatus and infraspinatus muscles. Along its path, there are two areas of the scapula where it can be tethered and compressed: the suprascapular notch and the neck of the spine (spinoglenoid notch). Compression results in pain or weakness. Symptoms may preclude pitching more than one or two innings. In the chronic situation, the correct diagnosis can be elusive, because the disease has an insidious onset and vague symptoms. Muscle pain and diminished endurance are the important presenting symptoms. There is weakness of external rotation and sometimes of abduction, depending on the location of the compression. Either the infraspinatus is involved alone or both shoulder muscles are, depending on the location of the compression. Physical examination demonstrates muscle atrophy. The trapezius overlies the supraspinatus muscle, but atrophy of this muscle can be appreciated as a depression over the supraspinatus fossa of the shoulder. Atrophy of the infraspinatus is easy to appreciate as a depression over the lower half of the scapula. EMG can be used to confirm the diagnosis. Suprascapular nerve palsy can be confused with rotator cuff tears, which present with pain in a similar area and weakness of the same musculature. Surgical exploration and decompression of the nerve generally give good results when the nerve is compressed at the suprascapular notch. In this case, the supraspinatus and infraspinatus muscles are both involved. However, if atrophy and EMG changes are confined to the infraspinatus muscle, the nerve is injured at the spinoglenoid notch. A ganglion cyst can be responsible, and MRI is helpful in determining its location.[41] When present, resection of the ganglion cyst is effective treatment. However, when the injury is a result of traction, nonsurgical treatment is recommended, because surgical decompression has not always been successful. The effects of chronic compression may not be reversible in either event. If the infraspinatus is not completely deinnervated, the authors have had good results with maximizing the residual muscle function with a daily exercise program. Because this muscle seldom contracts more than 30% to 40% of its maximum effort, the return to the prior level of pitching is sometimes possible. A meticulous history and physical examination for proper diagnosis early in the disease course yields the best chance for success.

Quadrilateral Space Syndrome

Bounded by the humerus, teres major, long head of the triceps brachii, and the inferior rotator cuff (the subscapularis anteriorly and the teres minor posteriorly), the quadrilateral space is crossed by the axillary nerve and the posterior humeral circumflex artery. Cahill and Palmer[19] described the cause of pain in this location as compression of the axillary nerve by fibrous bands in the quadrilateral space. Symptoms occur when the athlete abducts and externally rotates the shoulder as in the cocking stage in throwing. There is tenderness over the teres minor muscle, and symptoms can be reproduced by placing the shoulder into this position. The neurologic examination is usually normal, as are EMG studies. Arteriography of the subclavian artery, which enables visualization of the posterior humeral circumflex artery as it passes through the quadrilateral space, is helpful in making the correct diagnosis. With the arm at the side, the artery is patent, but with abduction and external rotation of the shoulder, the artery occludes. It is necessary to perform comparative studies of the contralateral shoulder. A test is positive only if the symptomatic side occludes and the normal side does not. In the athlete with this condition, a short period of rest and cortisone injections into the space are often helpful. If symptoms persist, operative decompression of the quadrilateral space is indicated. This can be performed through a posterior approach, inferior to the deltoid. The tendon of the teres minor can be incised to decompress the axillary nerve, but in our experience, the long head of the triceps is often the offending structure. Results have been favorable.

Rotator Cuff Injuries

Neer,[65] among others, described impingement syndrome as compromise of the space between the humeral head and the coracoacromial arch. In the classic case, the coracoacromial ligament and the anterior inferior aspect of the acromion are compressed against the bursal side of the rotator cuff during forward flexion of the shoulder. Anterior acromial spurs may be present. Unlike acute tears, chronic rotator cuff tears often present insidiously, with slow progression from subacromial bursitis to rotator cuff tendonitis and eventual tendon tear. Over time, small tears may progress to larger, more severe tears. Active range of shoulder motion is limited, and if the tear is severe, there will be atrophy of the shoulder muscles. Manual muscle testing demonstrates weakness. The Neer impingement sign is positive and the pain resolves with subacromial injection of lidocaine. Differentiating severe rotator cuff tendonitis from partial or small full-thickness chronic rotator cuff tears may be a difficult task.

Overhead-throwing athletes with rotator cuff injuries from classic impingement syndrome are generally an older population, more than 45 years of age. Tears are most common at the humeral insertion site of the supraspinatus tendon, where stress is greatest with the joint in abduction. Tears may involve either the partial or full thickness of the tendon. Tears are most common in the anteriormost portion of the supraspinatus tendon. Small tears are located here and more severe tears, even when the entire supraspinatus and portions of the infraspinatus tendons are involved, have a margin of the tear at the anteriormost supraspinatus tendon. The size may be small (less than 1 cm), medium (1 to 3 cm), large (3 to 5 cm), or massive (greater than 5 cm). Chronic rotator cuff tears may result partly from degeneration within the rotator cuff tendon. Poor vascularity and repetitive activity, especially in the athlete with a restricted subacromial space, may be contributing factors. A minor traumatic event may also cause a full-thickness tear in an athlete with mild or moderate tendon degeneration.

If the tear is small, a prolonged period of rest, lasting 4 to 9 months, may relieve symptoms. Range-of-motion exercises are also recommended, unless they cause significant discomfort. If this fails to control the symptoms, surgical repair of the tear is recommended. The thin degenerated tissue of a chronic rotator cuff tear makes surgical repair more difficult than repair of an acute tear. Surgical decompression of the subacromial space to remove spurs should also be performed.

Rehabilitation lasts from 6 months to a year, with gradual exercise progression needed to restore normal, or near-normal, function and strength. This varies with the tear size repaired and type of surgery performed. Typically, immediately after the procedure, passive motion and isometric strengthening exercises start, along with elbow-, hand-, and grip-strengthening exercises. At 6 weeks, the athlete may be able to begin low-intensity active strengthening exercises against gravity. The goals are to bring the athlete to normal strength with a functional, pain-free range of motion.

While the lesion location and size are helpful in describing the rotator cuff tear, symptoms do not correlate with these factors alone. Both epidemiologic and imaging studies indicate a high incidence of partial-thickness rotator cuff tears at younger ages and a high incidence of full-thickness rotator cuff tears at older ages. Small full-thickness rotator cuff tears may be asymptomatic as long as the force couple of the anterior and posterior rotator cuff is preserved. Instead, a number of other factors influence the severity of symptoms, including acute/chronic nature of the injury, patient age, activity level, humeral head superior migration, shoulder muscle strength, arthritis, pain tolerance, and workman's compensation.

A partial articular-sided tendon avulsion is much more common than a bursal side tear of the rotator cuff. As with other rotator cuff injuries, symptoms may resolve with appropriate physical therapy and analgesics. Yet, some individuals with a partial-thickness tear have persistent or recurrent symptoms. If a conservative program of exercises and gradual return to activity does not lead to steady improvement, then further diagnostic evaluation with ultrasonography, MRI, or arthroscopy may be helpful. Arthroscopic débridement of the abnormal cuff may promote healing in athletes with partial-thickness posttraumatic tears. Following débridement, immediate resumption of range-of-motion and muscle-strengthening exercises begins. Typically, it requires 6 to 12 months for a throwing athlete to return to athletics following arthroscopic débridement of a partial-thickness rotator cuff tear.

In those younger than 35 years of age, when symptoms of impingement syndrome are present, it is almost exclusively associated with anterior glenohumeral instability. Classic impingement syndrome is unusual in these young overhead-throwing athletes. They have positive impingement signs and a positive relocation test. Tears are found in the posteriormost portion of the supraspinatus tendon and most commonly are partial-thickness, articular-sided tears. The cause of these rotator cuff injuries may be internal impingement or the loads of prolonged, repetitive overhead throwing. In these young athletes, treatment of the instability results in resolution of the rotator cuff injury.

Vascular Problems

Shoulder and arm pain in the overhead athlete does not often elicit a diagnosis of vascular injury, but misdiagnosis can result in disastrous outcomes, such as arterial thrombosis and embolization. Identifying vascular compression injuries is difficult in the overhead athlete, and these injuries are relatively uncommon. Unless vascular compromise is recognized as a possible source of pain, misdiagnosis is likely.

Early fatigue of the upper extremity in a well-conditioned athlete is a frequent initial complaint. In baseball pitchers, decreased endurance and measurable loss of pitch velocity after three innings are common findings. Forearm pain, throwing arm heaviness, and hand coldness are sometimes present. Loss of pitching control is not a symptom. Physical examination may reveal a diminished pulse or a loud bruit. Doppler ultrasonography and duplex scanning indicate that subclavian artery compression by the scalene muscles is the most likely cause in symptomatic athletes.[58,66] When the shoulder is positioned in abduction and external rotation, this finding is common in asymptomatic individuals as well as overhead athletes[80] and, therefore, is not specific. Physical examination findings must correlate. Sometimes symptoms are the result of subclavian artery aneurysm with thrombosis, and in these cases, embolization to the hand and severe ischemia may occur.[66] Compression of the axillary artery,[66] the posterior humeral circumflex artery,[43] the suprascapular artery, or the subscapular artery[58] can also result in localized symptoms. Treatment includes avoidance of all exacerbating activities until symptoms subside and a carefully supervised program of muscle strengthening for the entire shoulder girdle. Postural training may also be helpful, but this requires strengthening and conditioning of the trunk muscles. If conservative measures fail, the involved areas can be surgically decompressed with excellent results.[66]

MANAGEMENT PRINCIPLES IN THE COMPETITIVE ATHLETE

Management of the overhead athlete begins with a meticulous history and physical examination. Loss of velocity, accuracy, and distance usually alert the overhead athlete to injury more serious than usual aches and pains. The effect of the injury on the patient's activities of daily living may provide as many clues to the diagnosis as does the effect of the injury on sports performance. Pain is a subjective symptom, but careful assessment can provide insight into disease pathology. The duration, anatomic location, and character of the pain should be specifically assessed. In addition, the presence of night pain and analgesic requirements should be considered. The temporal relation to sports activity and the postural relation to arm motion should be obtained from the patient with shoulder pain. For example, a pitcher who complains of shoulder pain during the cocking stage of the throwing motion usually has anterior shoulder instability. The pitcher who has pain during the follow-through stage may have posterior shoulder instability. The onset of night pain, especially when lying on the affected side, indicates rotator cuff injury, as does pain with overhead activities.

The physical examination of the shoulder consists of several phases, including visual inspection, palpation, range-of-motion testing, strength testing, neurovascular assessment, and general physical evaluation. Visual inspection includes examining the skin and the contour of the entire shoulder girdle. Special attention should be given to areas of swelling or muscle atrophy. Side-to-side differences should be recorded, with knowledge that there are some normal changes that occur in the throwing arm. For example, the musculature is usually hypertrophied and the scapula is often displaced slightly inferiorly in the throwing arm. Palpation should be performed from the neck to the fingers on all sides of the upper extremities. Motion of all four joints (sternoclavicular, acromioclavicular, scapulothoracic, and glenohumeral) is essential to normal shoulder kinematics. This can be assessed both actively and passively. Differences between active and passive motion can be the result of a deficiency, such as a rotator cuff tear. Motion should always be compared with the contralateral shoulder. Brown and coworkers[15] found that Major League pitchers have different ranges of motion between the shoulders. In the pitching arm, with the shoulder in abduction, there is 11 degrees less extension, 15 degrees less internal rotation, and 9 degrees more external rotation. Therefore, comparison with the contralateral arm should be done with this variance in mind.

MRI, computed tomography (CT), and other imaging studies can be used to reinforce the physical findings in the difficult case. They can also be helpful in a few special circumstances. If there is suspicion of rotator cuff abnormality, the MRI is useful in delineating the extent of injury. An MRI can also be used when the radiographs are not sufficient in identifying humeral and glenoid abnormalities. It is also helpful in the athlete with a strong suspicion of instability, but without a strong indication for surgery unless a specific labral lesion can be identified. The use of an intraarticular contrast agent is generally recommended. Iodine injection into the glenohumeral joint before CT or gadolinium or saline injection into the glenohumeral joint before MRI is helpful to outline the labrum.

Nonoperative and Operative Decision Making

Treatment of the overhead athlete may differ depending on timing of the injury to the athlete's sporting season. Factors to take into consideration include the athlete's ability to effectively participate, propensity for further injury, number of years for play remaining, and the effectiveness of operative and nonoperative treatments. In general, operative treatments require a longer period of rehabilitation than nonoperative treatments. For athletes who play professionally, consideration of the value for continued participation may also be important.

If the athlete believes that effective participation is possible and the caregiver has determined that propensity for

further injury is small, then overhead throwing in a controlled setting is indicated. In the case of a baseball pitcher, throwing pitches under the supervision of the coach and the athletic trainer or physical therapist may be best before the athlete returns to participation. The athlete's condition is carefully monitored during play. Recurrence of the injury necessitates further evaluation, and worsening of the injury precludes further participation. The optimal time for treating injuries in the overhead athlete is at the conclusion of the season. This leaves the athlete with the longest length of time for recovery.

In Season

As is true throughout the year, shoulder injuries suffered during the season necessitate a thorough evaluation that yields the correct diagnosis. Then, initial treatment for most injuries is a period of rest and nonsteroidal antiinflammatory drugs, followed by supervised rehabilitation. If 6 weeks of this treatment is ineffective, a magnetic resonance arthrogram may be helpful in determining the severity of the injury. If the athlete returns to play, careful supervision of performance is needed to minimize the risk of the injury worsening. In a recent study of return to play, most athletes were able to return to their sport and complete their seasons after an episode of anterior shoulder instability.[18] However, many of these were not overhead athletes and over one-third had at least one recurrence of shoulder instability before the end of the season. If a surgical procedure is able to improve the athlete's condition, it may be performed at the end of the season to minimize the time lost from participation.

Off Season

Treatment of shoulder injuries in the overhead athlete that occur during the off season require consideration of the additional factor of the length of rehabilitation. If treatment of the injury requires many months of rehabilitation and injury occurs near the beginning of the season, return to play at the start of the season may not be possible. Also, operative treatment may not be best if it requires a longer period of rehabilitation than nonoperative treatments and the previous considerations have been discussed with the athlete.

REHABILITATION OF THE ATHLETE AND RETURN TO SPORTS

Treatment of the overhead athlete requires knowledge of what is normal and what is abnormal in these athletes. For example, external rotation of the abducted shoulder is greater than normal in baseball players, while internal rotation is diminished. It is erroneous to consider the "excessive" external rotation as pathologic as the athlete has no symptoms and competes successfully. Likewise, it is important to understand which shoulder structures are at risk during the overhead activity. The underlying cause of most baseball players' shoulder problems is instability of the glenohumeral joint. Treating associated symptoms of rotator cuff injury does not prevent further problems. Wise practitioners always ask themselves, "Is this the core of the problem or simply a secondary effect?"[51] The earlier the core of the problem is diagnosed, the better the chances are for a quick recovery. An astute practitioner who has knowledge of the athlete's sport-specific mechanics has the best opportunity for success. Early in the course of the problem, complaints may be vague; the athlete may recognize that it takes longer to warm up or "get in the groove." If the injury progresses, performance diminishes. Unfortunately, many athletes continue to participate and injury occurs to shoulder structures. Recovery of the secondary injury may be necessary before the underlying core of the problem can be diagnosed.

The goals in rehabilitation of the overhead athlete's shoulder are to restore flexibility, reestablish joint stability, strengthen shoulder muscles, and restore proprioception. This must be done without exacerbating injury. Ice is valuable in diminishing pain and inflammation and other modalities such as ultrasound and electrical stimulation may also be helpful. It is usually best for the overhead athlete to rest for a period of time to allow for healing. The length of rest is dictated by the severity of the injury. For mild injuries, a week of rest is usually sufficient, but for moderate to severe injuries, 4 to 12 weeks of rest may be best. When the athlete returns to overhead throwing, its length and intensity is modified to a pain-free level.

Also important is restoration of shoulder motion, particularly internal rotation and horizontal adduction. There may be meaningful loss of internal rotation of the abducted shoulder in overhead athletes, particularly in baseball pitchers. This often results from posterior capsular contracture, and specific stretches and flexibility exercises that passively stretch the abducted shoulder in internal rotation are best. These stretches are best done with the scapula stabilized. The humerus of the abducted shoulder is stretched in internal rotation with the athlete positioned on the scapula of the affected side. Athletes may also be instructed to do posterior capsular stretching, known as a "sleeper stretch," by themselves. The athlete lies on a table on the effected side with the arm in front. Rolling toward the arm places the shoulder into horizontal adduction. Then, with the elbow bent to 90 degrees, the athlete pushes the forearm toward the table, stretching the shoulder in internal rotation (Fig. 10-15).

The shoulder muscles must be strengthened to overcome weakness, which may have preceded and, in part, resulted in the shoulder injury, which may have been from shoulder pain, and which may have resulted from the

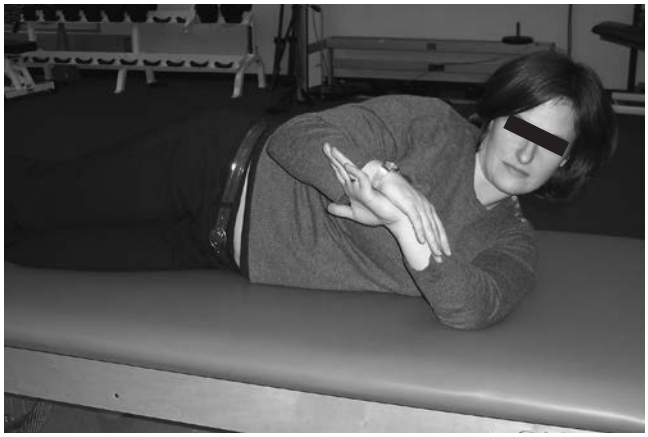

Figure 10-15 An athlete doing a sleeper stretch.

enforced rest that allowed for healing. Because there are numerous muscles essential to the normal shoulder, rehabilitation can be complex. An easy way to remember the strengthening concepts for the overhead athlete's shoulder is to think of "E^{14} and the 4 Ps":[51] Effective, efficient exercises focus on the glenohumeral "protectors" (i.e., the rotator cuff muscles), the scapulothoracic "pivotors" (i.e., the scapular rotator muscles), the humeral "positioners" (i.e., the three heads of the deltoid muscle), and the shoulder "power drivers" (i.e., the pectoralis major and the latissimus dorsi). The glenohumeral protectors and the scapulothoracic pivotors are strengthened first to allow for restoration of glenohumeral stability. The humeral positioners are strengthened next because they are important for normal synchronous motions of the shoulder. The power drivers are strengthened last to prevent overpowering the other three groups when the others are weakened.[51]

If the injured athlete is extremely sore or painful, it is best to start with submaximal isometric exercises; conversely, if there is only mild soreness, then lightweight isotonic exercises can be started.[94] Exercises to restore joint stability are aimed at the force couples of the shoulder. Reciprocal isometric muscle contractions are begun for the internal and external rotator muscles of the shoulders, as are agonist and antagonist co-contraction exercises for these and other muscles. Exercise of the rotator cuff muscles is initiated with the athlete lying on the side. To exercise the external rotators, the athlete lies on the opposite side with a small pillow between the thorax and the arm to be exercised. The arm is rotated away from the body and then back to the starting position and repeated. Progressively larger weight is added to make these exercises more challenging. Prone rowing into external rotation can also be used as it has been shown to elicit high EMG activity of the posterior rotator cuff muscles.[27] To exercise the internal rotators, the athlete lies on the affected side with the arm slightly in front of the thorax and is rotated toward the body. Exercises of prone horizontal abduction with external rotation and supine horizontal adduction with internal rotation are also added as strengthening continues. It is especially important to strengthen the teres minor muscle by externally rotating the shoulder in positions of abduction. This is done with the shoulder in about 70 degrees of abduction. Excessive external rotation and excessive horizontal abduction are blocked to minimize symptoms of anterior shoulder instability.

The supraspinatus muscle is isolated when the shoulder is elevated in the plane of the scapula. This motion, termed "scaption," was reported by Jobe and Moynes[49] to result in high levels of EMG activity in the supraspinatus muscle with the "empty can" exercise of internal rotation; this places the hand in the thumb-down position. Highest EMG activity in the supraspinatus muscle results when the shoulder is elevated from 90 to 120 degrees, but this may exacerbate rotator cuff tendonitis if it is present.[87] The "full can" exercise may be helpful to exercise the supraspinatus muscle in athletes who have rotator cuff tendonitis. With the patient lying prone and with the arm abducted to 100 degrees and fully externally rotated, high EMG activity is produced in the supraspinatus muscle,[13] but this position may exacerbate anterior shoulder instability if it is present. It is best that the therapist personalize exercise of the supraspinatus muscle to minimize the athlete's symptoms.

Eccentric exercises are then begun under the supervision of a therapist. The athlete also progresses to isokinetic exercise of the rotator cuff muscles. This begins at slow speed and submaximal effort and progresses to high speed and large effort.

Full, active scapular motion is very important to the overhead athlete, as is strengthening of the scapular pivotors. The muscles responsible for this motion are the trapezius, levator scapulae, rhomboids, serratus anterior, and pectoralis minor. These muscles act to position the scapula for maximum glenohumeral joint stability while minimizing impingement syndrome. For example, scapular upward rotation entails three complementary actions: upward rotation, retraction, and an anterolateral force from the inferior angle of the scapula.[42] The upward rotation results from the upper and lower portions of the trapezius and the serratus anterior.[51] The retraction results from the middle portion of the trapezius and the rhomboids and the anterolateral force by the serratus anterior. Muscles from all three boarders of the scapula function.[51] Rowing and horizontal abduction exercises exercise all portions of the trapezius, levator scapulae, and rhomboid muscles. Flexion and scaption exercises are also valuable scapular muscle strengthening. Wall pushups and the shoulder shrug are good for initiating scapular muscle strengthening and the pushup plus can be used later in the rehabilitation program. The "plus" exercise during the pushup is performed by emphasizing scapular protraction at the "top"

Figure 10-16 The pushup-plus exercise emphasizes scapular protraction at the top of the pushup.

of the pushup and has been demonstrated with EMG to be the very beneficial (Fig. 10-16).[51] The athlete must be careful to lower the chest only as far as the shoulder during the pushup-plus exercise to avoid putting the shoulder in the position of apprehension for anterior instability. It is best for the therapist to start the athlete on the knees and forearms when the athlete begins to do these exercises on the floor to prevent excessive scapular muscle effort early in the rehabilitation program.

Because serratus anterior muscle activity is often high during overhead activity,[73,74,83] it is important to introduce endurance exercises for this muscle. An upper-extremity ergometer can be used with the height of the hand pedals varied to ensure optimal training of all portions of this broad-based muscle.[51] Specific exercise drills may be used to enhance neuromuscular control of the scapulothoracic articulation (Fig. 10-17).[93]

Strengthening of the humeral positioners and the power drivers ensues only after the protectors and pivotors are strong. Scaption, forward flexion, and prone horizontal abduction are good exercises for the humeral positioners. When adding weights to these exercises, the therapist must note if the athlete substitutes muscle weakness by shrugging the shoulder. Additional exercises include rowing and the military press. Strengthening of the pectoralis major and the latissimus dorsi muscles, the power drivers, are the last shoulder muscles to be incorporated into the rehabilitation program. Actually, most power during the overhead throw comes up the body from the legs to the trunk to the

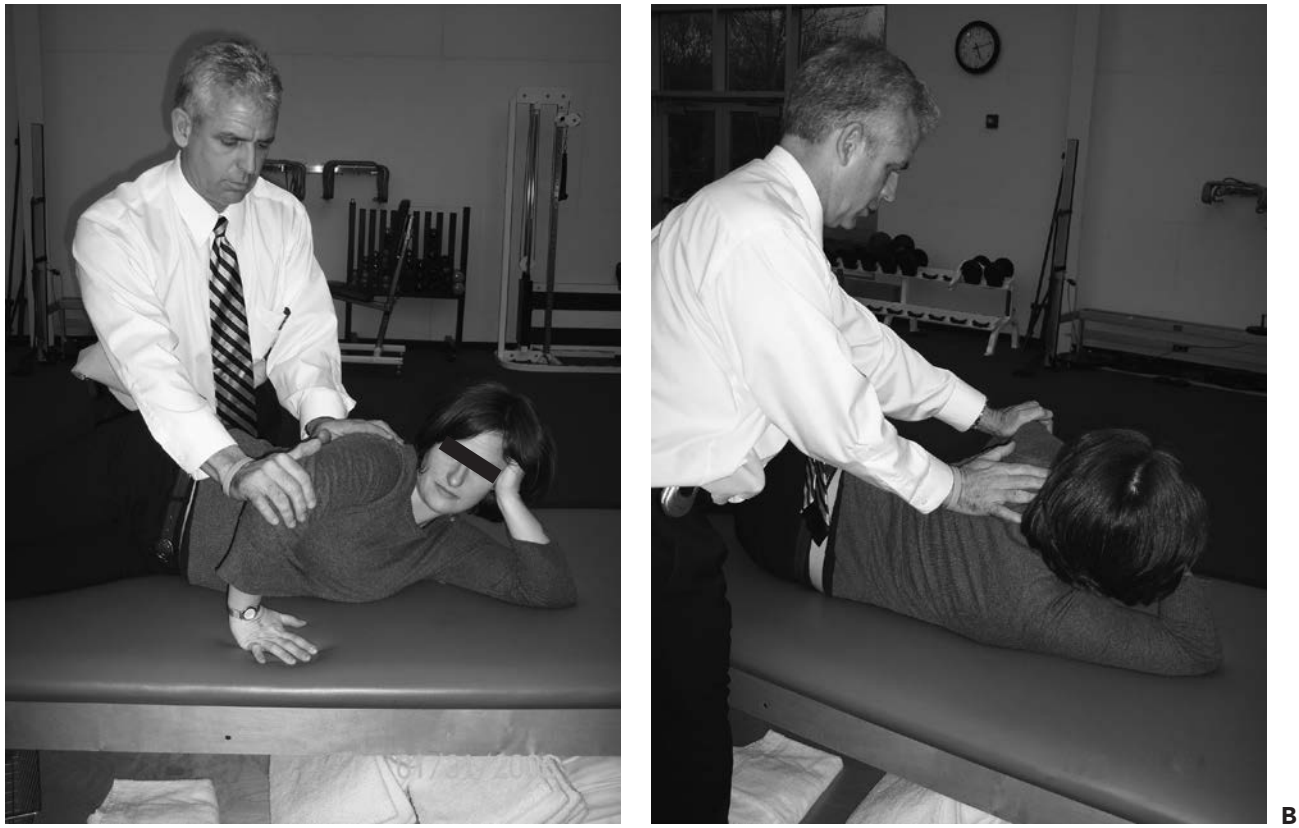

Figure 10-17 Neuromuscular control exercise drill for the scapular muscles: The athlete lies on her side with the hand placed on the table **(A)** and the clinician applies manual resistance to resist scapular movements (such as protraction and retraction) **(B).** The athlete is instructed to perform slow and controlled movements.

arm and out to the projectile object.[92] The pectoralis major and the latissimus dorsi muscles are the only muscles of the upper extremity to demonstrate a positive correlation between peak torque, measured in isokinetic testing, and pitching velocity.[4] The seated press-up, the bench press, and the pull-down are all good exercises to strengthen these muscles.

The overhead athlete should also perform core strengthening exercises for the abdomen and lower back musculature. Lower-extremity strengthening is also initiated with a running program, including jogging and sprinting. Fatigue can diminish performance. Once the overhead athlete is fatigued, shoulder external rotation decreases and ball velocity diminishes, as does lead knee flexion and shoulder adduction torque. Voight and coworkers[89] documented a relationship between muscle fatigue and diminished proprioception. Lyman and coworkers[57] reported that the predisposing factor that correlated to the highest percentage of shoulder injuries in Little League pitchers was complaints of muscle fatigue while pitching. Thus, endurance exercises are critical for the overhead athlete. Specific endurance exercise drills include wall dribbling with a ball, wall arm circles, and isotonic exercises using small weights with high repetition. Other techniques that may be beneficial to enhance endurance include throwing a ball weighted more or less than that of normal play. Also, during this phase the athlete may perform shadow throwing or mirror throwing; this mimics throwing mechanics without actively doing so. This allows the athlete to work on proper throwing mechanics.[93] Drills to enhance proprioception and neuromuscular control, such as pushups onto a ball, can also be done.

The last phase of the rehabilitation program is the return-to-throwing phase. A throwing program is initiated once the athlete can fulfill the criteria of (1) satisfactory clinical examination, (2) painless range of motion, (3) satisfactory isokinetic tests, and (4) appropriate rehabilitation progress. The throwing program is designed to gradually increase the quantity, distance, intensity, and type of activity needed for the gradual restoration of normal biomechanics.

We use baseball as an example, but the principles we describe apply to all overhead athletics. Overhead throwing is initiated from 30 to 45 ft and progresses to throwing from 60 ft. The athlete is instructed to use a crow-hop type of throwing mechanism and lob the ball with an arc for the prescribed distance. Flat ground, long-toss throwing is used before throwing off the mound to allow the athlete to gradually increase loads to the shoulder while using proper throwing mechanics. In addition, during this phase of rehabilitation, we routinely allow the position player to initiate a progressive batting program. We routinely use a program that progresses the athlete from swinging a light bat, to hitting a ball off a tee, to soft-toss hitting, to batting practice.

For baseball pitchers, we progress the long-toss program to 120 or 145 ft, whereas position players would progress to throwing from 180 ft. Once the pitcher has successfully completed throwing from 120 or 145 ft, he or she is instructed to throw 60 ft from the windup on level ground. Once this is accomplished, throwing from the mound is allowed. Position players continue to progress the long-toss program to 180 ft and then perform fielding drills from their specific position. While the athlete is performing the throwing program, the clinician should carefully monitor the overhead athlete's mechanics and throwing intensity. In addition, during this last phase of rehabilitation, the overhead athlete is instructed to continue all of the earlier exercises to maintain upper-extremity strength, power, and endurance. The athlete is also instructed to continue the stretching program, core exercise training, and lower-extremity strengthening activities. Lastly, the athlete is counseled on a year-round conditioning program. To prevent the effects of overtraining or throwing when poorly conditioned, it is critical to instruct the athlete on specific exercises to perform throughout the year.

SUMMARY

To successfully treat the overhead athlete, knowledge of the biomechanics of the sport and common shoulder pathology is necessary. There may be several pathologic lesions in the shoulder of an overhead athlete, so attention to symptoms and signs are essential to appropriate treatment. Appropriate imaging studies, examination while the patient is anesthetized, and arthroscopy may also aid the clinician in making the proper diagnosis. Rehabilitation of the shoulder muscles and appropriate surgical intervention optimize return to overhead athletics. Lastly, one should consider when the injury has occurred relative to the season of play to optimize the athlete's participation and performance.

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11 Diagnosis of Glenohumeral Instability

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INTRODUCTION

Our knowledge of glenohumeral instability has evolved over the years. The original description of a single "essential lesion" by Perthes[95] and Bankart[2] has expanded with the identification of several causes and mechanisms of instability. A clear understanding of the various types of instability and associated pathology is essential to the formulation of a proper diagnosis and treatment plan. Clinical evaluation through a detailed history, physical examination, radiographic imaging, and the appropriate use of other modalities (such as magnetic resonance imaging, examination under anesthesia, and diagnostic arthroscopy) will facilitate an optimal treatment strategy for most patients. This chapter presents an algorithmic approach to the evaluation of glenohumeral instability.

DEFINITION OF INSTABILITY

Consideration of the different types of glenohumeral instability requires the distinction between laxity and instability. *Laxity* is the normal, asymptomatic, and necessary passive translation of the humeral head on the glenoid that allows normal shoulder motion. Laxity may be affected by age[17] gender,[29,70] and congenital factors. It is also a trait that varies among individuals. Harryman et al. found considerable

variability in glenohumeral translation in normal subjects, with substantial humeral excursion in some asymptomatic normal shoulders.[37] Similarly, Hawkins et al. noted overlap in the amount of translation found during examination under anesthesia between symptomatic unstable and normal shoulders, affirming the normal variability in capsular laxity among individuals.[43] *Instability,* on the other hand, is a pathologic condition of excessive translation of the humeral head on the glenoid during shoulder motion that manifests in pain and clinical symptoms of subluxation or dislocation. Both conditions may exist independently, as patients with hyperlaxity may be asymptomatic or can exhibit pathologic instability. Moreover, hyperlaxity may be a risk factor for the development of shoulder disorders.[28,85]

CLASSIFICATION

The importance of a designated classification system for glenohumeral instability lies in the ability to develop optimal treatment strategies for a given set of pathologic findings. Some factors common to most currently accepted schemes include direction, etiology, frequency, degree, and volitional control.[27,28,31,35,44,55,59,102,114]

Prior to the description of more sophisticated shoulder classification systems, glenohumeral instability had been simply characterized by direction: anterior or posterior. Neer and Foster[76] expanded on that theme and described multidirectional instability as either anterior or posterior, in addition to an inferior instability component. The "sulcus sign" represented passive inferior subluxation of the humeral head on physical examination and was also the hallmark of multidirectional instability. However, as previously noted, the ability to passively subluxate the shoulder inferiorly only determines the degree of laxity in an individual, whereas the diagnosis of instability rests in whether the inferior subluxation is symptomatic, manifesting in pain or apprehension.[28]

Etiologic categorization of instability was addressed by Thomas and Matsen,[123] who described the acronyms TUBS and AMBRI to classify most patients with glenohumeral instability. TUBS (Traumatic Unidirectional Bankart treated with Surgery) refers to patients with a traumatic lesion who have a unidirectional component to their disease that frequently has a Bankart lesion and responds well to surgery. AMBRI (Atraumatic Multidirectional Bilateral treated with Rehabilitation or Inferior capsular shift) refers to patients who have an atraumatic cause of their multidirectional disease that often has bilateral shoulder findings and responds to rehabilitation. With the further understanding of multidirectional instability and the high incidence of rotator cuff interval lesions, the latter acronym was modified to AMBRII and included an inferior capsular shift with rotator interval repair.

Rockwood[100] added the concept of volition in describing four types of instability: type I—traumatic subluxation without previous dislocation; type II—traumatic subluxation after a previous dislocation; type IIIA—voluntary subluxation in patients with psychiatric problems; type IIIB—voluntary subluxation in patients without psychiatric problems; and type IV—atraumatic involuntary subluxation.

Gerber and Nyffeler[28] developed a classification scheme that divides instability into three classes: static, dynamic, and voluntary. Static instabilities (class A) are diagnosed radiographically and lack the classic signs and symptoms of instability. The humeral head is fixed in either an anterior, a posterior, or a superior position. Dynamic instabilities (class B) are posttraumatic (either through microtrauma or a single traumatic event), resulting in capsulolabral lesions or bone deficits that render the shoulder symptomatically unstable. Voluntary dislocations (class C) are classified separately from instabilities as the patient has complete control over the event.

The frequency of instability can be defined as either a primary or recurrent episode. While most acute primary dislocations are recognized, recurrent events, if overlooked or missed, can become chronic, locked dislocations that are associated with significant morbidity or dysfunction.[41,73]

Classification based on degree of instability differentiates subluxation from dislocation. *Dislocation* is defined as a complete separation of the articular surfaces often requiring a reduction maneuver to restore joint alignment. *Subluxation* is the symptomatic excessive translation of the humeral head against the glenoid without complete dissociation of the articular surfaces.

In general, as our understanding of the natural history, pathology, and treatment of shoulder instability has evolved, so has the classification scheme. The authors prefer to use an algorithmic approach to the classification of instability based on cause, direction, frequency, and volition (Fig. 11-1).

Traumatic Anterior Dislocation

The most common mechanism of a traumatic initial dislocation is an anteriorly directed force applied to the posterior aspect of the externally rotated and abducted arm. The humeral head becomes levered anterior in relation to the glenoid typically when the arm is in the overhead position. The patient's age and the force required to cause an initial dislocation are important factors in determining prognosis and associated injuries. Younger individuals typically sustain anterior shoulder dislocations during contact sports, whereas a low-energy fall may be the cause in the elderly. Patients who are younger than 30 years of age at the time of initial dislocation are more likely to experience recurrent dislocations, whereas patients older than 40 years of age are more likely to incur rotator cuff tears with the initial dislocation. Neviaser et al.[80] noted an 85.7% and 10.8% incidence of rotator cuff tears and axillary nerve palsy, respectively, in patients with an initial traumatic

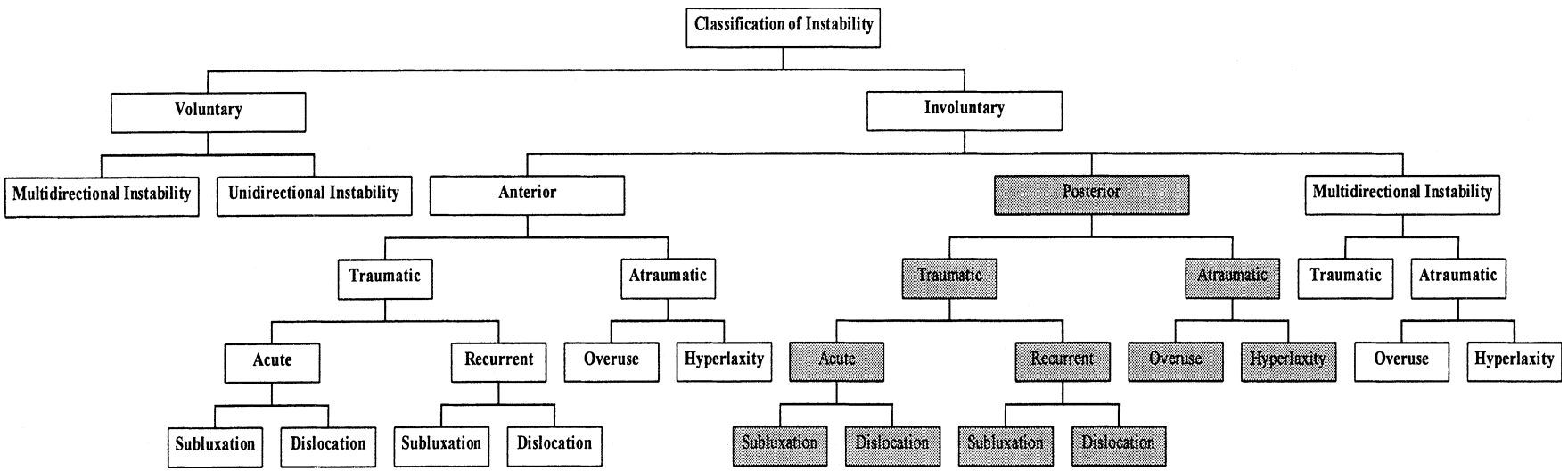

Figure 11-1 Classification of instability. (Modified from ref. 81 with permission.)

shoulder dislocation occurring after age 40. In patients older than 60, Gumina and Postacchini reported a 61% incidence of rotator cuff tears and a 9.3% incidence of axillary nerve palsy.[34] The inability to lift the arm overhead in older patients after an initial traumatic anterior dislocation is more likely to be related to a rotator cuff tear than to an axillary nerve palsy.

Traumatic instability may involve injuries to the capsulolabral complex or the osseous structures of the shoulder. These injuries typically take place with the arm in the "at risk" position of shoulder abduction and external rotation. Injuries to the inferior glenohumeral ligament complex may be at the humeral insertion site (HAGL—humeral avulsion of glenohumeral ligament),[132] midsubstance, glenoid (Bankart lesion), and rarely at both the humeral and glenoid.[21] The lesion may also be associated with a stripping of the periosteum off the glenoid (ALPSA—anterior labroligamentous periosteal sleeve avulsion).[81] Anterior inferior glenoid rim fractures (bony Bankart) and Hill-Sachs lesions can occur as the humeral head passes anterior to the glenoid. Large osseous lesions of either type may result in significant instability.

An anterior shoulder dislocation is diagnosed on the basis of the physical and radiographic examinations. Patients may present with an adducted and internally rotated arm and may be unable to externally rotate or fully abduct the extremity. The humeral head can often be palpated along the anterior aspect of the shoulder, whereas the posterior aspect may appear hollow. The posterior acromion may also seem more prominent. Manual reduction maneuvers are required to relocate the shoulder.

Recurrent anterior dislocations after an initial traumatic event can occur secondary to both traumatic and atraumatic causes. The rate of recurrent instability varies widely and is age dependant. Patients younger than 30 years have a higher redislocation rate when compared to older populations. Estimated rates of dislocation in patients younger than 30 years have been reported to be as high as 92% in some studies.[129] In a study of patients with primary traumatic anterior dislocations, Kralinger et al. found the only factor associated with recurrent dislocation was age between 21 and 30 years.[60] Other factors have also been noted to influence the rate of dislocation, including activity level, compliance with rehabilitation, contralateral shoulder instability,[86] glenoid rim avulsion fractures,[5] and large Hill-Sachs lesions.

Recurrent atraumatic instability (e.g., positional related dislocations) suggests a structural hyperlaxity with significant associated dysfunction. The time interval between the initial traumatic episode and subsequent nontraumatic dislocations will depend on the treatment after the original event and the degree of injury to the soft tissue or osseous stabilizers of the shoulder.[51] Early recurrent dislocations are seen in patients with concomitant large rotator cuff tears, severe disruption of the capsulolabral complex, and large fractures of the glenoid rim and/or greater tuberosity. Prompt operative fixation is recommended when these conditions are present.[99]

Anterior Subluxation

In an anterior subluxation, it is often difficult to identify an initial traumatic event. Rowe and Zarins[104] described recurrent transient subluxation of the shoulder causing a "dead arm syndrome." It is characterized by a sudden, sharp or paralyzing pain in the affected arm when the shoulder is forcibly moved into a position of maximum external rotation in elevation or is subjected to a direct blow. This condition is most common in overhead-throwing athletes.

The shoulders of baseball pitchers are exposed to high-energy repetitive stresses that can exceed the rate of tissue repair.[62] This process can damage or stretch the static stabilizing structures and, in turn, place an increased amount of demand for stability on the dynamic stabilizers. As the static stabilizers become attenuated, increased stress is placed on the dynamic stabilizers or rotator cuff muscles, which can fatigue. Anterior subluxation may occur as the compensatory mechanism becomes overloaded. This, in turn, can lead to subacromial impingement as the humeral head subluxates anteriorly and contacts the coracoacromial arch. Moreover, with anterior subluxation, the tendinous portion of the supraspinatus and infraspinatus may impinge on the posterosuperior border of the glenoid rim, resulting in glenoid impingement. Swimmers, volleyball players, and other overhead-throwing athletes may experience similar symptoms. These overhead athletes generally present with pain or discomfort that affects performance, rather than presenting with frank episodes of dislocation.

Occult instability may also be present in nonthrowing athletes. For weight lifters,[32] anterior subluxation may be the primary etiologic factor of shoulder dysfunction and pain. Weight-lifting maneuvers that produce forced abduction, extension, and external rotation (such as military presses, flies, bench presses, and latissimus pull-downs) may cause subluxation.

Multidirectional Instability

Patients with multidirectional instability have symptomatic instability in more than one plane of motion. Signs and symptoms can be subtle, and the condition in athletes is often mistaken for unidirectional instability. Most commonly, multidirectional instability is atraumatic, resulting from repetitive stress on a loose or hyperlax shoulder. The pathology is related to a large, lax capsule. This laxity may extend anteriorly, inferiorly, and even posteriorly. However, multidirectional instability may also present in patients without excess laxity of the shoulder. An acquired form can develop in individuals who sustain multiple traumatic events or repetitive microtrauma to the shoulder.

The traumatic forms may also exhibit a labral detachment in association with capsular laxity.[97]

Although diagnosis is frequently difficult, certain physical examination findings differentiate this condition from unidirectional instability. The patient with multidirectional instability exhibits inferior instability on examination of the affected shoulder.[77] Application of downward longitudinal traction on the humerus may exhibit a sulcus sign. A 2-cm distance between the humeral head and inferior acromion or an asymmetrical difference in sulcus signs between the shoulders is considered positive when symptomatic. When the sulcus sign is asymptomatic, it is considered a sign of inferior laxity.[28,71] These patients may also have generalized ligamentous laxity. Swimmers and gymnasts with multidirectional instability will often demonstrate capsular laxity without other pathologic lesions, in contrast to athletes in contact sports who will frequently exhibit an associated Bankart or Hill-Sachs lesions.[1,78]

The accurate diagnosis of multidirectional instability is essential as treatment options differ significantly from unidirectional instability. Neer and Foster highlighted three types of errors in diagnosis in their description of the inferior capsular shift[77]:

1. Treatment of subacromial impingement or biceps pathology rather than instability
2. Failure to address the redundant inferior capsule with resultant residual inferior instability
3. Overtightening of a hypermobile joint with secondary fixed subluxation or dislocation resulting in severe arthritis

Significant pitfalls exist in both the over- and underdiagnosis of multidirectional instability. Patients with unidirectional instability may undergo unnecessary procedures in an attempt to prevent laxity in other directions. Or, those with multidirectional instability, if unrecognized, may undergo stabilization procedures that do not address the inferior instability. McFarland et al. recognized the importance of establishing clearly defined parameters for diagnosing multidirectional instability. They demonstrated significant variability in the number of patients diagnosed with multidirectional instability when comparing four existing classification systems. The use of capsular laxity testing without uniform and well-defined criteria resulted in an overestimation of patients with multidirectional instability.[71]

Acute Posterior Dislocation

Acute posterior dislocations are rare and account for approximately 5% of all dislocations. They are considered acute when recognized within the first 6 weeks of injury.[42] Direct trauma to the front of the shoulder, a posteriorly directed force on an adducted arm (e.g., fall on an outstretched hand), and indirect muscle forces (e.g., seizure or electric shock) can all cause posterior dislocations. Acute posterior dislocations are unrecognized in 50% to 80% of patients at initial presentation[42,73,104] and are often misdiagnosed as a frozen shoulder. Emphasis on clinical examination and complete radiographic evaluation are essential in establishing the diagnosis. Posterior dislocations are also associated with impaction fractures of the humeral head (reverse Hill-Sachs lesion) and sometimes fractures of the surgical neck or tuberosities.

Chronic Posterior Dislocation

Chronic posterior dislocations are missed acute episodes that have remained unrecognized for at least 6 weeks. Chronic dislocations usually present with large impression fractures or reverse Hill-Sachs lesions that lock the humerus in a posterior position and demonstrate limited motion, shoulder dysfunction, and deformity.

Volitional Recurrent Posterior Subluxation

Voluntary recurrent posterior subluxation describes a group of patients with an underlying conscious or unconscious ability to subluxate their shoulder by using abnormal patterns of muscular activity. In this group, there is no initial anatomic pathology of the glenohumeral joint. Over time, stretching of the glenohumeral ligaments can occur such that an involuntary component to the instability develops. Some of these patients have underlying psychiatric disorders as a cause for willful and voluntary posterior subluxation. Rowe et al. labeled these patients habitual dislocators.[103] Habitual dislocators are distinguished from other patients with posterior subluxation who may have learned how to reproduce their instability by their willful desire to subluxate their shoulders (Fig. 11-2). Despite the best intentions of the treating physician, habitual dislocators will frustrate all treatment efforts (operative and nonoperative) because of their abnormal psychological need to subluxate their shoulder.[103] The predominant pathologic process in this group of patients is psychologic and treatment should be directed toward their psychological needs. Surgical intervention in this group is contraindicated.

A second group of patients can voluntarily reproduce their instability, but they have no underlying psychological need to do so. This is a learned behavior that over time may develop an involuntary component. It is this involuntary component that is bothersome to the patient and often initiates evaluation by a physician. This type of instability is not based on secondary gain, but can be demonstrated repetitively by the patient.

Electromyographic evaluation of these patients demonstrates selective inhibition of certain muscle groups that results in an unbalanced force couple leading to posterior subluxation. Activation of the deltoid and pectoralis major, without opposition from the posterior short rotators,

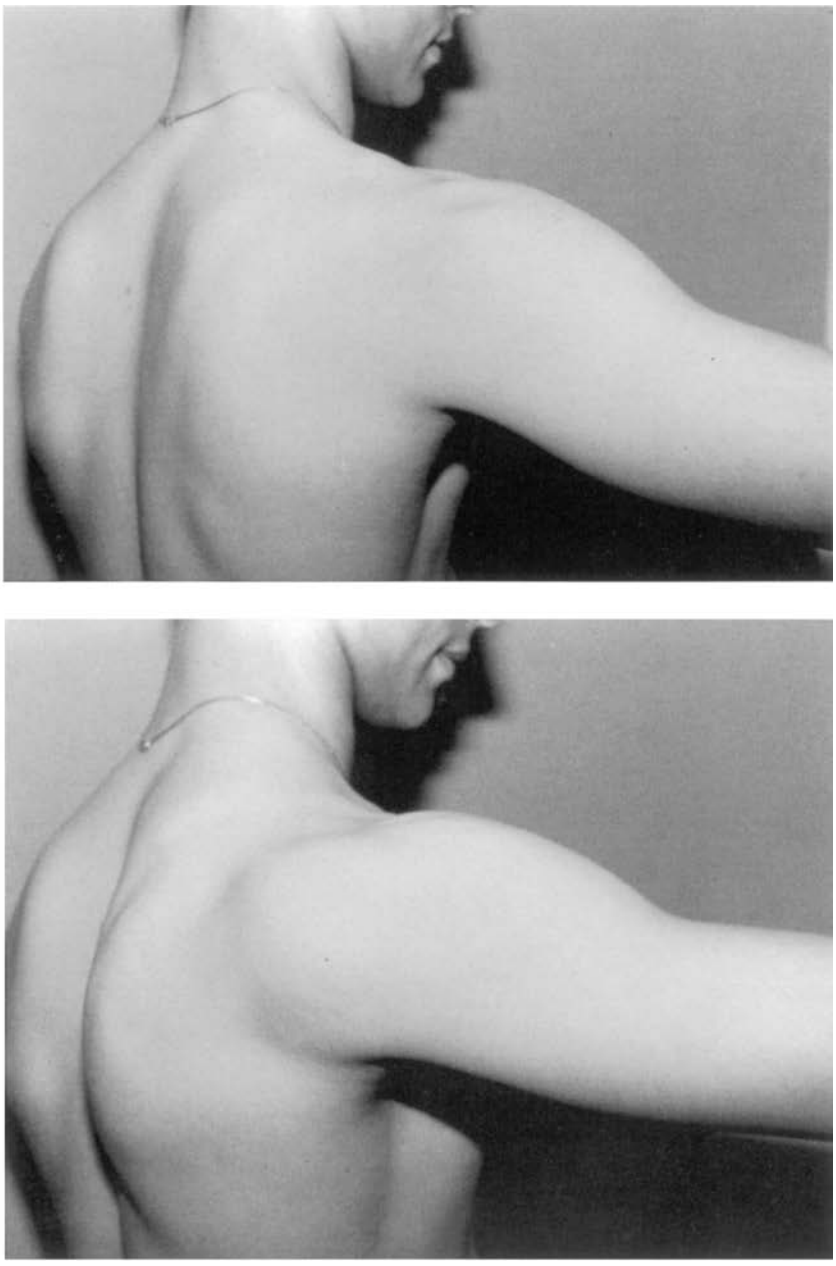

Figure 11-2 Photograph of a patient **(A)** before and **(B)** after dislocating his shoulder posteriorly with asymmetrical muscular contraction.

resulted in pushing of the humeral head posteriorly in several patients.[103] Alternately, Pande et al. demonstrated that unopposed activation of the posterior short rotators and posterior deltoid could pull the humeral head posteriorly.[92]

Dysplastic Recurrent Posterior Subluxation

Dysplasia of the glenohumeral joint is another uncommon cause of recurrent posterior subluxation.[15] Localized posterior glenoid hypoplasia, increased glenoid retroversion, and increased humeral head retroversion are potential causes of recurrent posterior subluxation. Recent investigations have documented a relatively low incidence of abnormal bony architecture in patients with instability and have postulated that developmental bony deformities can be a cause of recurrent posterior subluxation.[26,98,130] In a cadaveric study of 11,000 shoulders, up to 35% of the specimens had deficiencies in the posteroinferior aspect of the glenoid.[15] While the anatomic findings are of interest, correlation with instability could not be ascertained.

Recent computed tomography (CT) scan studies assessing glenoid version vary widely on the incidence of abnormal glenoid geometry and its contribution to instability. Gerber et al. and Randelli and Gambrioli found no correlation between altered glenoid version and instability.[26,98] Conversely, Hurley et al. and Wirth et al., in their respective

series, demonstrated increased glenoid retroversion and isolated posterior glenoid hypoplasia, respectively, in all patients with recurrent posterior subluxation.[49,131] While the true incidence of increased glenoid retroversion or hypoplasia in patients with recurrent posterior subluxation is unknown, it is likely that these factors can contribute to recurrent instability in some cases.[130]

Several findings on routine shoulder radiographs have also been associated with this condition. Radiographic findings include a shallow or irregular glenoid fossa, prominent coracoid process, enlarged acromion, hooking of the distal clavicle, associated hypoplasia of the upper ribs, and flattening of the humeral head.[9,18,119] The appearance and degree of these abnormalities vary in patients with this condition. CT scans can also be useful in delineating the severity of glenoid hypoplasia. Smith and Bunker reviewed 12 patients with primary glenoid dysplasia and noted that while younger patients may respond well to physical therapy, results will become less predictable with increasing age.[116]

Abnormalities of humeral torsion can lead to glenohumeral instability. While accurate measurement of humeral retrotorsion can be difficult, Schutte et al. described a reliable measuring technique using CT imaging.[108] This allows direct measurements of humeral retrotorsion, thereby eliminating the inaccuracy of plain radiographs. Kronberg and Brostrom have documented a correlation between decreased humeral retrotorsion and anterior glenohumeral instability in some patients. However, a relation between increased humeral retrotorsion and recurrent posterior subluxation has not been established.[61]

Reports of proximal humerus rotation osteotomy for the treatment of recurrent posterior subluxation are not supported by radiographic data demonstrating increased humeral retrotorsion.[9,121] The rationale for osteotomy in these series is that patients with recurrent posterior subluxation can provoke symptoms of instability by internal rotation of the arm. Limiting internal rotation through osteotomy was believed to correct the problem by preventing the patient from placing the arm in a position that would incite dislocation.

Acquired Recurrent Posterior Subluxation

Recurrent posterior subluxation is predominantly acquired either as a result of repetitive microtrauma or a single traumatic event. Traumatic events can cause both osseous and soft tissue abnormalities resulting in recurrent posterior instability. More importantly, in this group, the underlying pathologic lesion is critical to treatment and is used to categorize or define the instability. Lesions of the capsule, labrum, rotator cuff musculature, and glenoid can all contribute to recurrent posterior subluxation. The most common deficiency is a redundancy of the posterior capsule.[110] Additionally, dysfunction of normal scapulothoracic mechanics can place the glenohumeral joint at risk for recurrent instability.

Unlike the anterior capsule, the posterior capsule is thin. Together with the buttress provided by the glenoid labrum, they function as the primary static stabilizers to unidirectional posterior translation. Dynamic posterior stability is conferred by the rotator cuff musculature. The posterior capsule either stretches over time due to repetitive microtrauma or tears as a result of a single event. It can heal in an elongated position and thereby increase capsular volume. Posterior labral tears have been described with recurrent posterior subluxation; however, they are generally degenerative lesions rather than the rare capsular and labrum avulsion (i.e., reverse Bankart lesion) (Fig. 11-3).

The circle concept of capsuloligamentous stability addresses the relation between the anterior soft tissues and posterior stability.[109,110,117,122,127] Several biomechanical studies have investigated the contribution of anterior soft tissue structures to posterior stability. In addition to posterior capsular avulsion and partial or complete tearing of the tendinous portion of the posterior rotator cuff, various lesions of the anterior soft tissue structures have been identified. These include complete anterior capsular avulsions from the humeral neck and tears of the muscular portion of the subscapularis.[76,88]

Selective cutting of soft tissue structures thought to contribute to posterior stability has further defined the role of the anterior and posterior restraints to static posterior stability. Increased posterior translation consistently required a lesion of the posterior capsule, particularly the posterior band of the inferior glenohumeral ligament.[88] Isolated sectioning of the posterior rotator cuff musculature in the absence of a capsular lesion did not increase posterior translation.[87] Only when the posterior capsule was sectioned

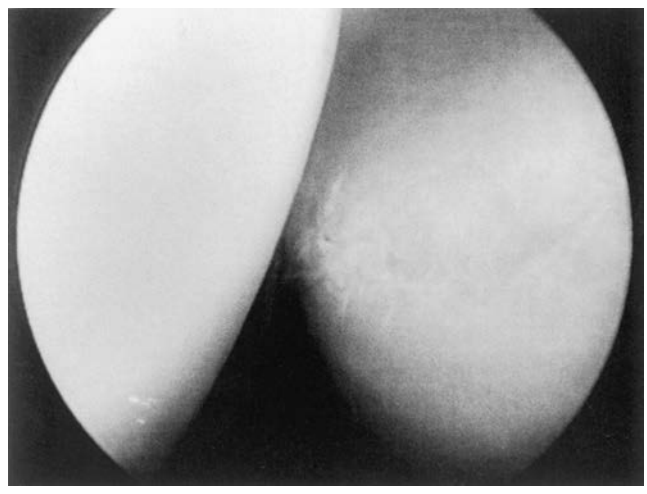

Figure 11-3 Arthroscopic photograph of a degenerative posterior labrum in a patient with recurrent posterior instability. Degenerative lesions of the labrum are typical for recurrent posterior instability. True reverse Bankart lesions are rare.

inferiorly was an increase in posterior translation noted. To effect subluxation or dislocation, sectioning of the anterior capsule and subscapularis tendon were required.

Several studies have found that the superior capsule (rotator interval) also plays an important role in posterior stability.[38,110,111] Sectioning the soft tissues of the rotator interval capsule often increased posterior and inferior translation to the point of dislocation, whereas imbrication of the rotator interval increased resistance to posterior and inferior translation.[38]

Acquired posterior subluxation is less commonly caused by posterior glenoid rim deficiency. Although it is uncommon, it can exist and should be investigated with imaging studies if suspected.[23,84,110] The relation between the degree of posterior glenoid erosion and recurrent posterior subluxation has not been established. It seems reasonable to assume that a large posterior glenoid defect will compromise the buttress effect of the glenoid to posterior translation.

In the resting state, the scapula lies on the posterolateral thorax at an angle of 45 degrees.[63] This position on the thorax places the posterior glenoid behind the humeral head, buttressing it against posteriorly directed forces. With shoulder elevation, the scapula rotates under the humeral head and provides a platform for glenohumeral motion.[101] A requisite for shoulder stability is that scapulothoracic and glenohumeral rhythm remains synchronous.[11,126]

Dysfunction of the scapulothoracic rhythm may compromise the stability of the glenohumeral joint.[126] The serratus anterior muscle plays a key role in this relationship. Its paralysis will result in scapular winging and loss of power in elevation that can potentially influence glenohumeral stability.[20] Warner et al. used Moire topographic analysis to study patients with glenohumeral instability and demonstrated abnormal scapulothoracic mechanics compared with those of asymptomatic patients.[126] Although no patient demonstrated severe scapular winging, the degree of scapulothoracic dysfunction was variable. In patients with glenohumeral instability and lesser degrees of scapulothoracic dysfunction, it is unclear whether instability is the result of altered scapulothoracic mechanics or the cause of it.

CLINICAL EVALUATION

A thorough history and physical examination are paramount to the diagnosis of glenohumeral instability. Plain radiographs will often augment or confirm the diagnosis; however, they are frequently normal. Further evaluation with more sophisticated imaging techniques such as fluoroscopy, CT, and magnetic resonance imaging (MRI) provides useful information but is not essential for the diagnosis. Examination under anesthesia and diagnostic arthroscopy are helpful tools in establishing the diagnosis in difficult cases and may serve as further confirmation of established clinical diagnoses.

History

Knowledge of the details surrounding the onset of symptoms is critical to establishing a diagnosis of glenohumeral instability. The age when symptoms began has significant prognostic value on determining the likelihood of recurrent instability. Detailing the mechanism of initial injury will help delineate between high-energy traumatic events (i.e., motor vehicle accidents), minimal traumatic events (i.e., throwing or swimming), or atraumatic events (i.e., combing hair). Similarly, the position of the arm during dislocation and the direction of the trauma help in understanding the potential direction of instability. The ease of relocation (i.e., need for manual reduction) and documentation of the direction of instability with radiographs aid in the diagnosis. Important information is gained by delineating further episodes of instability, the frequency at which they occur, the position at which they occur, and the ease of relocation after each event.

Prior treatment of any instability should be ascertained. If the patient was immobilized, then the position and duration of immobilization as well as any subsequent rehabilitation should be detailed. Past attempts at surgical management and the details of the operations should be obtained. Changes in the pattern of dislocation after surgery also provide essential information.

In nonlocked dislocations, patients will often complain of positional shoulder pain or discomfort; this pain may be present only after a frank subluxation or dislocation. Some patients may report shoulder pain that is dependant solely on the position of the shoulder, while others may only complain of a dull ache that is independent of arm position. Careful delineation between apprehension and pain is essential as pain may be indicative of other intraarticular pathology in the shoulder (e.g., rotator cuff tears). Apprehension with shoulder abduction and external rotation suggests anterior instability, while symptoms with the arm in flexion, adduction, and internal rotation suggest posterior instability. Patients may note pain while carrying heavy objects in an adducted position consistent with inferior instability. Overhead athletes may note a decrease in velocity, accuracy, or distance without episodes of dislocation that may be suggestive of more subtle forms of instability. In these athletes, the position of the arm where symptoms occur may help further elucidate the pathology. Pain during the cocking phase may indicate anterior instability, whereas pain in the follow-through phase may indicate posterior instability.

Further questioning is necessary to exclude other causes of shoulder pain. Pain radiating from the neck may be secondary to cervical spine disease. Pain referred from the hand may be secondary to compressive neuropathies.

Finally, the issue of volitional control over the instability should be addressed. A history of voluntary dislocation should alert the physician. Identification of the habitual dislocator, who dislocates for secondary gain, can have an important impact on prognosis.[102] The physician should be watchful of nonverbal clues that may signify underlying psychiatric illness. Patients who develop painful instability and subsequently learn to voluntarily dislocate, however, should be grouped separately.

Physical Examination

General Principles

It is important to obtain an overall evaluation of the musculoskeletal system through a systematic approach of inspection, palpation, range-of-motion analysis, strength testing, neurologic evaluation, shoulder stability assessment, and specialized testing. Range-of-motion and provocative testing of the cervical spine are required to exclude spinal disorders that may present as shoulder pain. Neurologic manifestations of cervical disease may be assessed by range-of-motion testing. It is unlikely that a patient with a full, painless range of motion will have clinically significant cervical spine pathology.

Visual inspection before examination may provide valuable information. The skin should be inspected for evidence of collagen disorders, which can present as thinning or widening of surgical or traumatic scars. Shoulder dislocation may injure the axillary nerve, so it is important to evaluate for deltoid atrophy and weakness. The sulcus sign, a visible depression inferior to the lateral edge of the acromion, may occur in patients with multidirectional instability caused by inferior luxation of the humeral head. Winging of the scapula may be evident in patients with anterior or posterior subluxation or secondary impingement and pain.

Both active and passive range of motion should be recorded. Forward flexion, abduction, internal rotation, and external rotation should be tested. The clinician should note dyskinesia and whether accessory muscles are activated with range-of-motion testing.

The shoulder girdle should be palpated for local tenderness and muscle tone. Patients with anterior subluxation often have tenderness over the posterior capsule, whereas those who are compensating for multidirectional instability may be tender along the medial angle of the scapula. Patients with anterior instability caused by subluxation and secondary impingement may have tenderness over the greater tuberosity or biceps tendon. Crepitation occurring on range-of-motion testing should be noted. While the motion of the upper extremity is being assessed, it is important to examine strength in the pain-free range of motion, paying particular attention to external rotation, internal rotation, and abduction strength to rule out rotator cuff pathology. These results should be compared with the uninjured side.

Generalized ligamentous laxity may be associated with shoulder instability. This should be assessed by evaluating range of motion in several joints. The degree of thumb hyperabduction with volar flexion of the wrist as well as index finger, elbow, and knee hyperextension should be noted. The tip of the thumb should not touch the volar aspect of the wrist and the index metacarpophalangeal angle should not exceed 90 degrees in the absence hyperlaxity (Fig. 11-4).

Specific Tests

After the preliminary examination, specific tests for instability should be performed. Asymptomatic shoulders often have considerable capsular laxity[37]; thus, the examination should first be initiated on the unaffected shoulder to obtain baseline data for each subsequent test. This approach also provides the

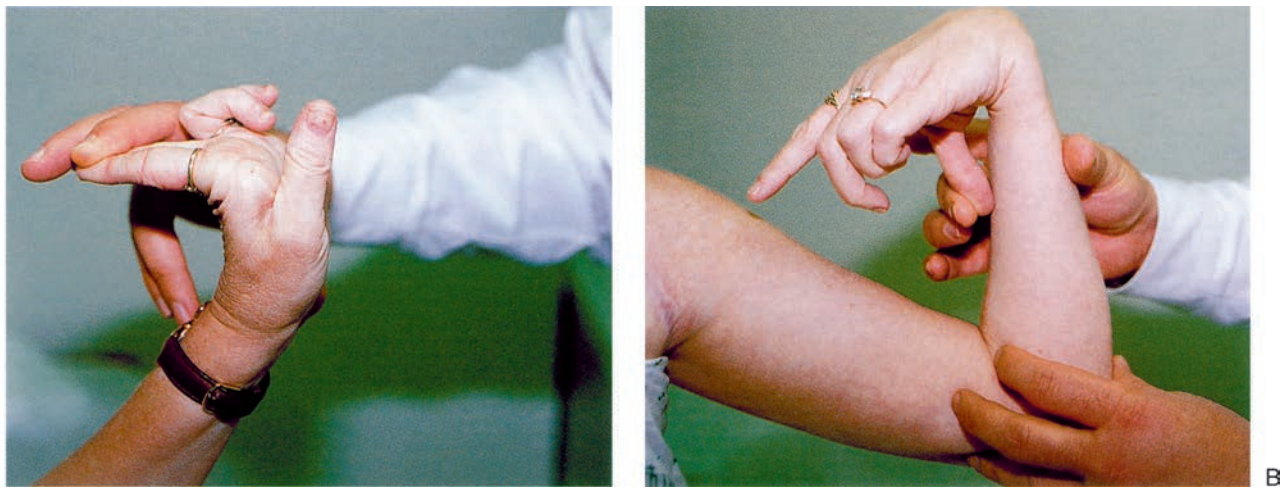

Figure 11-4 Patient with ligamentous laxity and index metacarpophalangeal angle of 90 degrees.

patient with the opportunity to become comfortable with each maneuver. Two components are considered when assessing the stability of the glenohumeral joint: (1) the amount of passive translation of the humeral head and glenoid fossa on stress testing, and (2) attempts to reproduce the symptoms of subluxation and apprehension by provocative testing of the shoulder in positions of compromise.

Sulcus Sign Test

The sulcus sign test[76] establishes the presence of inferior laxity, which if symptomatic is necessary to establish the diagnosis of multidirectional instability. In a patient with gross instability, a depression may be seen inferior to the anterior aspect of the acromion when the arms of a sitting patient are positioned along the side of the body. However, it is usually necessary to apply a traction force along the longitudinal axis of the humerus by pulling the humerus in an inferior direction. The distance between the acromion and the humeral head is then observed and recorded in centimeters. A measurement of 2 cm or more or an asymmetrical symptomatic sulcus sign is considered positive for inferior instability (Fig. 11-5).

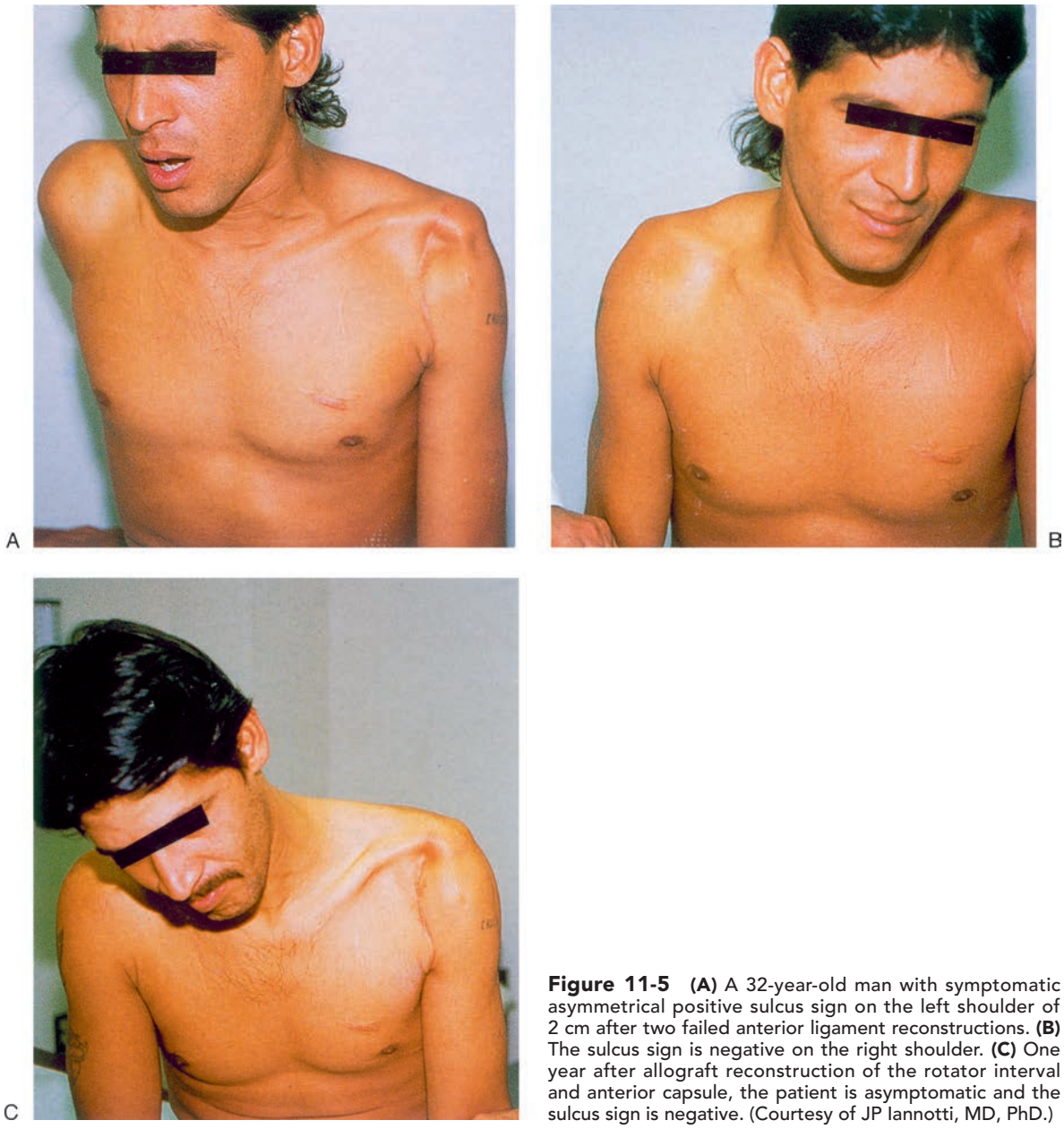

Figure 11-5 **(A)** A 32-year-old man with symptomatic asymmetrical positive sulcus sign on the left shoulder of 2 cm after two failed anterior ligament reconstructions. **(B)** The sulcus sign is negative on the right shoulder. **(C)** One year after allograft reconstruction of the rotator interval and anterior capsule, the patient is asymptomatic and the sulcus sign is negative. (Courtesy of JP Iannotti, MD, PhD.)

Translation Tests (Load and Shift)

Glenohumeral translation should be evaluated in both the upright and supine positions. The load-and-shift test establishes whether clinically significant translation of the humeral head is possible and if this translation reproduces the patient's symptoms. The examiner stands behind the seated patient with one hand on the affected shoulder. The other hand holds the proximal humerus with the thumb on the posterior aspect of the humeral head and the index finger on the anterior aspect. The initial movement is to load the humeral head into the glenoid fossa, reducing the inherent subluxation. Then the examiner attempts to shift the humeral head in anterior, posterior, and inferior directions while observing the area adjacent to the acromion for a sulcus sign.

Translation should also be evaluated in the supine position. The patient is positioned such that the center of the scapula rests on the edge of the examination table to eliminate a portion of scapulothoracic motion. The examiner grasps the humerus in a position of 20 degrees of abduction and forward flexion (the plane of the scapula) with neutral rotation. The humeral head is loaded and then stressed anteriorly, posteriorly, and inferiorly while the examiner notes the amount of excursion. The amount of excursion can be graded based on the degree of humeral head translation relative to the glenoid: grade 0, minimal movement; grade 1+, humeral head rides up onto the labrum (greater than contralateral side); grade 2+, humeral head subluxed but spontaneously reduces; and grade 3+, humeral head dislocates and remains dislocated.[1,125] The reproduction of the patient's symptoms during the translation tests confirms instability in the corresponding direction.

There have been several attempts to obtain an objective assessment of glenohumeral instability.[37,56,124] However, there has been no mechanism to assess quantitative translation of glenohumeral instability to date. The clinical methods described previously are subjective and rely heavily on clinical experience. Several studies have looked at the reproducibility of these tests. The reproducibility of the load-and-shift translation test was found to be 46% overall; however, when grades 0 and 1 were equalized, the reproducibility improved to 74%.[64] McFarland et al. and Ellenbecker et al. have found similar rates of reproducibility.[16,71]

Authors' Preferred Technique

Anterior Instability

Examination of the shoulder for instability using the load-and-shift technique is performed with the patient in the supine position. To test for anterior translation on the patient's right shoulder, the examiner positions the patient's arm in the plane of the scapula, at 45 to 60 degrees of abduction and neutral external rotation (Fig. 11-6). The examiner then places the left hand around the patient's arm

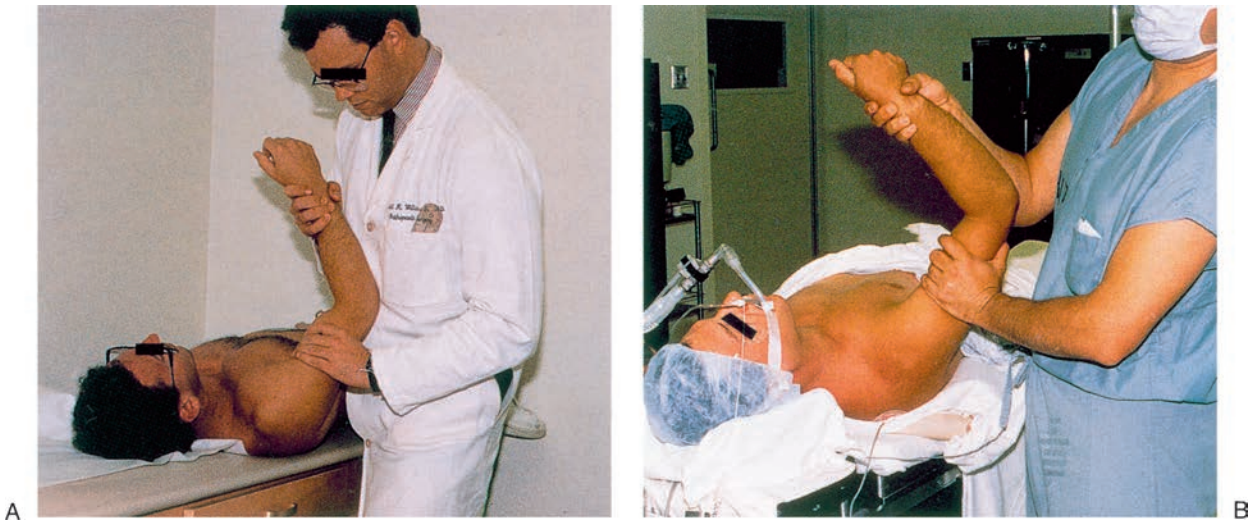

Figure 11-6 Initial position for load-and-shift test for anterior instability testing of the shoulder. For a right shoulder examination, the examiner's left hand grasps the patient's upper arm with the fingers anterior. The examiner's right arm positions the patient's arm and controls its rotation. The arm is placed in the plane of the scapula, abducted 40 to 60 degrees, and maintained in 0 degrees of rotation. The examiner's right arm places an axial load to the patient's arm through the humerus. The examiner's left hand then shifts the humeral head anteriorly, or anteroinferiorly over the glenoid rim. Grade 1 translation occurs when the humeral head rides over the anterior glenoid rim. Grade 2 translation occurs when the entire humeral head rides over the glenoid rim, but reduces when the dislocation force is released. Grade 3 translation occurs when the entire humeral head translates over the glenoid rim and does not reduce when the dislocation force is released. This test can be performed in **(A)** the relaxed awake patient or **(B)** the anesthetized patient.

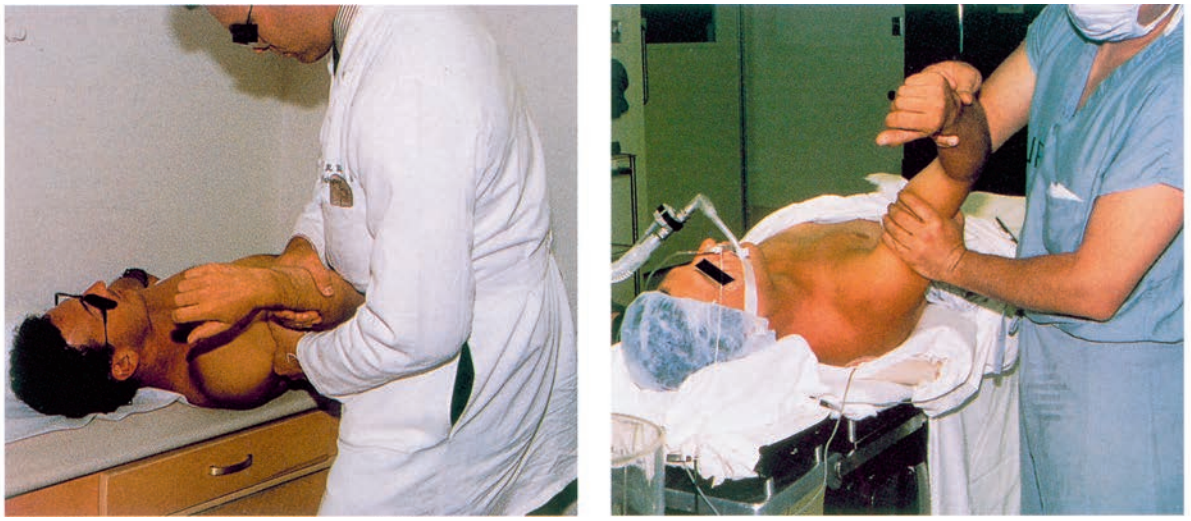

Figure 11-7 The second position for the load-and-shift test for the anterior stability is as described in Fig. 11-6 for the initial position, except that the arm is progressively externally rotated in 10- to 20-degree increments while the anterior dislocation force is alternatively applied and released. The examiner quantifies the degree of external rotation required to reduce the translation from grade 3 or 2 to grade 1. The examiner compares the normal and abnormal shoulders for this difference in translation with humeral rotation. The degree of rotation required to reduce the translation is an indicator of the functional laxity of the anterior inferior capsular ligaments. The examination is performed in **(A)** the relaxed awake patient or **(B)** the anesthetized patient.

at approximately the level of the deltoid insertion site with the thumb posterior and the remaining fingers anterior. The examiner's right arm controls arm position and provides an axial load to the humeral head to center it into the glenoid fossa. The examiner uses the left hand to provide an anterior or anteroinferior translation force to the humerus. When performed correctly, the scapula remains still and the amount of humeral head translation can be determined by both visual inspection and palpation. In almost all patients, there is some anterior or anteroinferior translation of the humeral head. As the examiner maintains an axial load to the humerus with the right arm, the patient's arm can be incrementally rotated into external rotation. With progressive external rotation of the humerus, the inferior glenohumeral ligament (IGHL) complex becomes taut (Fig. 11-7). With increased tension on the IGHL, there is decreasing anterior translation of the humeral head in the glenoid fossa.

Posterior Instability

To examine the patient's right shoulder, the arm is held in the plane of the scapula at 45 to 60 degrees of abduction and 45 to 60 degrees of external rotation. The examiner's hands are positioned in the same way as described for anterior translation testing, and an axial load is placed onto the humerus. The examiner's left hand is used to shift the head posteriorly. The examiner's right arm is used to incrementally internally rotate the patient's arm. During internal rotation, the posteroinferior capsule becomes increasingly tight, which should result in decreased posterior translation of the humeral head. It is important to note that in a normal shoulder, the humeral head can subluxate posteriorly up to 50%.[39,82]

Inferior Instability

The patient is examined in the seated position. The examiner first places a downward distraction force on the dependent humerus by applying traction at the distal humerus. The arm is in neutral rotation and the degree of humeral inferior translation (sulcus sign) is assessed in centimeters. The distraction force is then released, and the patient's arm is placed in maximum external rotation. The traction force is then reapplied and the sulcus sign is again measured. With external rotation, the anterior and rotator interval capsule is tightened (Fig. 11-8). Tightening these tissues should decrease the amount of inferior translation of the humeral head.

The examiner evaluates differences between shoulders, assuming the opposite shoulder is normal. In the awake patient, this examination may be limited by muscular guarding, which is not an issue with the examination under anesthesia. The examination in the awake patient may elicit painful grating or apprehension that can reproduce the patient's symptoms. The symptoms in the awake patient can help in defining the diagnosis; these symptoms are, of course, absent in the anesthetized patient. Therefore, it is our practice to perform this examination in both the awake and anesthetized patient as both examinations provide useful and complementary pieces of information.

Silliman and Hawkins[115] have provided a clinical grading system for this test. Grade I translation indicates that the humeral head can be felt to ride up on the face of the glenoid but cannot be moved over the glenoid rim. In grade II translation, the humeral head can be felt to glide over the glenoid rim, but it reduces spontaneously with release of pressure. This corresponds to clinical subluxation. Grade III

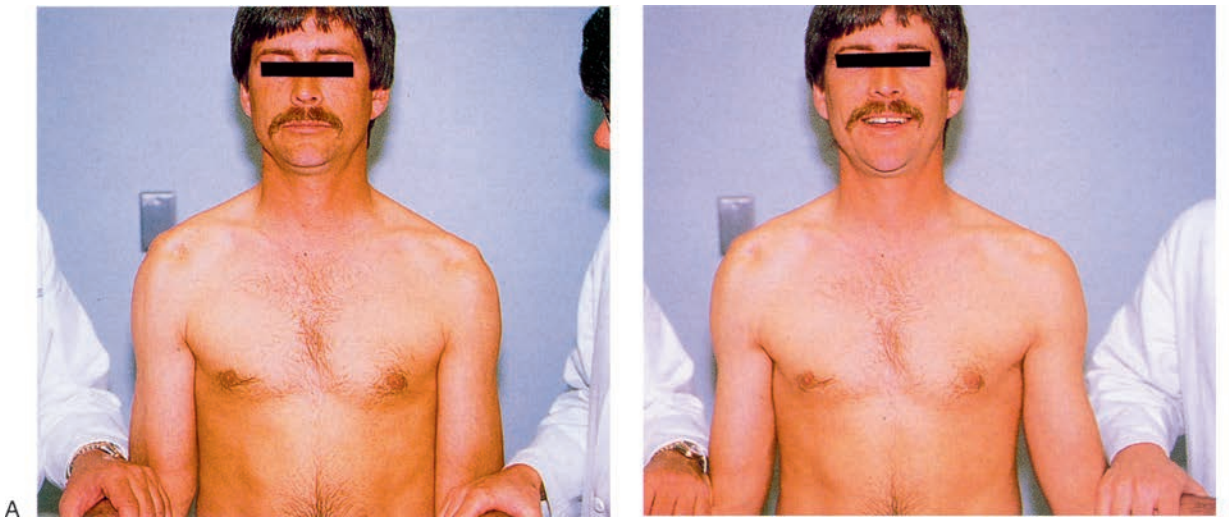

Figure 11-8 The sulcus sign tests the functional integrity of the superior glenohumeral ligament and rotator interval capsule. These tissues are lax in neutral rotation and tighten with external rotation. **(A)** A mildly positive sulcus sign (1 cm) on the *right*, which **(B)** does not reduce with external rotation, whereas the minor translation on the *left* does reduce with external rotation.

exists when the head remains dislocated after release of pressure, corresponding to clinical dislocation.

Apprehension Test

The apprehension test[104] places the shoulder in a provocative position of abduction and external rotation in an attempt to reproduce the patient's sensation of impending subluxation or dislocation (Fig. 11-9). This test should be performed in both the seated and supine positions. The examiner stands behind the seated patient, raises the patient's arm to 90 degrees of abduction, and begins to externally rotate the humerus. The hand of the examiner is placed over the humeral head with the thumb pushing the posterior aspect of the humeral head for extra leverage. Fingers are placed anteriorly to control any sudden instability

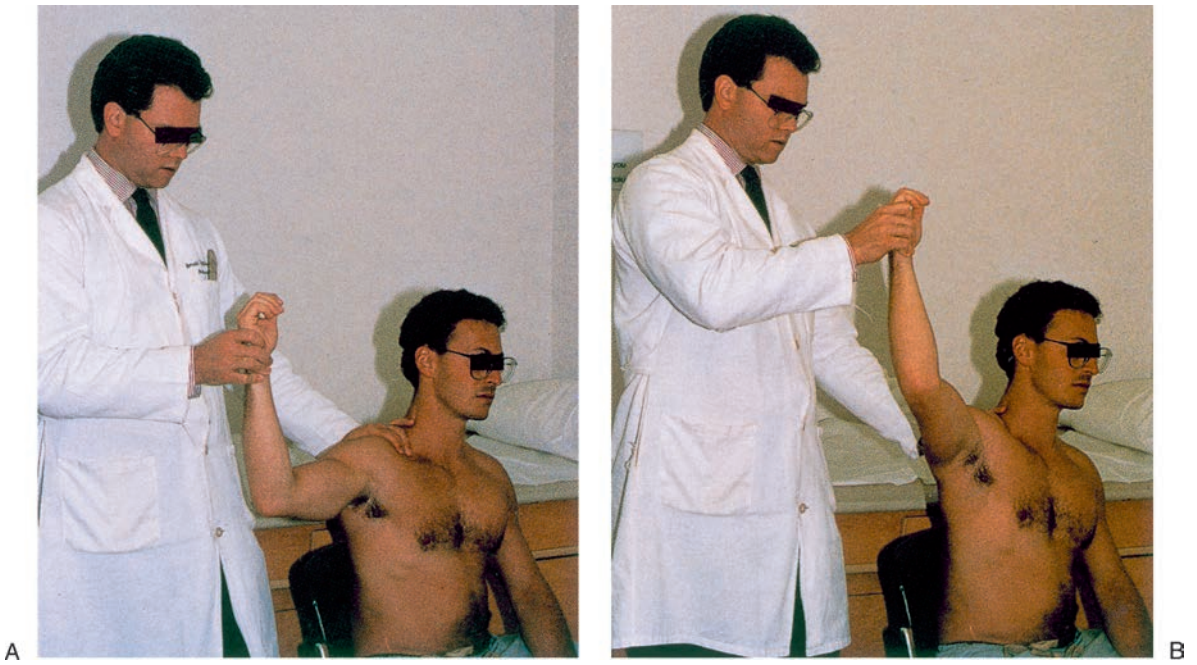

Figure 11-9 The apprehension test is performed by placing the arm in a position of abduction and external rotation in an attempt to produce a patient's feeling of subluxation or dislocation. The patient is tested in the seated position with the arm **(A)** at 90 degrees of abduction and **(B)** at 120 degrees of abduction. The higher degree of abduction places greater stress on the inferior capsular pouch.

that might occur. With increasing external rotation and controlled gentle forward pressure exerted against the humeral head, the patient may have an apprehensive feeling of impending instability. The patient communicates this apprehension verbally, by facial expression, or by protectively contracting the shoulder muscles.

This test is more easily performed in the supine position because the scapula is stabilized against the edge of the examining table, giving the examiner more control over the maneuver. The edge of the table acts as a fulcrum and the arm acts as a lever. As the humeral head is levered anteriorly, the amount of external rotation required to produce apprehension is noted.

In evaluating throwing athletes, Jobe et al.[54,55] described pain as the only positive finding in apprehension testing. Pain is suggestive of anterior subluxation, even in the absence of apprehension. Shoulder pain in this position must be differentiated from that of impingement by performing a relocation test (discussion follows). However, Speer et al.[118] showed that pain was poorly correlated with instability and that apprehension was more specific to the diagnosis. When apprehension rather than pain was used to determine a positive test, the accuracy of the relocation test improved from less than 50% to greater than 80%.[118] Pain with abduction and external rotation that is relieved by the relocation test may reflect internal glenoid impingement.

Relocation Test

The relocation test[55] should be performed in conjunction with the apprehension test (Fig. 11-10). With the arm in the position that produces apprehension, a posteriorly directed stress is exerted on the proximal humerus. Both the apprehension and the pain should disappear with this maneuver. This test presumably reduces the subluxation by pushing the humeral head posteriorly and thus relieving symptoms.

However, patients with rotator cuff involvement but no instability may have pain in the apprehension position and experience relief of pain with the relocation test. Such a patient may have a deep surface tear of the supraspinatus tendon, which is painful in the apprehension position because the tendon is trapped between the greater tuberosity and superior glenoid rim. The augmentation test, performed by pulling the humeral head forward while in the apprehension position, will increase the pain felt by rotator cuff patients, but will not augment pain in those with pure instability.

Release Test

The release test[115] is performed immediately after the relocation test. The examiner releases the posteriorly directed force on the humerus. A positive test occurs when the patient's symptoms return.

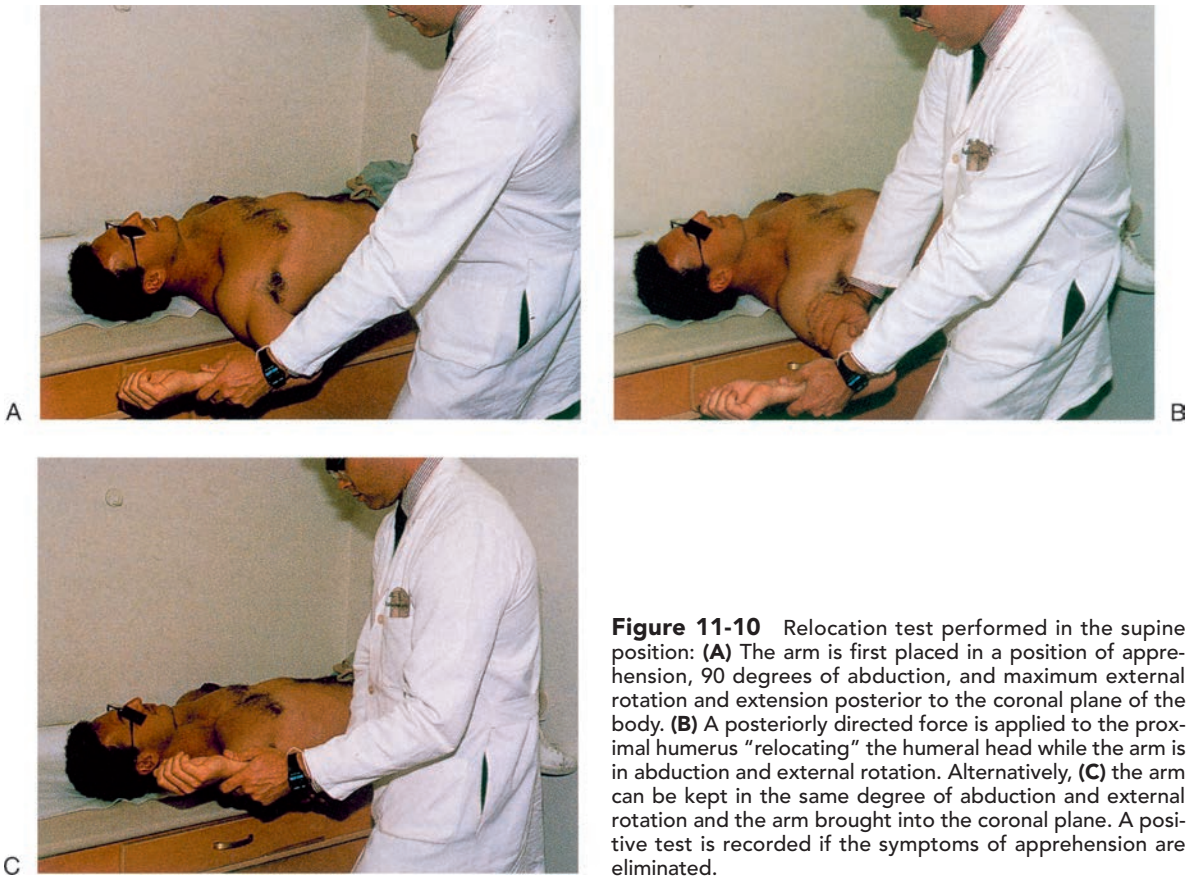

Figure 11-10 Relocation test performed in the supine position: **(A)** The arm is first placed in a position of apprehension, 90 degrees of abduction, and maximum external rotation and extension posterior to the coronal plane of the body. **(B)** A posteriorly directed force is applied to the proximal humerus "relocating" the humeral head while the arm is in abduction and external rotation. Alternatively, **(C)** the arm can be kept in the same degree of abduction and external rotation and the arm brought into the coronal plane. A positive test is recorded if the symptoms of apprehension are eliminated.

Tzannes et al. assessed the interexaminer reliability of provocative tests of the shoulder. Similar to the findings by Speer et al.,[118] they found the greatest consistency among examiners when apprehension rather than pain was used to determine a positive test. Additionally, they demonstrated a greater reliability for relocation and release tests than for the apprehension test.[125]

Lo et al. evaluated the validity of the apprehension, relocation, and release tests in detecting anterior instability. When the patient exhibited apprehension in all three tests, the positive and negative predictive values were 93.6% and 71.9%, respectively. The release maneuver was the single most accurate test of the three. Interobserver reliability was 83%.[66]

Posterior Apprehension Test (Posterior Stress Test)

The posterior apprehension test is performed with the shoulder adducted, internally rotated and flexed to 90 degrees. A posteriorly directed force is applied. A positive test occurs with subluxation causing pain. Apprehension typical of anterior instability is unusual.[40,96]

Hyperabduction Test (Range of Passive Abduction Test)

The hyperabduction test[24] was described to specifically address laxity of the inferior glenohumeral ligament. The physician stands behind the patient and places an inferiorly directed force on the scapula, while the neutral upper arm is abducted with the elbow flexed to 90 degrees. A positive test occurs with abduction of greater than 105 degrees. A positive hyperabduction test indicates laxity of the IGHL.

After a thorough physical examination, further diagnostic testing should be performed to clarify the nature and pattern of the instability.

Examination under Anesthesia

The numerous methods available for diagnosing shoulder instability may not provide sufficient information to proceed confidently toward operative treatment. Anesthesia is sometimes necessary to produce sufficient muscle relaxation and freedom from pain for an adequate examination. Under anesthesia, only a small amount of force is necessary to stress the ligaments of the joint capsule. Additionally, patients with subtle instability in the office may exhibit significant laxity under anesthesia. While examination under anesthesia (EUA) provides important information, it may not change the treatment algorithm.

Cofield and Irving[12] noted that EUA is the "most definitive, accurate, noninvasive test of shoulder instability." They described a systematic method for shoulder examination in the anesthetized patient that should be applied to both shoulders:

The examiner stands by the side being examined and supports the limb in one hand by holding the midforearm. The other hand rests across the top of the shoulder with the proximal aspects of the fingers resting on the acromion as a point of reference. The thumb can then be used to force the humeral head forward and the index and long fingers can be used to force the humeral head posteriorly. When assessing inferior translocatability, the hand supporting the limb moves proximally to the elbow to pull the arm downward. The sequence of examination is listed in Table 11-1.

TABLE 11-1
SEQUENCE OF INSTABILITY EXAMINATION

Direction	Force of Examining Hand	Force of Positioning Hand	Position of Arm
Anterior	Anterior	None	Abd 30° Ext 10° ER 0°, 45°, 80°
Anteroinferior	Anteroinferior	Toward joint in axis of arm	Abd 100° Ext 10° ER 0°, 45°, 80°
Posterior	Posterior	None	Abd 45° Flexion 30° IR 0°, 45°, 80°
Posteroinferior	Posterior	Toward joint in axis of arm	Abd 80° Flexion 45° IR 0°, 45°, 80°
Inferior	None	Away from joint (distraction)	Abd 0° Flexion 0° Rotation 0°

Abd, abduction; ER, external rotation; EXT, extension; IR, internal rotation.

The sensitivity and specificity of EUA is improved by examining the shoulder in various positions of shoulder elevation and rotation.[13] For the optimal determination of translation in the anterior direction, the arm is positioned in 80 degrees of external rotation and a load-and-shift test is performed. If the affected side demonstrates a higher grade of translation than does the unaffected side, then the test for instability is positive.[114] In one study,[114] negative examinations correlated with negative surgical findings in 28 of 28 cases, and 25 of the 27 patients with positive findings on examination under anesthesia had pathologic findings at surgery.

Faber et al. studied examination findings in patients with anterior instability while awake and under anesthesia. They found that anterior translation was higher during EUA than during awake examination in both symptomatic and asymptomatic shoulders. A subtle increase in instability during the awake examination was amplified during the EUA. They concluded that EUA is useful in confirming the direction and degree of glenohumeral instability.[19]

For the optimal determination of translation in the posterior direction, the arm is placed in 45 to 60 degrees of abduction and the humerus is placed in variable amounts of humeral rotation. With the arm in external rotation, the posterior capsular structures are lax, allowing posterior translation of the humeral head against a posteriorly directed force. Progressive internal rotation tightens these structures, resulting in capture of the humeral head and elimination of posterior translation against a posteriorly directed force. It is important to note the degree of internal rotation at which posterior translation is minimized. Differences in posterior translation in different arm positions when compared with the opposite shoulder are indicative of posterior capsular insufficiency.

Yoldas et al. evaluated patients with multidirectional instability. These patients demonstrated increased translation in all three directions when compared to the asymptomatic side during both the awake examination and EUA. The same study evaluated patients with posterior instability using awake examination and EUA. They found increased anterior translation in both symptomatic and asymptomatic shoulders during EUA; however, the posterior translation noted during awake examination did not change.[133]

EUA, when combined with diagnostic shoulder arthroscopy, can provide an accurate assessment of instability in most cases.

Diagnostic Arthroscopy

We routinely perform diagnostic arthroscopy on all patients before surgical repair of shoulder instability. It provides a precise diagnosis of the type of instability and associated injuries. A systematic approach should be developed in examining a shoulder with suspected instability.[68]

Shoulder arthroscopy may be performed in the lateral decubitus or beach-chair position. The beach-chair position requires additional assistance for control of the arm. It does, however, provide more versatility in arthroscopic examination and allows easier positioning for conversion to an open procedure if desired. After systematic examination of the joint for other pathology, we examine the inferior recess for loose bodies and the humeral head for the presence and size of a Hill-Sachs deformity. We evaluate for ease of movement of the arthroscope anteriorly through the glenohumeral joint and into the axillary pouch. A positive "drive-through" sign (described by Pagnani and Warren[90]) results from substantial laxity in the IGHL. A positive drive-through sign is considered present when, under distraction loading, the humeral head separates widely from the glenoid, facilitating easy passage (or "drive through") of the arthroscope from posterior to anterior. McFarland et al. recently reported a high sensitivity (92%) and low specificity (37.6%) for the drive-through sign in patients with instability. They concluded that the sign should not be used as the definitive or sole criterion for diagnosing instability. However, it is useful in identifying laxity.[72] Finally, we direct our attention to the anterior labrum and capsule to confirm the diagnosis and direct the appropriate treatment.

If present, a Bankart lesion is usually obvious, and it confirms the diagnosis of anterior instability (Fig. 11-11). It is not uncommon, however, to find a detached labrum with scar filling the defect. These lesions are not always apparent with initial inspection of the labrum, but can be

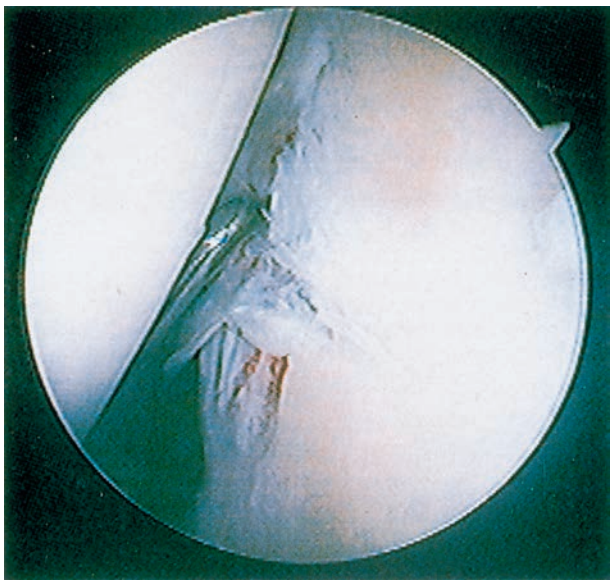

Figure 11-11 Arthroscopic photograph of an acute Bankart lesion.

defined with careful probing. A similar lesion of the posterior–inferior labrum seen in posterior or multidirectional posteroinferior instability was described by Kim et al.[58] This lesion is identified as a superficial tearing between the posteroinferior labrum and the glenoid articular cartilage. The labrum is not completely detached, but it appears flat with loss of its normal height.

Hintermann et al.[46] performed a prospective arthroscopic study of 178 patients, each of whom had at least one episode of shoulder dislocation. Reporting the arthroscopically determined pathologic findings, they concluded that associated injuries were more common than expected and that there were significant differences between preoperative and postoperative diagnoses. The most frequent arthroscopic findings were anterior glenoid labral tears (85%), ventral capsule insufficiency (80%), Hill-Sachs lesions (67%), glenohumeral ligament insufficiency (55%), rotator cuff tears (20%), posterior glenoid labral tears (8%), and superior labral anterior-to-posterior (SLAP) lesions (5%). They also noted that the labrum and anteroinferior glenoid rim showed abnormalities corresponding to different types of anterior instability. They concluded that prestabilization shoulder arthroscopy increases the accuracy of diagnosis and has the potential to identify the optimal surgical procedure. Caspari and Geissler[7] described other arthroscopic manifestations of anterior shoulder subluxations and dislocations that directed their surgical treatment. They included labral and anterior capsular injuries of varying severity.

Mok et al. reviewed 166 shoulder arthroscopies in patients with symptoms of subluxation without dislocation. Arthroscopy confirmed the working diagnosis in 80% and changed the diagnosis in 20%. They concluded that arthroscopy is instrumental in establishing a diagnosis in this difficult group of patients.[74]

Werner et al. reported the arthroscopic findings in patients with atraumatic shoulder instability that failed conservative treatment.[128] They found intraarticular lesions of the capsulolabral complex similar to those in posttraumatic instability.

Radiographic Evaluation

Initial plain film evaluation of the shoulder should include an anteroposterior (AP) view (Fig. 11-12), a true AP view of the glenoid (also called the Grashey projection; Figs. 11-13 and 11-14), a scapulolateral "Y" view (Fig. 11-15), and an axillary view (Fig. 11-16) Additionally, the West Point, Stryker notch, Didiee, and Velpeau views are useful.

West Point View

To obtain a West Point axillary view the patient is positioned prone with the shoulder abducted 90 degrees and the elbow bent and hanging off the edge of the table. The film cassette is positioned at the superior aspect of the shoulder (Fig. 11-17). The x-ray beam passes through the axilla at a 25-degree angle to the table top and is centered inferomedial to the acromioclavicular joint. This view

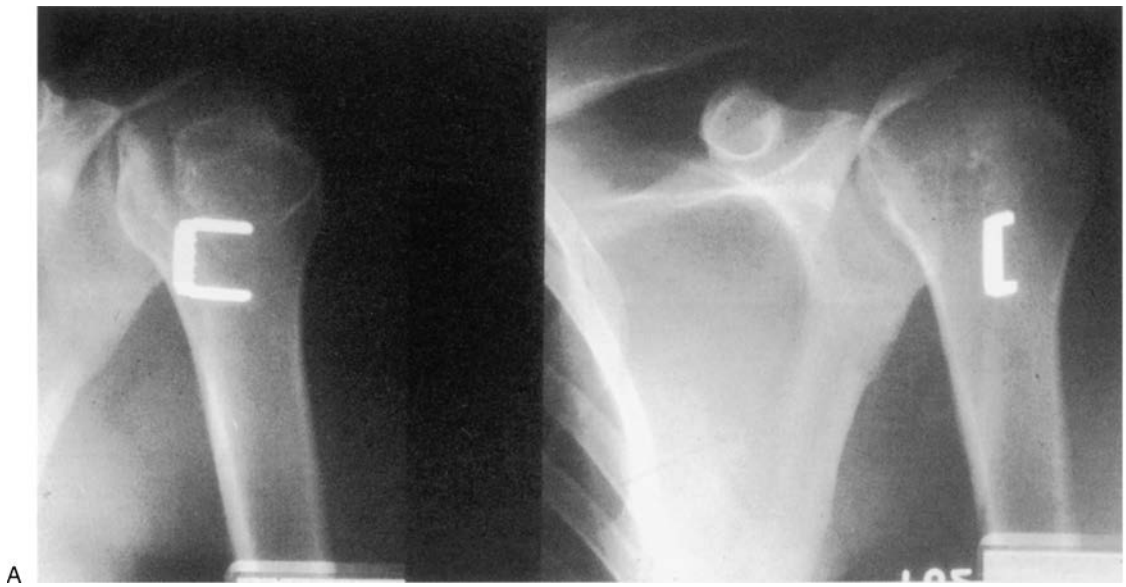

Figure 11-12 **(A,B)** Initial radiographic series should include an anteroposterior view of the shoulder. This example shows the glenohumeral articulation to be overlapping (i.e., the x-ray beam is taken in the coronal plane of the body). This x-ray film was taken in both internal and external rotation, as shown by the position of the tuberosities and the metal staple used for a Magnussen-Stack procedure.

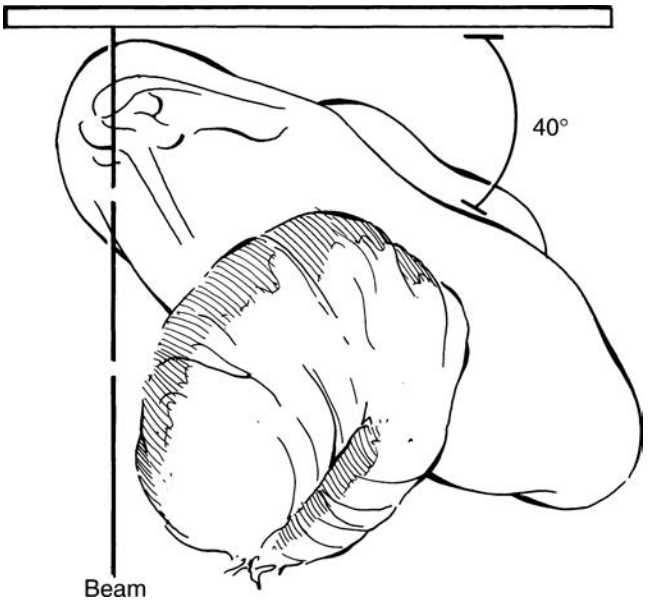

Figure 11-13 Positioning for the Grashey view.

provides the best evaluation of the anteroinferior glenoid rim, which is seen in tangent (Fig. 11-18).

Velpeau View

The Velpeau view is a modified axillary view that does not require arm abduction. With the arm in internal rotation at the side of the body (while wearing a sling), the standing patient leans backward 30 degrees over the cassette, which is on the table (Fig. 11-19). The beam is directed superoinferiorly through the shoulder.

Stryker Notch View

For the Stryker notch view, the patient is positioned supine, with the elbow elevated over the head and facing forward. The film cassette is placed under the shoulder, and the beam is directed cephalad at a 45-degree angle centered on the axillary fold (Fig. 11-20). This view demonstrates the posterolateral humeral head to advantage and is useful for evaluating Hill-Sachs deformity (Fig. 11-21).

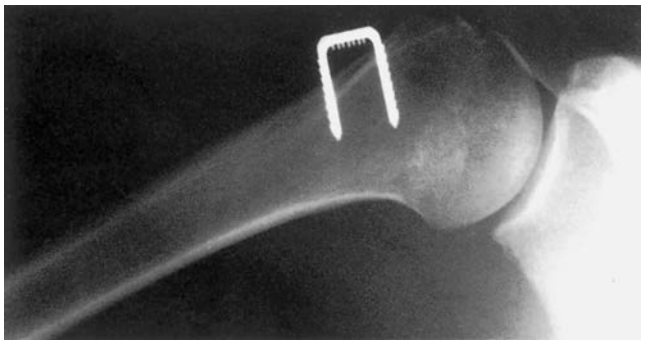

Figure 11-14 A Grashey/anteroposterior view of the glenoid: The same patient is shown as in Fig. 11-12. The glenohumeral articulation is not overlapping and the glenoid is better visualized. With the arm in external rotation, the metal staple is seen in profile. This radiographic view is taken in the plane of the scapula.

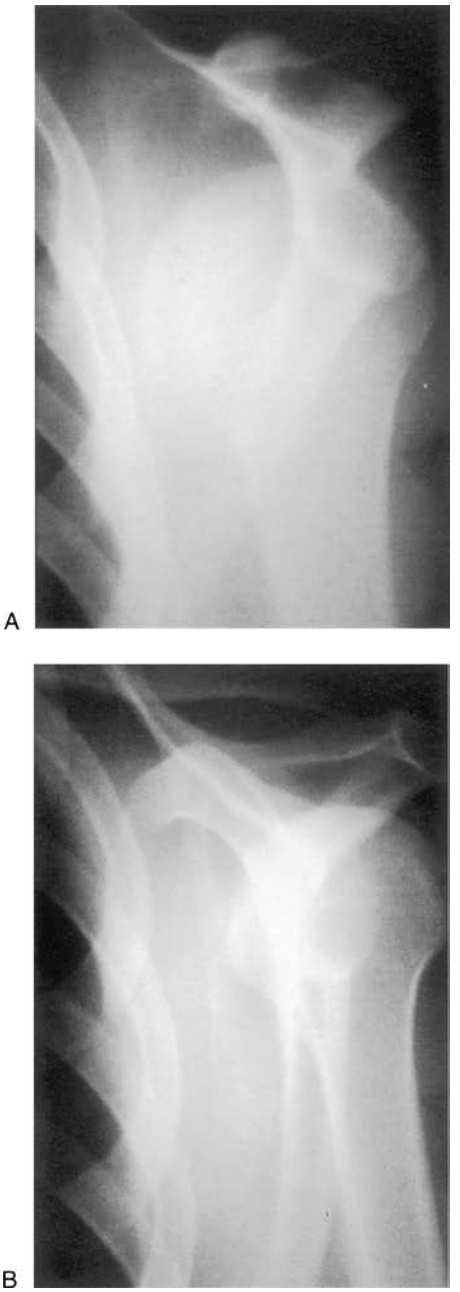

Figure 11-15 Scapulolateral "Y" view showing **(A)** anterior dislocation and **(B)** reduction of the glenohumeral joint.

Didiee View

The Didiee view is obtained with the patient in the prone position and the arm abducted and slightly flexed at the

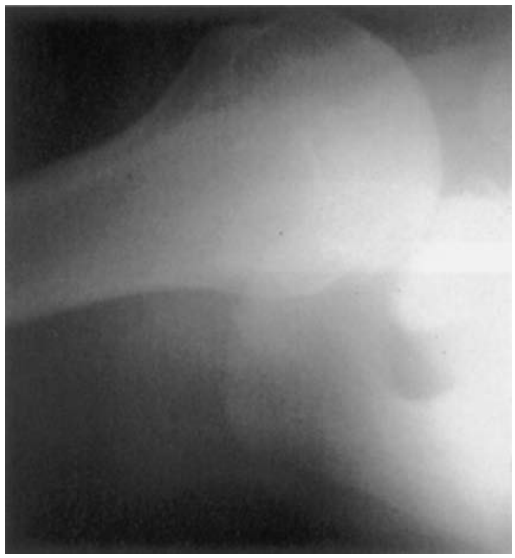

Figure 11-16 Axillary view of the shoulder showing a bony Bankart lesion.

elbow; the dorsum of the hand is on the iliac crest. The film cassette is placed under the shoulder and the beam is directed from the lateral aspect toward the humeral head at a 45-degree angle (Fig. 11-22) These radiographic views allow assessment of glenohumeral alignment as well as detection of fractures, degenerative changes, loose bodies, and calcification around the joint capsule.

Apical Oblique (Garth) View

The apical oblique view was described by Garth et al.[25] to best visualize the anterior inferior glenoid rim for fractures or calcification. The patient is seated, and the arm is on the patient's lap. The x-ray beam is angled 45 degrees to the thorax (plane of the scapula) and 45 degrees caudad. The beam is centered over the glenohumeral joint, and the cassette is placed posterior to the shoulder (Figs. 11-23 and 11-24).

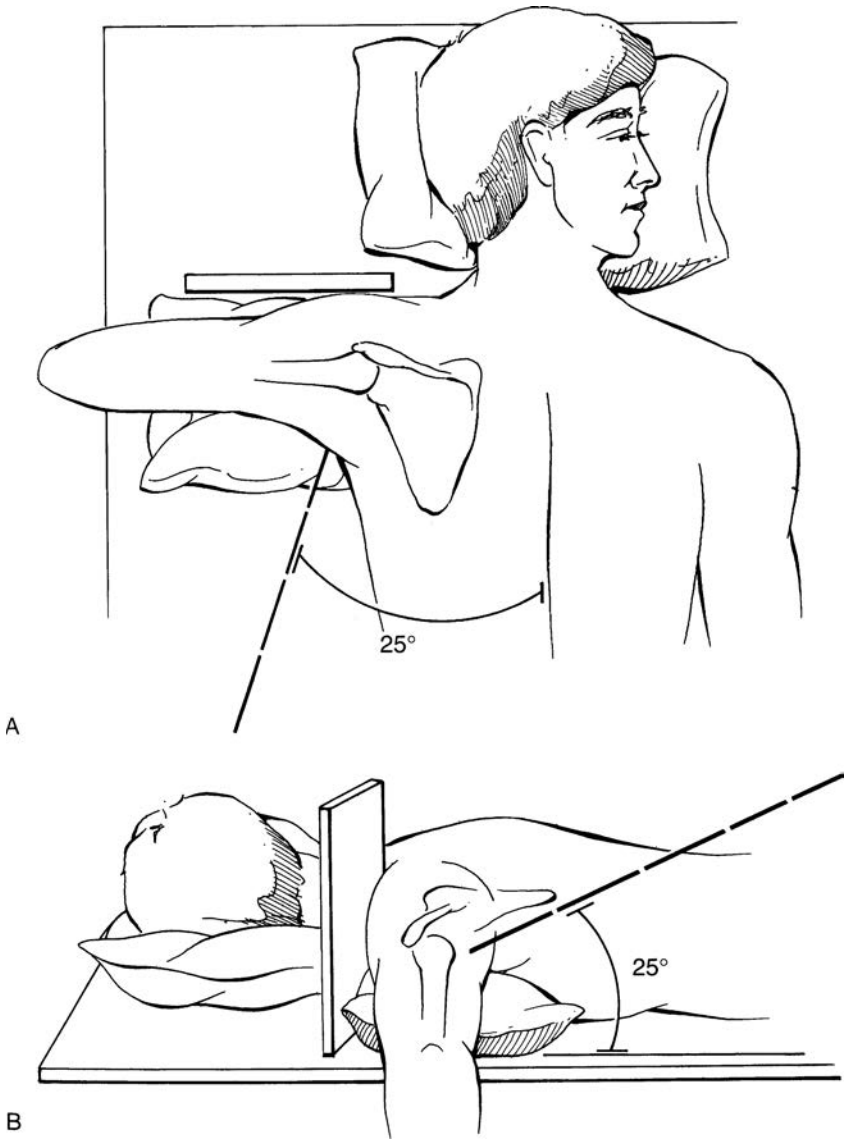

Figure 11-17 Positioning for the West Point view.

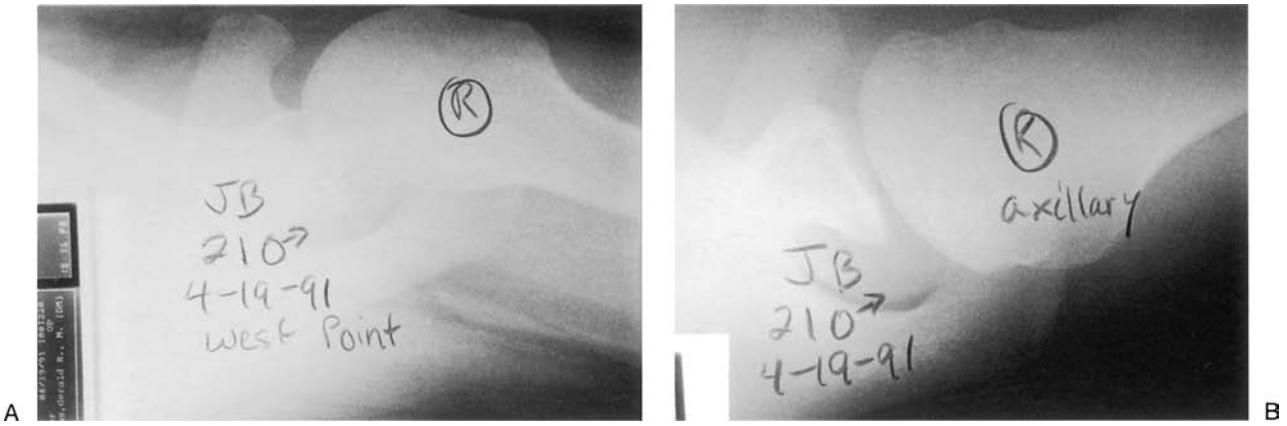

Figure 11-18 **(A)** West Point axillary view showing the anterior and posterior glenoid rim. There is no abnormal glenoid bone identified, although it appears as if there is some anterior glenoid wear. **(B)** In this patient, the standard axillary view demonstrates a small glenoid rim fracture.

General Considerations

Anterior Instability

Anterior dislocations are usually accompanied by a degree of inferior displacement that makes the injury apparent on the AP projection. Because of pain in the acute setting, the patient may be unable to abduct or rotate the shoulder, making the axillary, West Point, Stryker notch, and Didiee views difficult to obtain. The Velpeau and scapulolateral Y views are usually more comfortable for the patient because they do not require abduction. External rotation views are not recommended after reduction, as this position predisposes the patient to redislocation.

The anterior, inferior, and medial displacement that occurs during anterior dislocation of the shoulder causes the posterolateral aspect of the superior humerus to impinge on the anteroinferior rim of the glenoid. This may result in an osteochondral compression fracture of the posterolateral humeral head, also known as the Hill-Sachs lesion.[45] Fracture can occur as a sequela of the first or any subsequent dislocation episode. It is best seen on the AP view of the shoulder with internal rotation of the humeral head or on the Stryker notch view.[94,105]

Osteochondral glenoid lesions may also occur and are difficult to detect on prereduction films. A fracture of the glenoid rim, also called a bony Bankart lesion,[2,3] is best

Figure 11-19 Positioning for a Velpeau view.

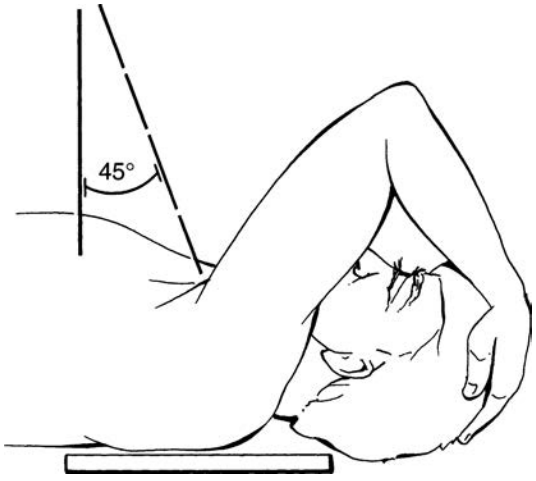

Figure 11-20 Positioning for a Stryker notch view.

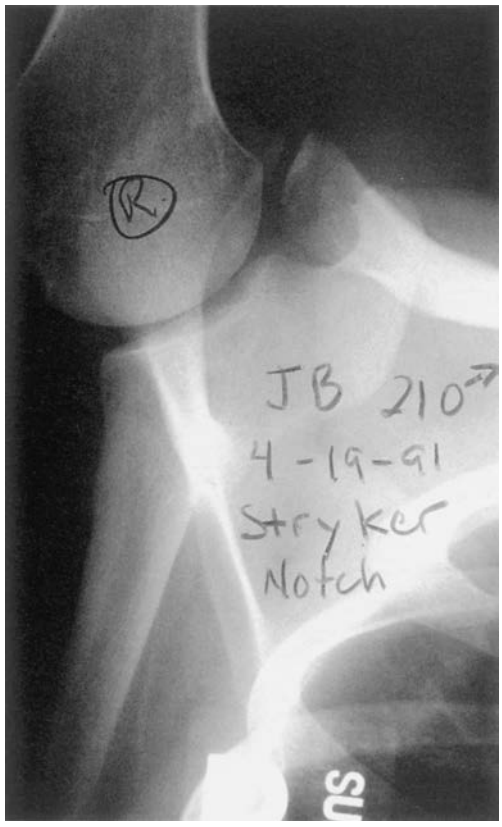

Figure 11-21 Stryker Notch view of the same patient in Fig. 11-18 shows a small wedge-shaped defect on the posterolateral humerus, which represents the Hill-Sachs lesion.

visualized on West Point or Didiee views[94] or on a Grashey view.[30]

The nonosseous Bankart lesion involves only the cartilaginous glenoid labrum; therefore, it cannot be evaluated by plain films. This lesion is best demonstrated by MRI (Fig. 11-25), but may also be visualized by CT arthrography (Fig. 11-26) or arthroscopy (Fig. 11-27).

Posterior Instability

In posterior dislocations, particular attention should be directed toward the axillary view. If positioning is not possible because of painful abduction, then a Velpeau axillary can be obtained. This will diagnose the position of the humeral head and any associated bony lesions. The classic radiographic features of posterior dislocation include humeral head overlap on the glenoid rim on an AP radiograph, an empty glenoid on axillary or lateral radiograph, fracture of the lesser tuberosity, and a reverse Hill-Sachs lesion.

In posterior subluxation without dislocation, routine radiographs may not demonstrate any abnormalities. The axillary view may show evidence of calcification of the posterior capsule, fracture or erosion of the posterior glenoid (Fig. 11-28), or reverse Hill-Sachs defects (Fig. 11-29).[23,83,84]

Stress axillary radiographs or fluoroscopy are generally not necessary, as the history and physical examination

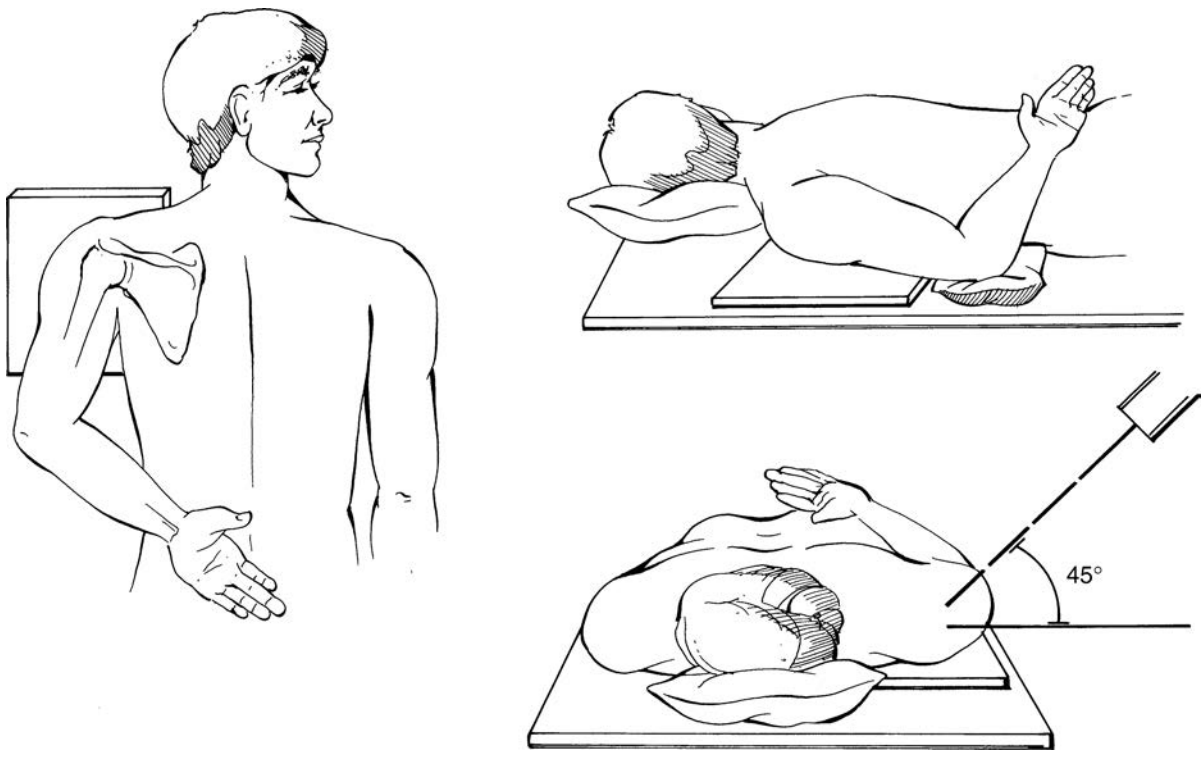

Figure 11-22 Positioning and technique for a Didiee view.

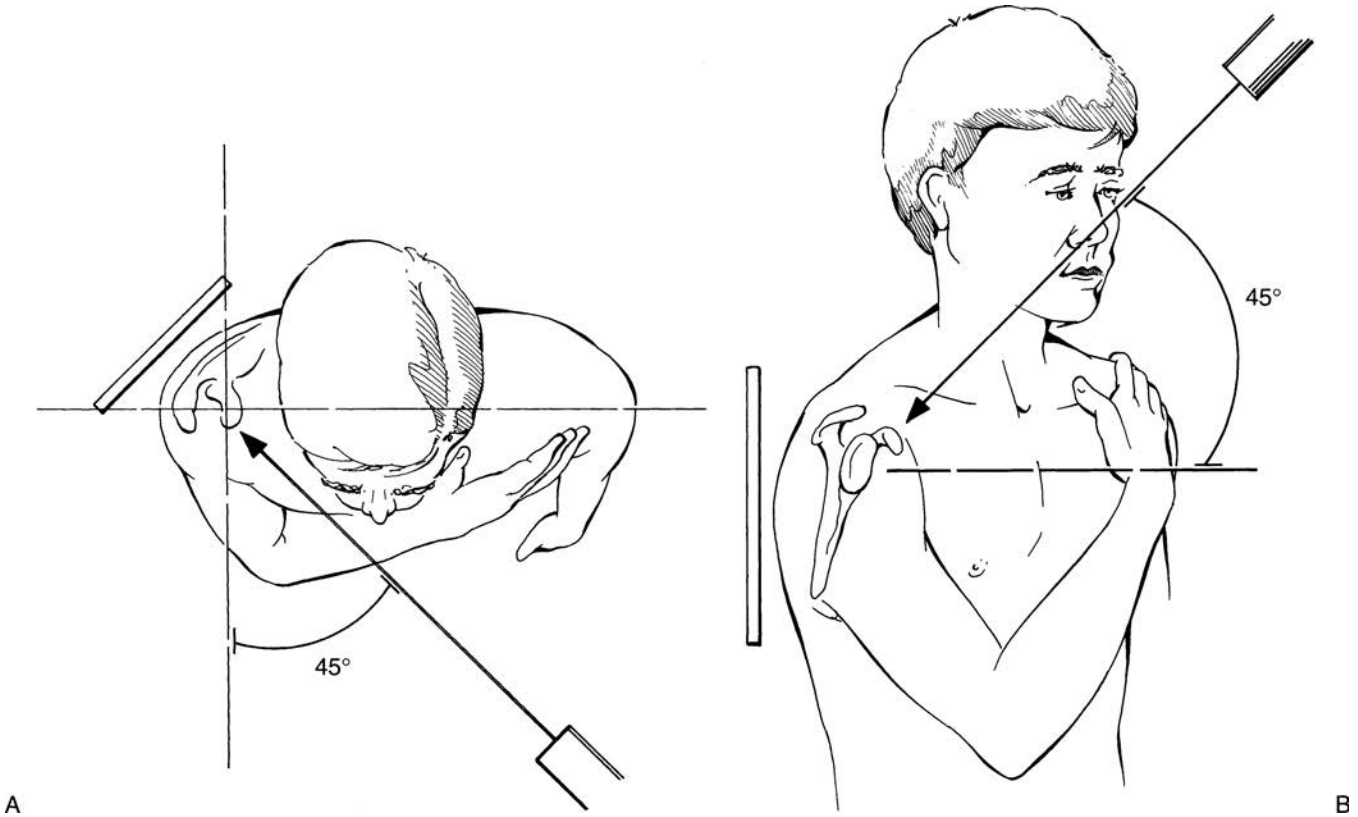

Figure 11-23 **(A,B)** Positioning and technique for the apical oblique (Garth) view.

usually clarify the diagnosis. However, some authors have found these imaging modalities quite helpful in the small group of patients with a dubious diagnosis.[18,53,82] Comparison with the unaffected side is recommended, as posterior glenohumeral translation of 50% has been demonstrated in normal subjects.[39,83]

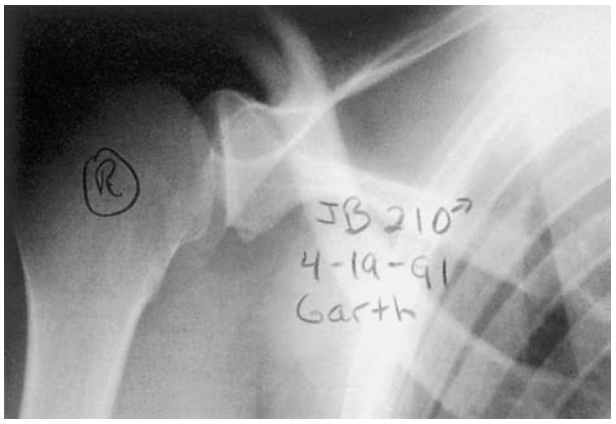

Figure 11-24 An apical oblique view of the same patient in Figs.11-18 and 11-21 demonstrates calcification of the anterior inferior glenoid rim associated with anterior glenohumeral instability.

Multidirectional Instability

In multidirectional instability, plain radiographs are generally normal. Nonetheless, they should be evaluated for the presence of humeral head defects and glenoid lesions. Stress radiographs can be used to demonstrate inferior subluxation; however, this technique is generally not needed.[53]

Fluoroscopic Evaluation

Although imaging studies can provide useful anatomic information, they do not reflect a dynamic situation. Additional information can be obtained by fluoroscopic stress evaluation of the glenohumeral joint. In a study of 50 patients, Papilion and Shall[93] evaluated fluoroscopic examination of shoulder instability under general anesthesia. Translation was expressed as the percentage of displacement of the humeral head relative to the glenoid. They found that up to 14% anterior translation and 37% posterior translation are "normal." A greater percentage of translation indicated instability. These criteria had an overall sensitivity of 93% and specificity of 100% for the diagnosis of the presence and direction of instability. It must be emphasized that an absolute degree of translation cannot be correlated with

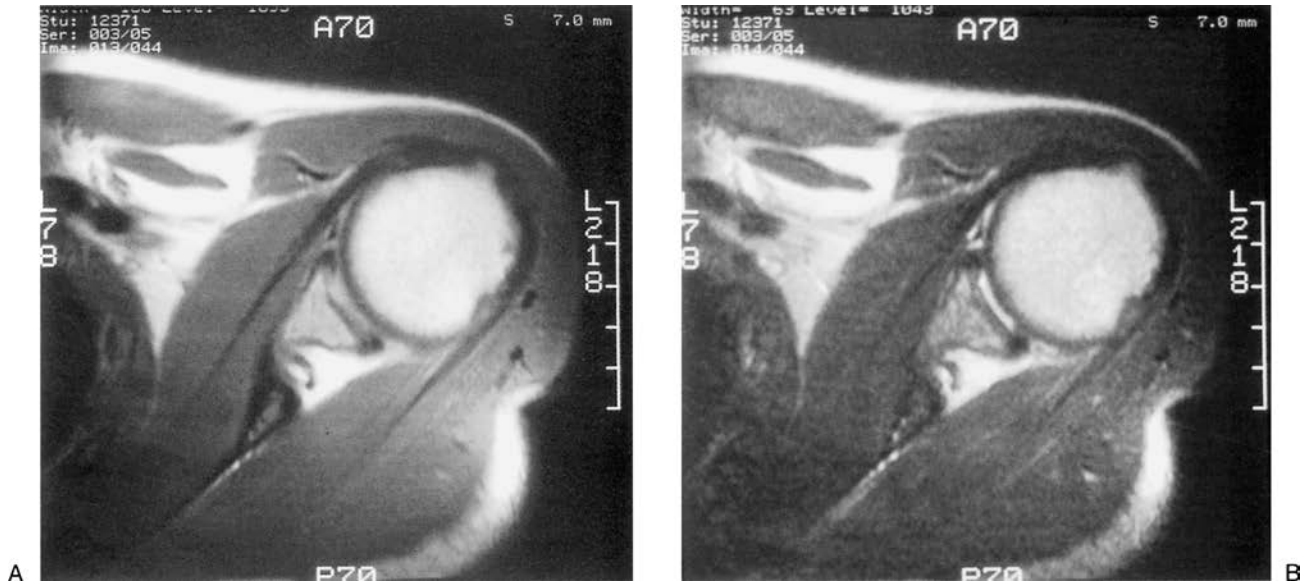

Figure 11-25 **(A)** T-1-weighted and **(B)** T-2 weighted axial cuts demonstrating a small Hill-Sachs lesion on the posterosuperior humeral head and an anterior labrum tear that is best indicated by the high signal fluid between the labrum and the anterior glenoid rim.

instability. The amount of translation can vary greatly among individuals, and comparative examination of the contralateral normal shoulder should be routinely carried out.

Computed Tomography Arthrography

Prior to the use of MRI, CT arthrography was widely used, as it was considered the standard for imaging the glenoid labrum. CT arthrography can reliably define the osseous structures of the shoulder and assess both labral and capsular integrity. CT also excels in its ability to define bony detail. If plain radiographs suggest abnormalities of glenoid version, glenoid hypoplasia, or glenoid erosion, CT is often the study of choice. The value of CT arthrography in defining intraarticular pathology has been studied. While Bigliani et al. found one-third of CT arthrograms to be

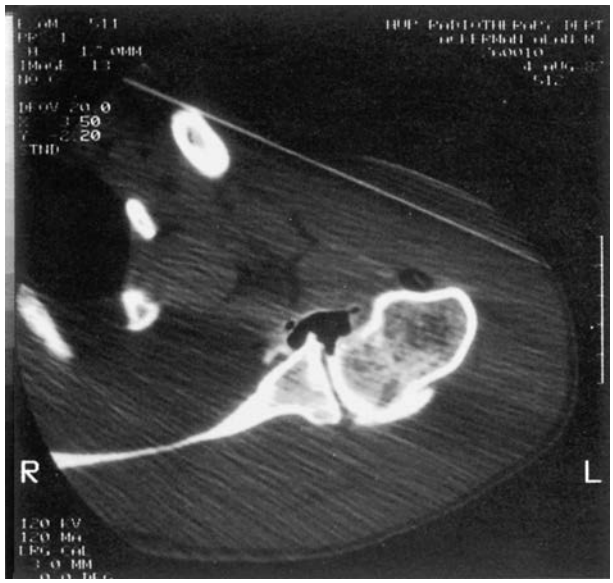

Figure 11-26 Computed tomography arthrogram demonstrating avulsion of the anteroinferior capsule from the glenoid rim.

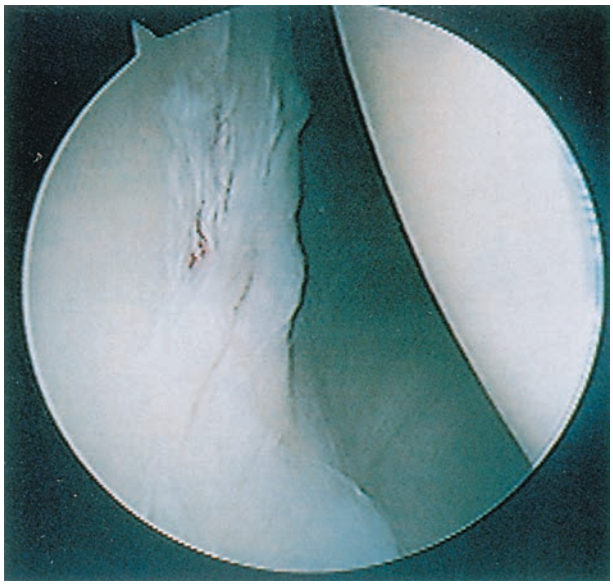

Figure 11-27 Arthroscopic photograph of an anteroinferior capsular avulsion (Bankart lesion).

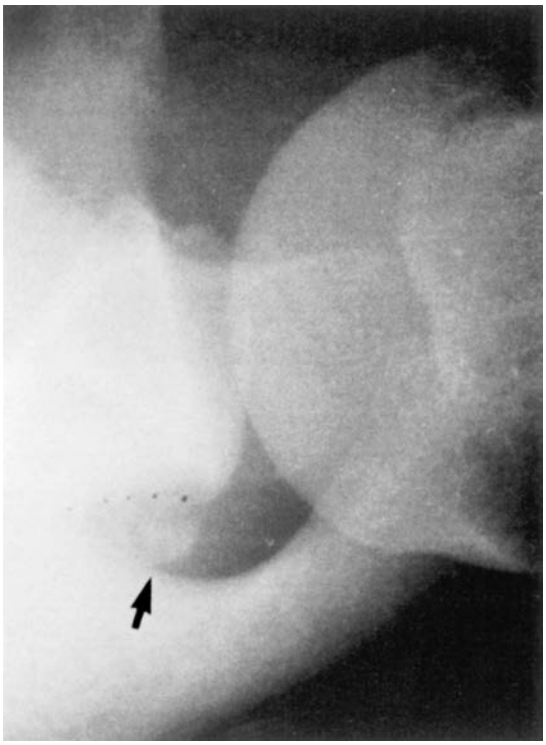

Figure 11-28 Axillary radiograph showing fracture of the posterior glenoid rim (*arrow*). The fracture fragment has healed to the posterior glenoid neck.

over- or under-read for labral pathology when correlated with findings at surgery, Callaghan et al. found CT arthrography to be 100% accurate, sensitive, and specific for posterior labral defects[4,6] (Fig. 11-30). While MRI and MR arthrography have been shown to be more sensitive in the detection of many labral abnormalities,[106] CT arthrography may be used in cases where MRI is contraindicated. The disadvantages of this technique include exposure to ionizing radiation, invasiveness of the procedure, and inability to assess partial-thickness tears of the rotator cuff, biceps tendon, or labrum.

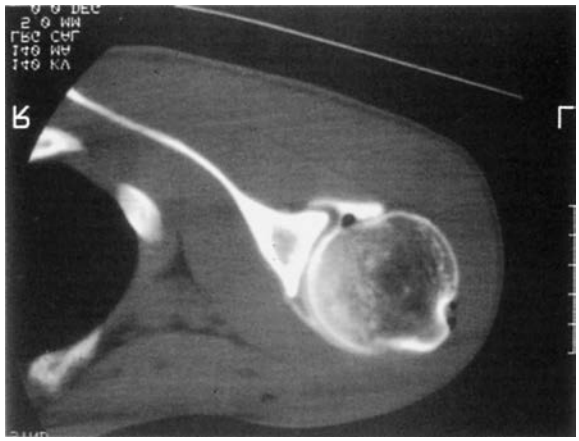

Figure 11-30 A computed tomography arthrogram of the shoulder. Intraarticular contrast improves visualization of the glenoid labrum.

Magnetic Resonance Imaging

MRI has improved our ability to assess soft tissue pathology about the shoulder. The advantages of MRI over plain radiographs and CT include no exposure to ionizing radiation, excellent soft tissue resolution, noninvasiveness, and ability to image in multiple planes. Numerous studies have shown that MRI is superior to other imaging studies at defining labral and capsuloligamentous pathology.[33,48,50,52,57,112] An MRI of the shoulder can demonstrate additional shoulder pathology, such as rotator cuff pathology, labral morphology, and osseous integrity.

Several investigators have studied labral morphology in normal patients. Although the posterior labrum maintains either a triangular or rounded shape, the appearance of the anterior labrum is variable. Neumann et al.[79] studied 30 asymptomatic volunteers and found cleaved, notched, or flat anterior labra in 30% and described absent portions of the anterior superior labrum in 6%. McCauley et al.[69] disagreed and found labral clefts in only 4% of healthy subjects.

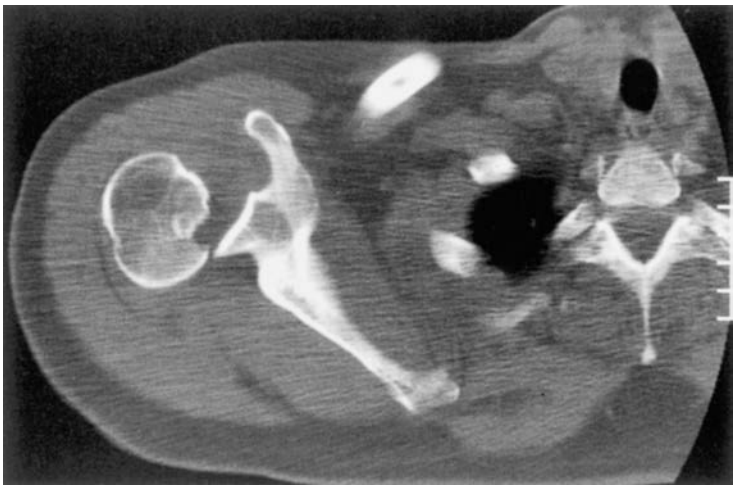

Figure 11-29 A computed tomography scan of a large reverse Hill-Sachs lesion.

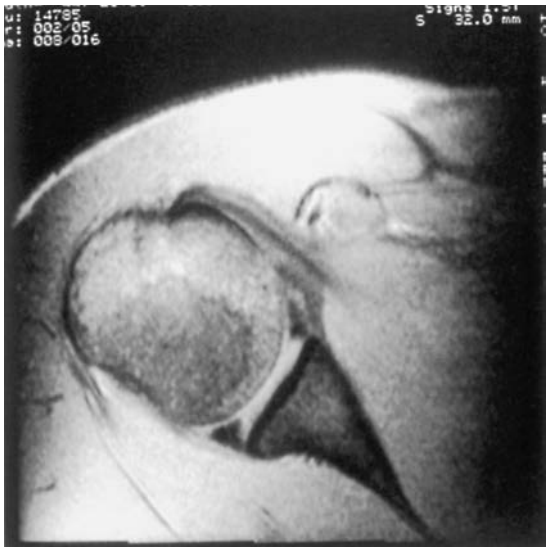

Figure 11-31 Magnetic resonance imaging demonstrating the posterior capsule insertion on the labrum and not the glenoid.

Congenital and age-related variations in the anterior labrum have also been noted. Because of the variability in the appearance of normal glenoid labra, an MRI scan may overestimate the frequency of labral tears.

Complex labral tears are easier to detect. One can often see fragments that have migrated into the axillary pouch or cysts associated with labral tears. Gusmer et al.[36] found noncontrast MRI to be 95% accurate in detecting labral tears. An MRI scan can also show capsular stripping from the glenoid, rotator cuff tears, muscle atrophy caused by suprascapular and axillary nerve injury, and osseous injuries such as Hill-Sachs lesions. Focal thinning of the articular cartilage of the glenoid may indicate areas of recurrent instability.

The posterior capsule inserts directly onto the posterior glenoid labrum, not the bony glenoid (Fig. 11-31). This anatomic relation of the capsulolabral complex often confuses accurate interpretation of MR images. The capsulolabral complex is best demonstrated with the arm in neutral position. Imaging in external rotation can simulate a labral tear by creating posterior capsular redundancy at the capsulolabral junction.[120] Additionally, in the nondistended joint, the close proximity of the glenohumeral ligaments to the posterior glenoid labrum can be mistaken for a labral tear.[65,79]

MR Arthrography

While MRI is considered superior to CT arthrography, several studies have shown MR arthrography to more consistently diagnose instability lesions. Chandnani et al. found MR arthrography to be superior to conventional MR and CT arthrography at detecting labral tears, detached labral fragments, and labral degeneration.[8] Flannigan et al.[22] believed that the presence of intraarticular contrast affords improved visualization of the labrum and rotator cuff and enhances the accuracy of MRI for the detection of labral and rotator cuff tears. Palmer and Caslowitz[91] evaluated MR arthrography and found this method to have 92% sensitivity and 92% specificity for the detection of labral tears. One pitfall of MR arthrography is that the presence of intraarticular contrast may lift the meniscoid superior labrum from the articular margin and simulate a labral detachment.[67] Normal anatomic structures adjacent to the labrum, such as the glenohumeral ligaments and glenoid articular cartilage, may be misinterpreted as labral tears.[67]

Recently, MR arthrograms have been performed by placing the arm in abduction and external rotation during imaging.[14] In this position, tension is placed on the inferior glenohumeral ligament, allowing for increased accuracy in the diagnosis of nondisplaced tears. This technique has been reported to demonstrate a sensitivity and specificity of greater than 95% for anterior labral tears.[14]

MR arthrography is able to detect variations of the capsulolabral pathology, which include the anterior labroligamentous periosteal sleeve avulsion lesion (ALPSA), glenolabral articular disruption (GLAD) lesions,[107] humeral avulsion of the glenohumeral ligament, humeral avulsion of the posterior band of the inferior glenohumeral ligament,[10] and floating anterior inferior glenohumeral ligament.[47]

The use of MR arthrography for multidirectional instability provides the joint distention necessary to evaluate the capsular volume and the uncommon labral or capsular detachments.

As noted by Oxner,[89] the clinical use of MRI in managing shoulder instability is seen best in four examples: making the difficult diagnosis, timing of treatment in the professional athlete, choosing the most appropriate surgical mode, and identifying unsuspected extraarticular pathology. As noted by Mok et al.,[74] patients with subtle instability and pain without dislocation often present with a diagnostic dilemma. MRI may help define the underlying pathology. In the treatment of professional athletes, choosing the timing of operative intervention after dislocation may be influenced by the identification of additional pathology on MRI (i.e., tears of the subscapularis tendon). MRI may help to better define the pathology and potentially influence the choice between arthroscopic and open management. Sher et al. noted that MRI examination impacted clinical decision making in 29% of patients with glenohumeral instability.[113]

Authors Preferred Technique

We prefer to obtain a series of plain films that include AP, Grashey, scapulolateral Y, and axillary or Velpeau views. We use CT of the shoulder to evaluate the glenoid rim and extent of a Hill-Sachs deformity and use MRI only if there is a question of rotator cuff pathology (Fig. 11-32).

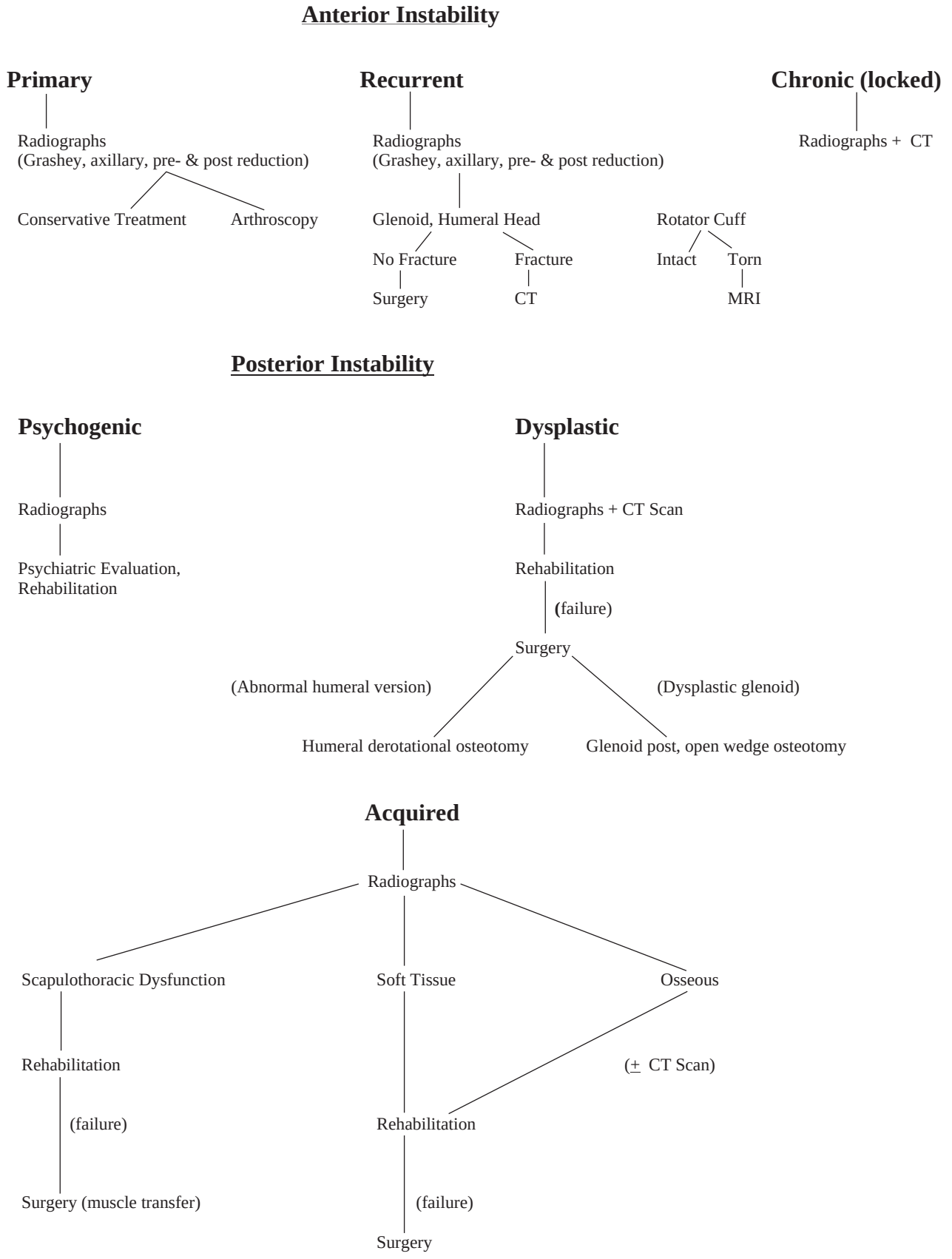

Figure 11-32 Diagnostic algorithms. CT, computed tomography; MRI, magnetic resonance imaging.

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12 Anterior and Anteroinferior Instability: Open and Arthroscopic Management

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INTRODUCTION

An understanding of the pathomechanics and treatment of the unstable shoulder has evolved over the past 100 years. The current belief that multiple mechanisms and anatomic variations combine to create an unstable shoulder has evolved from the original theory of Perthes[108] and Bankart[11] that a single "essential lesion" creates instability. Etiologic theories continue to develop based on the advances in understanding of the disease process. The manifestations of instability will vary based on host factors and the magnitude of force required to create the injury. A traumatic force creating an initial dislocation is the most frequent cause of anterior instability. More subtle events may create a spectrum of instability, such as subluxations in patients with increased capsular laxity and microtrauma secondary to repetitive large forces (e.g., in overhead-throwing athletes or in swimmers). The diagnosis and management of anterior shoulder instability should be individualized based on cause, various host factors, and associated pathology. This chapter presents an algorithmic approach to the treatment of anterior shoulder instability based on the natural history and pathology of the condition.

The clinician should distinguish between subluxation and dislocation events to help identify the severity of the event and determine prognosis. For the purposes of this chapter, subluxation is defined as a pathologic increase in glenohumeral translation without loss of articular contact

between the humerus and the glenoid; dislocation is the complete loss of contact between the articular surfaces.

CLASSIFICATION AND PATHOLOGY

Many classification systems have been proposed and exist. As the understanding of the natural history, pathology, and treatment of shoulder instability has evolved, so have the classification schemes. Thomas and Matsen[131] have described the acronyms TUBS and AMBRI to classify most patients with instability. TUBS refers to patients with a *t*raumatic lesion who have a *u*nidirectional component to their disease that frequently has a *B*ankart lesion and responds well to *s*urgery. AMBRI refers to patients who have an *a*traumatic cause of their *m*ultidirectional disease that often has *b*ilateral shoulder findings and responds to *r*ehabilitation. Rockwood[111] described four patterns of instability: type I, traumatic subluxation without previous dislocation; type II, traumatic subluxation after a previous dislocation; type IIIA, voluntary subluxation in patients with psychiatric problems; type IIIB, voluntary subluxation in patients without psychiatric problems; and type IV, atraumatic involuntary subluxation. O'Brien et al. have proposed classification based on some combination of degree, frequency, direction, and cause.[104] Gerber and Nyffler have proposed a classification system in which instability is static (class A), dynamic (class B), or voluntary (class C). Class A is further subdivided by direction of instability and class B by the presence or absence of hyperlaxity. The key point in this system is to differentiate hyperlaxity from instability, which will assist in treatment strategies. The general principle is that hyperlaxity is not pathologic, but may be a risk factor for shoulder problems and may be present in combination with pathologic instability.[38] The authors prefer to use an algorithmic approach (Fig. 12-1) to classify anterior instability based on cause, direction, frequency, and volition.

Although trauma is the most commonly identified cause of anterior instability, more subtle factors may exist in patients with anterior subluxation and multidirectional instability. Patients who are classified as having multidirectional instability by definition demonstrate a component of inferior instability. Multidirectional instability does not usually exist in all planes, but it commonly exists in two. For a diagnosis of instability, patients must have symptoms that accompany increased translation.

Acute Traumatic Anterior Dislocation

The most common mechanism of a traumatic initial anterior dislocation is an indirect force with the arm in the externally rotated abducted position. The force levers the humeral head anterior in relation to the glenoid, as when the patient's arm is in the throwing position. The inferior glenohumeral ligament is the main stabilizing force resisting anterior dislocation.[135] This structure is commonly injured in the acute traumatic anterior shoulder dislocation. The patient's age and the force required to create an initial dislocation are important factors in determining prognosis and associated injuries.

The pathology associated with an acute anterior shoulder dislocation in young patients has been well documented.[8,130] Taylor and Arciero[130] examined 63 shoulders with an average age of 19.6 years arthroscopically within 10 days of an acute traumatic dislocation and found complete detachment of the capsuloligamentous complex from the anterior–inferior glenoid rim and neck in 97%. This lesion (Bankart lesion) includes the inferior glenohumeral ligament and the anterior labrum with its periosteal insertion on the glenoid neck.[8,10,117] Ninety percent of patients had a Hill-Sachs lesion and 10% had a superior labrum anterior posterior (SLAP) lesion. No rotator cuff tears were observed in this patient population. Baker et al.[8] found similar pathology in a study of 45 shoulders. They found complete labral and capsular detachment in 62% and capsular tears with partial labral tears in an additional 24%. Hill-Sachs lesions were present in 18 shoulders and rotator cuff tears were found in five shoulders.

The pathology associated with an acute traumatic dislocation in young patients is manifested clinically as recurrent instability being either dislocations or subluxations. Multiple studies have looked at recurrence in this population, documenting recurrence rates between 33%[52] and 90%.[4,48,130,140]

Patients older than 50 years of age are more likely to suffer rotator cuff tears with the initial dislocation. In older patients, the inability to lift the arm over the head is more likely to be related to a rotator cuff tear than to an axillary nerve palsy, whereas the opposite is true in the younger population. Neviaser et al.[101] noted an 85.7% incidence of rotator cuff tears and a 10.8% incidence of axillary nerve palsy in patients with initial traumatic shoulder dislocation occurring after age 40.

Anterior shoulder dislocation is diagnosed on the basis of the physical and radiographic examinations. The patient may present with an adducted and internally rotated arm and may be unable to externally rotate or fully abduct the arm. The humeral head may be visible and palpated on the anterior aspect of the shoulder; whereas the posterior aspect may appear hollow and the acromion stepped off.

Anterior Subluxation

In patients with anterior subluxation, it may be difficult to identify an initial traumatic event. Rowe and Zarins[116] described recurrent transient subluxation of the shoulder, causing a "dead arm syndrome." Alternatively patients may feel their shoulder slide in and out of the joint, may

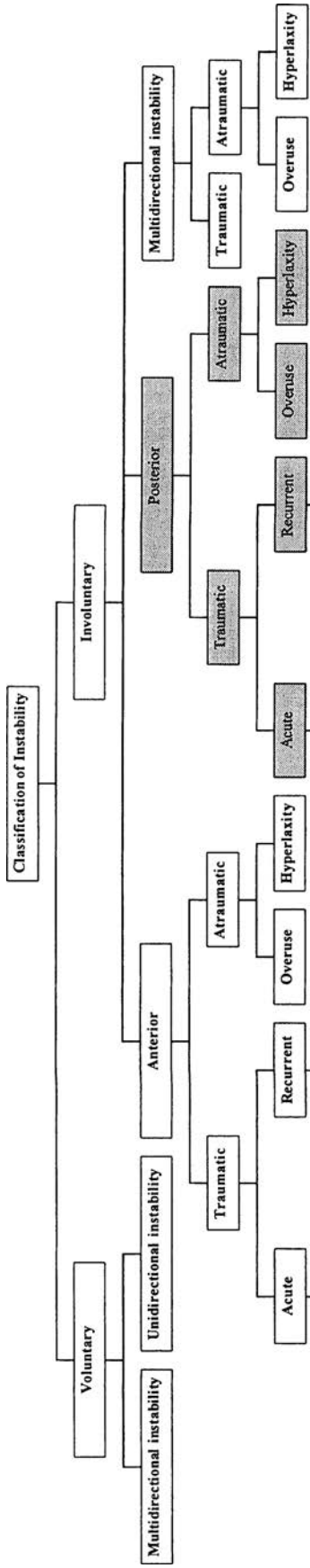


Figure 12-1 Classification of instability. (Modified from Silliman JF, Hawkins RJ. Classification and physical diagnosis of instability of the shoulder. *Clin Orthop* 1993;291:7–19.)

complain of clicking or catching in provocative positions, or may simply have pain.

The Overhead Athlete

The shoulders of baseball pitchers or other overhead athletes are exposed to high-energy repetitive stresses or overuse that can exceed the rate of tissue repair[74] or cause structural pathology. The static stabilizers, which include the bony geometry, labrum, capsule, and glenohumeral ligaments,[20] can be damaged and, in turn, place an increased amount of responsibility for glenohumeral stability on the dynamic stabilizers. These dynamic stabilizers include the deltoid, the biceps, and the rotator cuff muscles.[20] As the soft tissue static stabilizers become more attenuated and more stress is placed on the dynamic stabilizers, they may become fatigued. As the compensatory mechanism becomes overloaded, anterior subluxation may occur. As the humeral head subluxes anteriorly and contacts the coracoacromial arch, subacromial impingement occurs. Over time, anterior laxity with the arm in abduction and external rotation can occur. This anterior translation has been shown radiographically in patients with symptomatic anterior instability.[57] With anterior subluxation, the tendinous portion of the supraspinatus and infraspinatus may impinge on the posterosuperior border of the glenoid rim, resulting in glenoid impingement as well. This so-called "internal impingement" was suggested by Bennett and others[13,81] and is now well described.[27,64,93,94] Swimmers, volleyball players, and other overhead-throwing athletes are vulnerable to this pathophysiology.

Occult instability may also be present in nonthrowing athletes. For weight lifters,[43] anterior subluxation may be the primary etiologic factor of shoulder dysfunction and pain. Weight-lifting maneuvers that produce forced abduction, extension, and external rotation (such as military presses, flies, and latissimus pull-downs) may cause subluxation.

Glenoid Bone Loss

Glenoid bone deficiency related to shoulder instability has been described by various authors. The injury can occur as an associated anteroinferior glenoid fracture attached to the inferior glenohumeral ligament or can be related to continued episodes of dislocation or subluxation and eburnation or rounding off of the anterior glenoid. Bone defects of the glenoid have been documented and studied radiographically. Edwards and associates noted that with examination by fluoroscopically guided radiography, 79% of shoulders with recurrent dislocations had an osseous lesion of the glenoid.[33] Three-dimensional computed tomography (CT) has been used in the assessment of glenoid bone loss in recurrent instability. In a study of 100 shoulders with recurrent glenohumeral instability, 50% of patients had a bony fragment and an additional 40% had bone loss from erosion or compression.[128] A separate study using CT evaluation documented osseous lesions in 91% of recurrent dislocations and suggested quantification of moderate to severe bone loss using the contralateral, unaffected shoulder to determine a difference in the maximum glenoid width.[42] These high rates of glenoid bone defects have raised concerns about the associated bone loss as a negative prognostic factor resulting in higher failure rates and perhaps the necessity for alternative treatment plans to standard soft tissue procedures.

Burkhart et al.[22] proposed an arthroscopic quantification of this glenoid bone loss. The method involves locating the bare spot in the glenoid, which they showed to be reliably equidistant from the anterior and posterior rims of the inferior circular portion of the articulating glenoid. Significant bone loss is that amount which increases the risk of recurrence following a soft tissue–only repair. They propose that if the bare spot–anterior rim distance is less than half that of the bare spot to the posterior rim, then the shoulder is at high risk for failure of soft tissue repairs only as treatment for their anterior instability (Fig. 12-2). Arthroscopically, the appearance of the glenoid is that of an "inverted pear" (Fig. 12-3). Burkhart et al. have documented a 61% recurrence rate following arthroscopic Bankart repairs when the glenoid bone loss caused an "inverted pear" appearance. The authors noted that a loss of greater than 25% of the diameter of the inferior glenoid will create the "inverted pear" appearance.[21,22] They recommended treatment of this bone defect as well as the soft tissues when glenoid bone loss of this magnitude is seen.

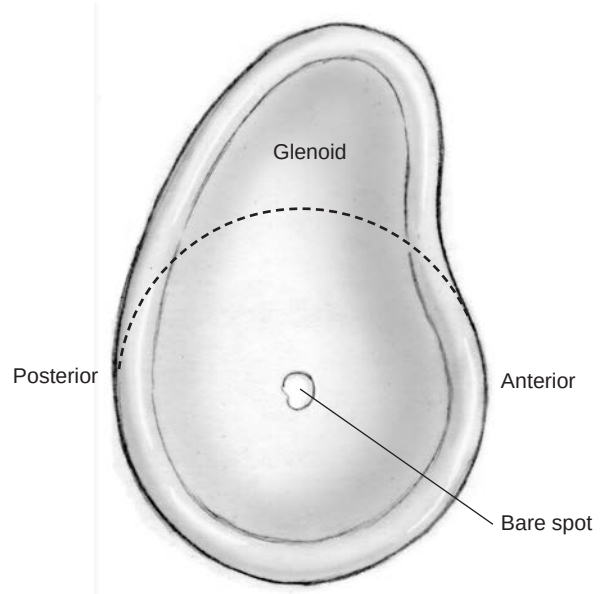


Figure 12-2 Glenoid morphology. (Modified from Burkhart SS, Debeer JF, Tehrany AM, Parten PM. Quantifying glenoid bone loss arthroscopically in shoulder instability. *Arthroscopy* 2002;18[5]: 488–491.)

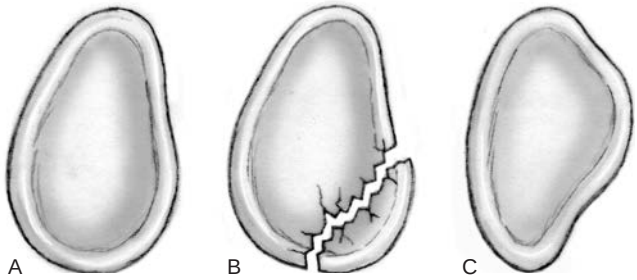


Figure 12-3 **(A)** Normal glenoid morphology. **(B)** Glenoid bone loss. **(C)** Inverted pear glenoid with glenoid bone loss. (Modified from Burkhart SS, De Beer JF. Traumatic glenohumeral bone defects and their relationship to failure of arthroscopic Bankart repairs: significance of the inverted-pear glenoid and the humeral engaging Hill-Sachs lesion. *Arthroscopy* 2000;16[7]:677–694.21)

Bigliani et al. also recommended that bone loss of greater than 25% of the anterior–posterior diameter should be treated with coracoid transfer.[15] In a cadaver study, Itoi and associates[62] simulated Bankart lesions with varying degrees of glenoid bone loss. They examined anterior–inferior stability with and without soft tissue repair. After soft tissue Bankart repair, a decreased force was required to cause instability when the width of the defect was 21% of the superior–inferior glenoid length. In a separate radiographic study, Griffith et al.[42] propose 1.5 cm as a critical length of the lesion.

Humeral Bone Loss

A posterior–superior humeral head impaction fracture was originally described by Flower in 1861.[36] In 1940, Hill and Sachs described the mechanism by which these lesions occur, that is, compression of the humeral head against the anterior glenoid rim.[50] These so-called Hill-Sachs lesions are common in glenohumeral instability;[33,52] however, their quantification is not well described. Burkhart and De Beers described "engaging Hill-Sachs lesions" in which the long axis of the grooved humeral head defect is parallel to the anterior rim of the glenoid when the shoulder is in the position of abduction and external rotation.[21] They suggested that these lesions contributed to failure of arthroscopic Bankart repair, which is consistent with others who advocate that Hill-Sachs lesions contribute to recurrent instability.[52,117] Miniaci[97] has described a group of patients with Hill-Sachs lesions larger than 25% of the humeral articulating surface with or without associated glenoid bone loss that may require attention to the humeral head defect. These patients commonly demonstrate significant apprehension even at levels of abduction below 70 degrees. The usual history is one of multiple traumatic dislocations or failed repairs, often associated with contact sports, and can be present in patients with seizure disorders and anterior instability. These patients often have reduced external rotation and significant apprehension at low levels of abduction, and may have failed previous surgical attempts at repair. Because the large humeral defect alters joint stability, it may need surgical attention in certain cases. The surgical management of those soft tissue and bone lesions that contribute to recurrent instability is discussed later in this chapter.

MANAGEMENT

The goal of management of anterior shoulder instability is to return the patient as rapidly as possible to his or her preinjury level of activity. The first step toward this goal is adequate, prompt, and atraumatic reduction of an anteriorly dislocated shoulder. Decisions (often individualized) then need to be made for immobilization and rehabilitation (Fig. 12-4).

Once the diagnosis is made, the glenohumeral joint should be reduced as rapidly and gently as possible. If diagnosed in the field, a reduction maneuver may be attempted immediately before spasm of the shoulder muscles has occurred. A neurovascular examination should be performed before and after the reduction. When attempting such a reduction in the field without sedation, we prefer to place the patient's shoulder in a small amount of forward flexion and abduction and then slowly internally rotate the arm. If this does not achieve the desired results, the patient should be transported to an emergency room where reduction under sedation may be performed.

Many reduction maneuvers have been successfully performed with appropriate muscle relaxation. One technique is that of the traction and countertraction method described by Matsen et al.[86] (Fig. 12-5). In this method, the patient lies supine with a sheet passed around his or her thorax and around the assistant's waist. The assistant should be standing on the unaffected side of the patient to provide a countertraction force. The surgeon should be standing on the affected side of the patient with a second sheet looped around his or her waist and the patient's forearm. The surgeon leans back and applies steady, gradually increasing traction while grasping the forearm at the same time as the assistant provides countertraction around the patient's thorax. The surgeon may be required to gently rotate the arm internally and externally or to attempt to apply gentle outward pressure to the humeral head with his or her hand in the axilla.

In Stimson's technique, the patient is placed in the prone position on the edge of the examining table, with the affected arm hanging over the side. If the weight of the arm is not sufficient to reduce the shoulder, gentle downward traction is applied with an appropriate weight (usually 5 lb or less) attached to the wrist. With this technique, reduction may take 15 to 20 minutes[86] (Fig. 12-6).

The Milch technique involves placing the patient's affected extremity in an overhead position. This can be

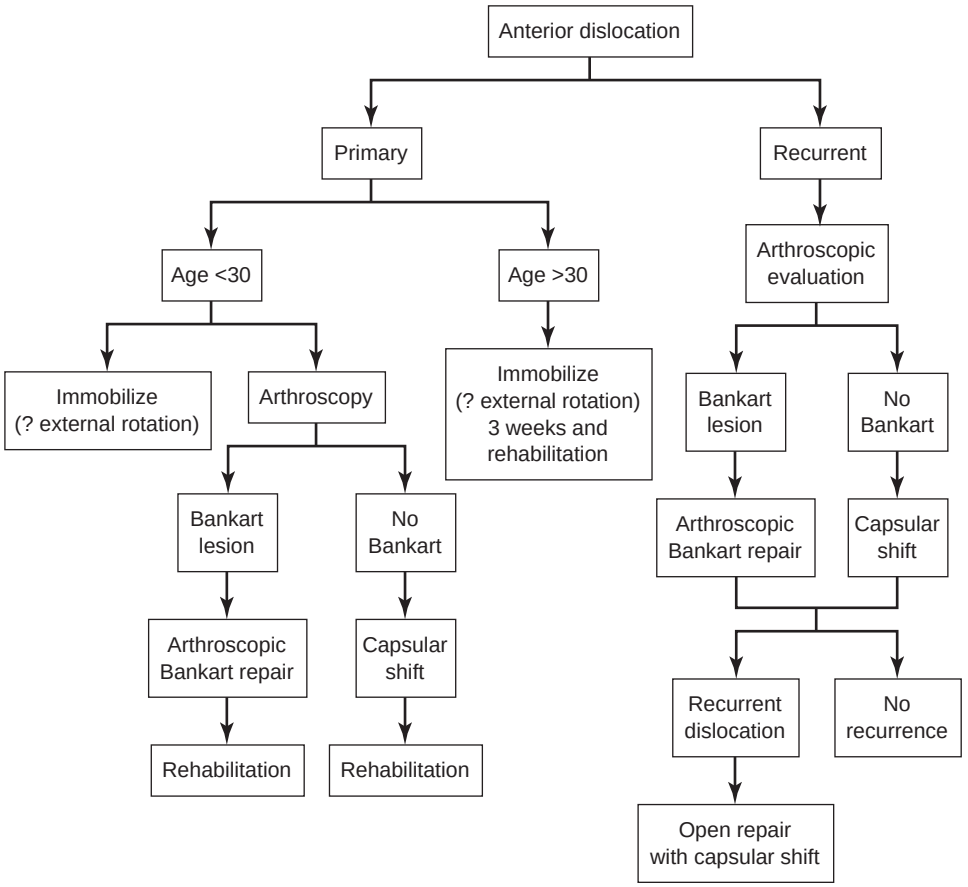


Figure 12-4 Algorithm for treatment.

achieved slowly with little discomfort. With the arm overhead and fully abducted at the shoulder, gentle traction and external rotation is applied. Gradual adduction and pressure applied to the humeral head are helpful maneuvers if reduction is not achieved initially.[136]

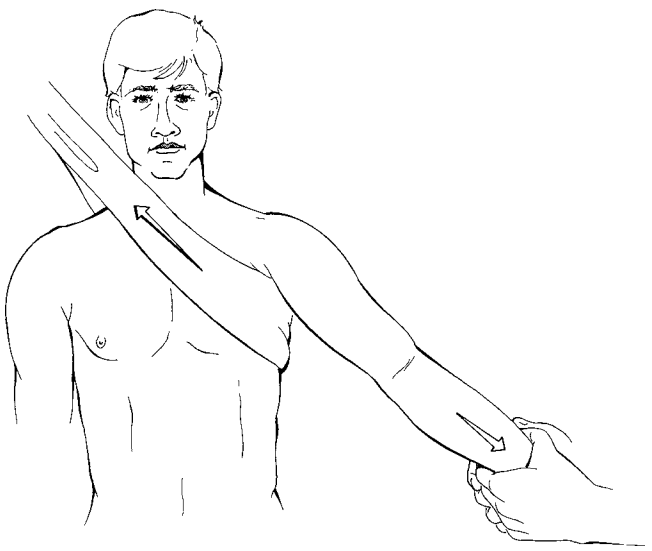


Figure 12-5 Reduction technique using the traction–countertraction method.

Reduction by scapular manipulation is based on the principle of repositioning of the glenoid fossa. The patient may be prone or seated with the scapula exposed. Longitudinal traction is placed on the extremity with the shoulder at 90 degrees of forward flexion. The superior aspect of the scapula is stabilized while adducting the tip of the scapula, effectively moving the glenoid toward the anterior and medial location of the dislocated humeral head.[136]

The Spaso technique is performed with the patient in the supine position. The affected extremity is held by the wrist and gentle traction is applied toward the ceiling. While applying traction, the extremity is externally rotated. This technique may take several minutes; however, it is generally well tolerated by the patient.[136]

Nonoperative Indications and Management

Nonoperative management, although controversial, begins with shoulder immobilization (the duration and position of which is controversial), followed by physical therapy and (occasionally) bracing. Indications for nonoperative treatment include atraumatic instability, voluntary instability, children with instability, and selected athletes whose sport requires supraphysiologic motion at the glenohumeral joint.[86]

Figure 12-6 Stimson's technique for closed reduction of dislocation

Immobilization

Shoulder instability in athletes has been managed by brace treatment, but with unclear results. The purpose of bracing is to prevent abduction and external rotation, thereby avoiding the position that places the patient at risk for dislocation. In sports such as basketball, baseball, and volleyball, this is nearly impossible because of the shoulder mobility required. Football players may have varying results based on the field position. Wide receivers, quarterbacks, and defensive backs require more shoulder motion than these braces usually will allow. However, braces have been used successfully for football linemen, hockey players, and wrestlers, where the motions of extreme abduction and external rotation are not necessary for participation in the sport. Restriction of motion is adjusted by a trainer to each individual athlete's needs to prevent subluxation and dislocation. Data available on the usefulness of these braces are largely anecdotal, and no biomechanical data exist, despite manufacturer's claims of success.

Controversy remains over the role of traditional sling immobilization (arm in internal rotation) after first-time anterior shoulder dislocations and this treatment's ability to affect the rate of recurrence. Available data on sling immobilization also fail to provide conclusive evidence of the appropriate length of treatment.

Hovelius et al.[53] prospectively followed 257 patients (all younger than 40 years) after first dislocation. Approximately half were treated with immobilization for 3 to 4 weeks; the others were treated with early range of motion. At the 2-year follow-up, recurrence rates were similar, with the highest recurrence rates seen in the younger patients, regardless of treatment. Yoneda et al.[145] reported the results of a more prolonged period of immobilization (5 weeks) and limited range of motion for an additional 6 weeks in a young, athletic population. At 13 years follow-up, 17.3% had had a recurrence; 13.5% of those required surgical repair. Kiviluoto et al.[72] showed a higher incidence of recurrence among patients younger than 30 years and, in this younger group, there was a higher incidence in those immobilized 1 week compared with those immobilized for 3 weeks. These authors recommended longer immobilization (3 weeks) for patients younger than 30 years old and shorter immobilization (1 week) for patients older than 30 years.

A young, athletic population when treated with sling immobilization can have recurrence rates as high as 90%.[4,48,130,140] However, in an older population, recurrence is much less of a problem, with recurrence rates documented as low as 10% in patients over 40 years of age.[92] Rowe reported a recurrence rate of 14% in his series of patients over 40 years old.[113]

Recent studies show decreased recurrence rates with arthroscopic stabilization after an initial, traumatic, anterior glenohumeral dislocation when compared to immobilization.[18,70] However, these studies compared arthroscopic stabilization to traditional immobilization in internal rotation followed by rehabilitation.

Recent work by Itoi et al. has provided data from a cadaveric study,[61] a magnetic resonance imaging (MRI) study,[63] and a preliminary clinical study[60] to support immobilization in external rotation. The rationale for this theory is that in external rotation, the Bankart lesion is reapproximated to its correct anatomic position on the glenoid, thereby allowing healing of this pathology in its anatomic position. The cadaveric study showed that a simulated Bankart lesion is approximated to the glenoid throughout an arc of motion from full internal rotation to 30 degrees of external rotation. The MRI study revealed that a Bankart lesion secondary to an acute traumatic dislocation was reduced better in external rotation than in internal rotation. The most recent data is a clinical study in which 40 patients were treated with immobilization. Twenty patients were treated with traditional immobilization in internal rotation and 20 were treated with immobilization in external rotation for 3 weeks following shoulder dislocation. At an average follow-up of 15.5 months, the

traditional immobilization group had a recurrence rate of 30% and 45%, whereas the external rotation group had a 0% recurrence rate. Many studies are now ongoing evaluating this concept. If these early results can be reproduced, the treatment of first time dislocators will be altered and directed toward immobilization in external rotation.

Rehabilitation

A physical therapy program is appropriate for patients with anterior instability, regardless of the underlying pathology. The patient's ultimate goals should always be taken into account when a rehabilitation program is designed. We are uncertain whether a rehabilitation program will affect the primary outcome measure (recurrence rate) or secondary outcome measures (pain, range of motion, function). The willingness to modify one's activity, however, can significantly affect outcome. Several studies support the usefulness of early rehabilitation programs in diminishing the rate of redislocation.[4,6] When using a strict rehabilitation protocol in a population of Naval midshipmen, Aronen and Regan[6] reported a 75% success rate in preventing dislocation over a 3.5-year period. Arciero et al.[4] reported an 80% reduction rate in consistently treated West Point cadets after primary dislocation. It is uncertain what other factors were involved in the development of further instability. Hovelius[51] reported that age at time of initial dislocation was more important than rehabilitation or length of immobilization. Simonet and Cofield found that activity restriction in a group of patients with 9-year follow-up had a significant role in reducing recurrence.[121]

Individualizing a rehabilitation program should take into account several factors, including knowledge of the patient's instability pattern and associated injury pathology. Patients with multidirectional instability should be encouraged to avoid activities that stress the shoulder in an inferior direction, and those who have had severe trauma causing dislocation should avoid prolonged time in positions at risk. Patients with subluxation may require less immobilization time than those patients sustaining a severe traumatic dislocation before initiating a therapy program. As a general rule, it should be stressed that throughout the rehabilitation period, the patient should not be exercising in pain. If pain develops during a particular exercise, the patient should limit or modify the existing exercise level and avoid progressing to the next level until the pain resolves.

Attempting to restore range of motion is the initial goal in any shoulder rehabilitation program. After an acute injury and appropriate initial immobilization, modalities should be performed to decrease the amount of pain. Ice, heat, ultrasound, and electrical stimulation treatments may improve pain and swelling. Oral nonsteroidal antiinflammatory medications may help decrease inflammation. Pendulum exercises are begun as soon as the immobilization is removed.

Isometric exercises are also initiated for resisted internal rotation and resisted adduction at this time. The position of abduction is to be avoided. When the patient can perform isometric exercises without pain, he or she may advance to isotonic exercises that strengthen the stabilizers. The scapular stabilizers are strengthened to provide a stable base for humerus rotation and maintain the glenoid in a position that has maximal congruency with the humeral head. Any change in position of the scapula will produce increased stress on the rotator cuff. In addition, many overhead athletes present with anterior tilt and lateral displacement of the scapula. This produces additional stress on the anterior structures of the shoulder. Moseley et al.[99] described a series of four scapular stabilizer exercises based on electromyographic findings to strengthen the scapula-stabilizing musculature. They recommended rowing, pushups with maximal protraction, scaption (which involves elevation of the humerus in the scapular plane with the humerus externally rotated), and press-ups (which utilize the pectoralis minor). Closed-chain exercises, such as pushups, enhance joint stability by providing a compressive force across the joint and diminish tensile forces across the capsule. However, care must be taken to avoid abducting the shoulder, which places increased pressure on the anterior joint.

Muscle strengthening of the rotator cuff may help stabilize the humeral head by providing compression across the glenohumeral joint and preventing impingement. Initially, exercises for shoulder strengthening should occur in the scapular plane. This provides several advantages.[118] This position offers an optimal length–tension relation between the deltoid and rotator cuff muscles and minimizes the stress on the anterior and posterior capsule and rotator cuff.[31] The supraspinatus may be selectively strengthened in the scapular plane or by prone horizontal lifts with the shoulder slightly externally rotated. The subscapularis may be strengthened by side-lying internal rotation lifts against resistance. The isotonic program should be followed by an isokinetic strengthening program as pain allows.

Internal and external rotation exercises can be performed using Theraband tubing. When strengthening the rotator cuff, it is important to include both concentric and eccentric strength exercises.[30] Before progressing to strengthening in an overhead position, apprehension must be eliminated. Muscular balance of the rotator cuff is important in restoring dynamic stability, and all muscular stabilizers must be taught to act synchronously.

Many overhead-throwing athletes present with excessive external rotation, reduced internal rotation, and a tightened posterior capsule.[19] Posterior tightness can lead to superior migration of the humeral head and anterior instability symptoms, with secondary impingement occurring as a result. When asymptomatic, such a patient should begin a stretching program to correct posterior capsular

tightness. Gentle internal rotation stretches in 90 degrees of shoulder abduction is a more specific stretch for posterior capsular tightness, which tend to stretch the scapular muscles,[41] than cross-body adduction stretches.

The biceps brachii should be strengthened to assist in anterior stabilization, and the deltoid should be able to provide abduction. The latissimus dorsi and pectoralis major should then be strengthened because they play an important role in deceleration of the shoulder in overhead activities.

Proprioception and neuromuscular control must also be emphasized in rehabilitation for shoulder instability. Lephart et al.[78] recommended four steps for the progression of activities. First, joint position and kinesthesia is accomplished through functional arcs of abduction and external rotation. Second, dynamic joint stabilization is accomplished with a wobble board to stimulate the activation of shoulder muscle force couples. Third, reactive neuromuscular exercises in the form of open-chain plyometric exercises can then be performed. Fourth, once joint sensibility and dynamic muscular control is obtained, training for functionally specific activities may be initiated. Continued muscle strengthening as well as endurance training specific for each sport should be performed. Posterior capsular stretches should be continued. An interval training program should be initiated and continued until the patient is able to return to full function in the chosen activity.

Athletes require strong legs and trunk to provide torque to their arms. The legs and trunk are responsible for more than 50% of the kinetic energy consumed during throwing.[12,142] Therefore, any rehabilitation program involving the arm must include conditioning exercises for the legs, hips, abdominal muscles, and back muscles before the patient resumes overhead activities to diminish the amount of work required of the arms in reestablishing preinjury overhead performance.

Surgical Indications and Management

Significant advances have occurred in the arthroscopic and open surgical treatment of glenohumeral instability. These advances include basic science principles of shoulder biomechanics as well as surgical techniques. A careful clinical evaluation with consideration of indications can yield appropriate patient selection for a given procedure.

In general, indications for surgical treatment include irreducible, open, or recurrent dislocation, failed nonoperative treatment, young patient age, or failed procedures with significant glenoid or humeral bone defects. Significant defects can be described as greater than 25% of the glenoid and large, engaging Hill-Sachs lesions.[21,22,97] First-time dislocation in a young patient is a controversial indication for operative treatment; however, multiple studies have shown a high recurrence rate in young patients treated nonoperatively as well as the ability of early operative intervention to decrease these recurrence rates.[4,18,52,53,70] Whether newer techniques of immobilization in external rotation will be successful may change recent trends to early operative intervention.

Role of Surgery for Patients with Initial Dislocations

Management of initial dislocations in young, active, athletic patients has become controversial. A high incidence of recurrence in young, active, athletic patients has been previously reported.[51,121] Physical therapy alone has provided mixed results. Aronen and Regan[6] reported a 75% success rate among 20 midshipmen at the Naval Academy using a strict physical therapy regimen. Because these results have not been replicated by others, many clinicians believe that patients with initial dislocations would benefit from a more aggressive surgical approach, given the natural history and associated pathology. The high rate of redislocation that exists in patients younger than 30 years old reinforces the concept that such patients may have a different clinical course than those older than 40, who have a much lower redislocation rate. In a cadaveric study of 182 shoulders, Hertz[49] demonstrated a difference in pathology in the two age groups. He showed that identical movements producing dislocation caused different lesions. In the younger age group, there was either disruption of the labrum from the osseous margin of the scapula or splitting of the fibers near their bases. In the older age group, there were no lesions of the labrum, but ruptures of the joint capsule occurred. Increased forces were required to produce dislocations in the younger group. Hertz[49] believed that these data supported the explanation that the primary disruption of the labrum in younger patients was responsible for redislocation and that these lesions could not heal by immobilization alone. This work supported a previous cadaveric study by Reeves,[109] who showed decreased capsular tensile strength as individuals aged. This occurred while the strength of the glenoid labral attachment remained constant.

Baker et al.[8] reported arthroscopic results on the pathology of first-time dislocators younger than age 30. They examined 45 shoulders within 11 days of the initial dislocation and developed a classification system based on the pathology observed: group I, capsular tear with no labral lesion; group II, capsular tear with partial labral detachment; and group III, capsular tears with complete labral detachment. Group III patients were grossly unstable on examination under anesthesia, whereas group I patients were stable. Of the 45 patients in the study, only six (13%) had no labral lesion; 39 (87%) had Bankart lesions.

Military personnel have provided a large population for studying the results of treatment in young, active patients with first-time dislocations. Wheeler et al.[140] reported a

92% recurrence rate after initial anterior dislocation of the shoulder in West Point cadets. This occurred despite traditional nonoperative treatment programs, which included 3 weeks of immobilization and a physical therapy program that was poorly followed. Given these retrospective results, Wheeler et al. performed an arthroscopic Bankart repair in nine subsequent patients and noted labral detachment in all nine, but no interstitial damage in the inferior glenohumeral ligament (IGHL). The recurrence rate for the nine was only 22%.

Arciero et al.[4] performed a prospective study using two treatment modalities for patients with first-time dislocations. One group of patients was treated nonoperatively with immobilization for 4 weeks and subsequent rehabilitation. The second group underwent diagnostic arthroscopy, arthroscopic Bankart repair, and the same rehabilitation program as the first group. Of the 15 patients in the nonoperative group, 80% developed recurrent instability. Of the 21 patients in the operative group (all with grade III Bankart lesions), only 14% developed recurrent instability. Arciero et al. noted that after initial dislocation, one would not expect to see a degenerated or absent labrum, marked capsular attenuation, or a large Hill-Sachs lesion, which are commonly encountered in a patient with chronic instability. This led them to believe that acute dislocation provides the optimal situation for success with arthroscopic repair. Neither West Point study, however, addressed the treatment of patients with other than grade III lesions of the labrum. This is inconsistent with Baker's study, which showed that 18 of 45 patients had such lesions.

Kirkley et al.[71] demonstrated decreased recurrence rates at an average follow-up of 75 months with immediate arthroscopic stabilization. This long-term follow-up also showed that those patients treated surgically had 11% higher quality-of-life scores. While not statistically significant, the authors concluded that this small difference is likely clinically meaningful. In a previous study, Kirkley et al.[70] showed that patients treated with traditional immobilization who did not experience a dislocation still had a significant deficit in disease-specific quality-of-life scores.

At present, the concept of immobilization is radically changing based on Itoi et al.[63] observations and recommendations that shoulders be immobilized in external rotation. Until we evaluate this concept, it will be very difficult to make definitive recommendations regarding a surgical versus a nonsurgical approach in a first-time dislocator.

Arthroscopic Surgical Treatment

The role of arthroscopy in the treatment of anterior shoulder instability has continued to evolve. Although there are no clearly defined indications for arthroscopic repair of anterior instability lesions, the ideal patient is a noncontact-sport athlete with anterior instability secondary to a traumatic injury, with a thick mobile Bankart lesion and little or no discernible capsular laxity.

Historically, the rate of recurrence reported with open surgical procedures has been less than 10% (Table 12-1).[41] However, one should also consider that many of these procedures resulted in significant reduction of shoulder motion helping prevent further instability. This reduced range of motion is not without consequences and therefore, some of the secondary outcome measures will need to be evaluated before we can truly determine the benefits of one technique over another. Nevertheless, arthroscopic repair has several theoretical advantages compared with open procedures, including minimizing surgical dissection, damage to surrounding tissues, and scarring. These factors may allow more rapid rehabilitation and improved range of motion. The arthroscope also allows improved visualization of the capsulolabral complex as well as other articular lesions[124] and may be associated with less morbidity than open techniques. Theoretically, increased scarring associated with open surgery may contribute to postoperative loss of motion. Limited external rotation after anterior shoulder surgery may be caused by loss of normal elasticity of the shoulder or by overtightening of the anterior capsule and subscapularis. Unlike some open procedures, all arthroscopic methods address the pathology of the anterior capsulolabral complex. However, the technical manner by which the anterior capsulolabral complex is reattached to the glenoid rim and how much the anterior capsule is advanced also differs between arthroscopic and open procedures. One potential disadvantage of the arthroscopic approach is that it may not afford as precise a repair of the attenuated anterior aspect of the capsule as the open approach.

Early reports of arthroscopic capsulorrhaphy involved the use of metal staples.[29,87] Significant complications of loose

TABLE 12-1
SUMMARY OF RECURRENCE RATES CUMULATED FROM 50 PUBLISHED SERIES

Type of Procedure	Recurrence Rate (%)
Putti-Platt	3.0
Magnuson-Stack	4.1
Eden-Hybbinette	6.0
Gallie	2.9
DuTolt and Roux	2.0
Bristow-Laterjet	1.7
Bankart	3.3
Anterior capsulolabral reconstruction	3.4

From Griffin LY. Chronic shoulder problems in athletes. In: *Orthopaedic knowledge update: sports medicine.* Rosemont, IL: American Academy of Orthopaedic Surgeons, 1994:153–163, with permission.

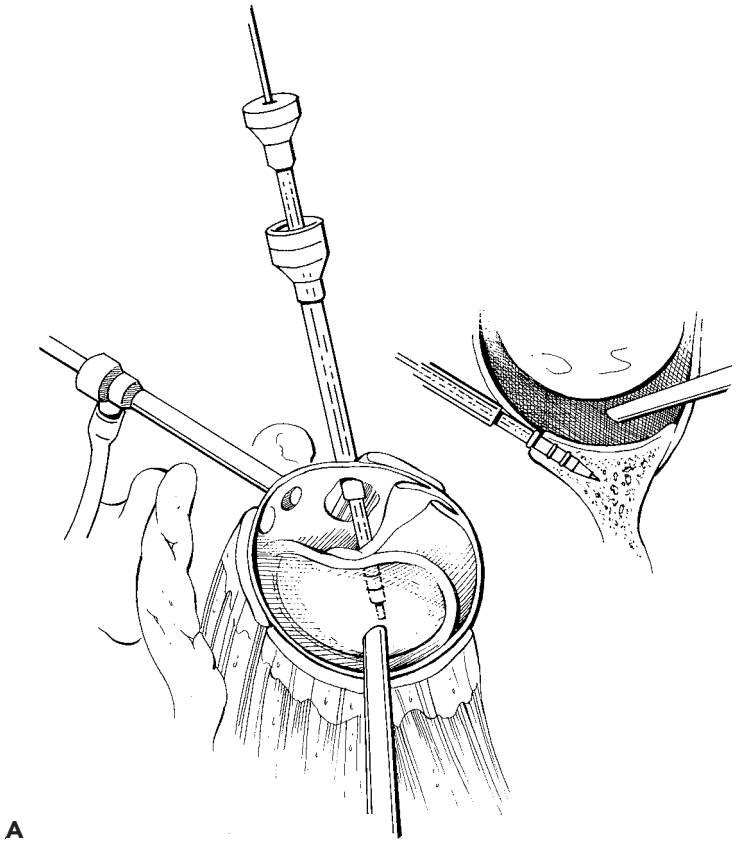
or painful hardware, articular cartilage injury, and recurrent instability led to the introduction of transglenoid sutures[90,98] (Fig. 12-7). This method was technically difficult for most surgeons. Fixation was dependant on tying of knots over the posterior fascia and was implicated as a possible cause for variable rates of recurrence. Some have shown success rates as high as 90%,[133] but other long-term follow-up showed unacceptable recurrence rates.[40] Risk of suprascapular nerve injury was also present with this technique. The most recent literature describes stabilization with bioabsorbable tacks, suture anchors, thermal capsulorrhaphy, and capsule-ligament suture plication.

Bankart Repair

Arthroscopic repair of Bankart lesions continues to evolve and reports of the effectiveness of this procedure continue to increase in the literature. A common theme in many of the reports is appropriate patient selection. Relative contraindications to arthroscopic repair include significant glenohumeral bone defects, humeral avulsions of the glenohumeral ligaments, and capsular insufficiency.[5,127] Some would add collision athletes to this list, although no study exists to show that the technique and not the pathology is the important factor in high recurrence rates. Each individual patient's mechanism of injury, expected postoperative activity level, and personal preferences should also be considered.

Repair of Bankart lesions using cannulated bioabsorbable tacks was reported by Speer and associates in 1996.[125] These barbed tacks are used as a transfixing anchor placed through the capsulolabral tissue (Fig. 12-8). Documentation on the effectiveness of bioabsorbable tacks has come mostly from studies comparing arthroscopic to open stabilization. Karlsson and associates[67] reported on 117 cases, 66 of which were arthroscopic. They showed a recurrence rate of 15% in the arthroscopic group and 10% in the open group. Rowe and Constant scores showed no significant differences between the two groups. Sperber et al.[126] reported on 56 patients, 30 treated arthroscopically and 26 treated with an open procedure. The recurrence rate in the arthroscopic group was 23% and in the open group was 12%.

Figure 12-7 Transglenoid suture technique of arthroscopic stabilization. (From Pagnani M, Warren RF. Arthroscopic shoulder stabilization. *Oper Tech Sports Med* 1993;1:276–284, with permission.)



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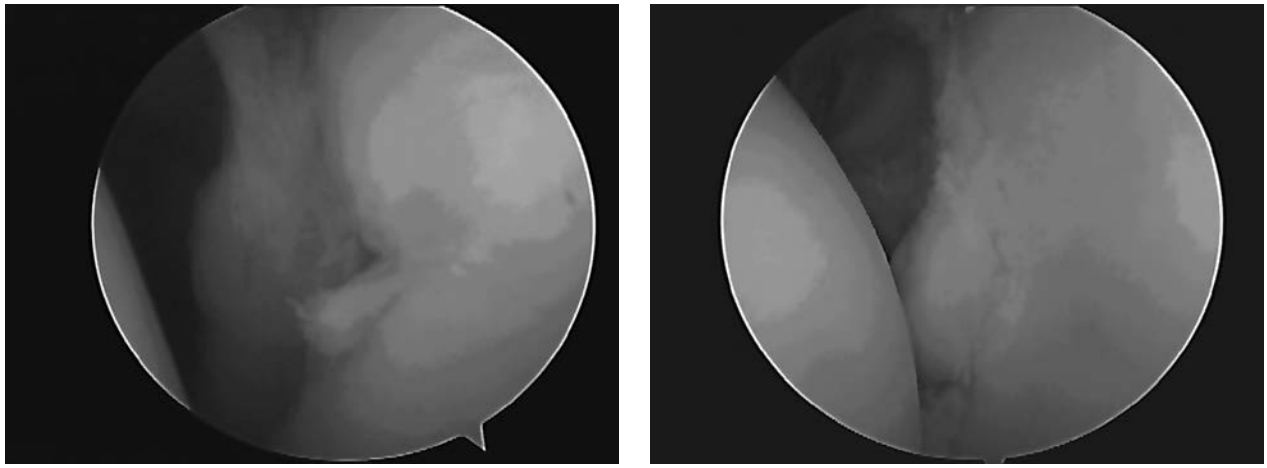

B

Figure 12-8 **(A)** Arthroscopic procedure for repair of Bankart lesion using bioabsorbable tack. **(B)** Arthroscopic view of an absorbable tack stabilization of an anterior labrum tear (Bankart lesion). (Panel A from Speer KP, Warren RF, Pagnani M, Warner JJ. An arthroscopic technique for anterior stabilization of the shoulder with a bioabsorbable tack. *J Bone Joint Surg Am* 1996;78(12): 1801–1807, with permission.)

Several studies illustrate the importance of patient selection in arthroscopic Bankart repair with bioabsorbable tack fixation. Aciero et al.[3] and DeBerardino et al.[28] described arthroscopic stabilization for initial anterior dislocation in a young, military patient population and showed recurrence rates to be comparable to documented recurrence rates of open procedures. Laurencin et al. also showed a low recurrence rate of 10% when indications were limited to traumatic, anterior instability with an isolated labral lesion and no significant bone defects.[76] Cole and associates[24] performed a study on 63 patients with recurrent instability. The patients were divided into arthroscopic or open groups based on findings during examination under anesthesia and diagnostic arthroscopy. Thirty-nine

patients with only a labral lesion and anterior instability were treated with bioabsorbable tack fixation. Twenty-four patients were found to have anterior–inferior instability and capsulolabral laxity and underwent open capsular shift. No significant differences were found between the two groups at mean follow-up of 54 months. These results demonstrate, again, the importance of patient selection when using bioabsorbable tacks. The advantages of this technique include decreased operative time and that it is technically easier than intraarticular knot tying. The disadvantages of this method of stabilization are no proven effective ability to address capsular laxity and the risk of synovial reaction, which has been reported in a few small reports[25,137] but never proven as a general problem.

Arthroscopic Bankart repair with suture anchors was reported in 1991[139] and has since been described by Wolf[144] using absorbable sutures and by Synder[123] using nonabsorbable sutures. Many studies have documented recurrence rates using suture anchors (Table 12-2). The differences in the definition of recurrence or failure make absolute comparisons among studies difficult. However, this has also been the case historically and therefore the trend in these studies toward a recurrence rate of less than 10% may indicate an improvement over previously documented recurrence rates of 15% to 33% for arthroscopic repair.[24,44,67,126]

Kim et al.[69] reported on 167 patients treated with arthroscopic stabilization with suture anchors. Mean follow-up was 44 months. They found 95% good to excellent results according to the Rowe scale and a recurrence rate of 4%. Ninety-one percent of patients returned to their preinjury level of activity. Fabbriciani et al.[35] recently described a comparative study on arthroscopic versus open treatment of Bankart lesions with 60 patients, 30 in each group. No recurrences were noted in either group at 2-year follow-up. The only significant difference was the mean value for range of motion (ROM) using the Constant score. The open group had a lower ROM score. The authors concluded that both procedures were effective and that the open procedure negatively affects postoperative ROM in the treatment of an isolated Bankart lesion. These recent studies continue to build a body of evidence that supports the theory that arthroscopic Bankart repair is an effective procedure and comparable to open stabilization.

The appropriate procedure (arthroscopic vs. open) for glenohumeral instability in the contact or collision athlete continues to be controversial. Unacceptable rates of recurrence have been seen with arthroscopic stabilization,[73] and Pagnani and Dome[107] reported on 58 professional American football players treated with open stabilization with suture anchors and had only two postoperative subluxations. They concluded that open stabilization was the procedure of choice in contact or collision athletes. Two recent studies have looked at arthroscopic treatment in athletes. Mazzocca et al.[89] reported on 18 collision or contact athletes that were treated with suture anchor, capsulorrhaphy, and adjunct thermal treatment of the capsule and rotator interval closure as needed. The recurrence rate was 11% (two recurrences). All patients returned to sport and only one of the two recurrences occurred during a collision sport. Ide et al.[59] reported on 55 athletes, 21 of whom were contact athletes, treated with arthroscopic stabilization with suture anchors. Overall recurrence rate was 7%, and there was no statistically significant difference between contact and noncontact athletes. The authors concluded that arthroscopic stabilization was a reliable procedure in this patient population. Further prospective randomized clinical trials comparing open and arthroscopic stabilization in this patient population are needed before any definite conclusions will be evident. However, the burden of proof lies with the arthroscopic procedures, as the open stabilization remains the gold standard.

TABLE 12-2
ARTHROSCOPIC RECONSTRUCTION USING SUTURE ANCHORS

Authors	No. of Patients	Mean Follow-up (months)	Recurrence (%)	Comments
Bacilla et al.[7]	40	30	7	High-demand patients
Gartsman et al.[37]	53	33	8	Adjunct thermal rx
Cole and Romeo[26]	32	26	0	3 to 4 anchors per shoulder
Kim et al.[69]	167	44	4	
Kim et al.[68]	58	39	10	Comparative series
Abrams et al.[1]	61	35	6.6	
Ide et al.[59]	55	42	7	Athletes
Mazzocca et al.[89]	18	37	11	Contact and collision athletes
Fabbriciani et al.[35]	60	24	0	Comparative series

Modified from Cole BJ, Millett PJ, Romeo AA, et al. Arthroscopic treatment of anterior glenohumeral instability: indications and techniques. *Instr Course Lect* 2004;53:545–558.

Thermal Capsulorrhaphy

Thermal capsulorrhaphy is based on the response of the collagen molecule to thermal energy applied via a laser or radiofrequency probe (monopolar or bipolar). The thermal energy interrupts the collagen triple helix, thus shortening the molecule. The collagen fibers contract up to 60% of their length.[46] Cadaveric studies also showed decreased translation of the humeral head following treatment of the capsule with thermal energy[132] as well as reduction of capsular volume of up to 33%.[66]

Clinical studies followed and paralleled the information about the basic science rationale. These studies investigated thermal capsulorrhaphy for the treatment of many diagnoses including multidirectional instability (MDI), unidirectional instability, and rotator interval insufficiency. Miniaci and McBirnie[96] reported on 19 patients with MDI and showed nine recurrences at 9-month follow-up and significant complications of stiffness and neurologic symptoms. Others have also shown unacceptable failure rates in patients treated with thermal capsulorrhaphy only for MDI.[103]

In the setting of unidirectional instability with a Bankart lesion, several studies have investigated thermal capsulorrhaphy as an adjunct to capsulolabral repair. Gartsman et al.[37] followed 53 patients for an average of 33 months after arthroscopic treatment of anterior–inferior instability. The Bankart lesion was repaired with suture anchors and the capsule was treated with laser thermal capsulorrhaphy in 48 of 53 patients. Rotator interval repair was performed in 14 of 53 patients. According to Rowe score, 92% had good to excellent results. They found only four failures and concluded that the procedure provided results similar to open repair. Another similar study using suture anchors to repair a Bankart lesion and supplementing the repair with thermal capsulorrhaphy and rotator interval closure when indicated showed 11% recurrence in collision and contact athletes.[89] Thus, evidence exists that thermal capsulorrhaphy can be used as an adjunct to traumatic anterior and anterior–inferior instability and achieve low recurrence rates.

Controversy exists over the use of thermal energy in overhand athletes. Decreasing anterior capsular laxity and thus excessive external rotation with thermal capsulorrhaphy has been investigated. Evaluation of baseball players with internal impingement treated arthroscopically with and without adjunct thermal capsulorrhaphy showed that 97% of those treated with thermal energy returned to play. Significantly less, only 80%, of those treated without thermal energy returned to play.[80] It is important to note that the thermal treatment of the capsule was an adjunct to the treatment of other existing pathology and was not used in isolation. Enad et al.[34] reported on 19 overhand athletes treated with isolated thermal capsulorrhaphy with an average follow-up of 23 months. Six athletes were unable to return to play and the authors concluded that as an isolated treatment, thermal capsulorrhaphy was inadequate treatment.

In summary, thermal capsulorrhaphy may be effective as an adjunct treatment for anterior or anterior–inferior glenohumeral instability. Controversy remains over its role in the overhead athlete, but it may have a role as an adjunct in this patient population. Further studies are needed to define the clinical effectiveness and determine if any benefit outweighs the potential complications of nerve injury, stiffness, and attenuation of capsular tissue.

Capsule-Ligament Suture Plication

The open capsular shift has been described for the stabilization of instability without a labral lesion and for a patulous capsule after a labral lesion has been repaired. Few articles have documented the effects of arthroscopic suture plication of the capsule. It has been shown in a cadaveric model that a reduction of capsular volume of 19% can be achieved.[66]

Clinical studies have documented capsular suture plication. Caspari and Savoie[23] described an arthroscopic modification of the open anterior–inferior capsular shift. When a Bankart lesion is present, the capsulolabral complex is shifted superiorly on the glenoid and repaired to bone. In the absence of a Bankart lesion, the capsule is removed from the anterior inferior neck of the glenoid and shifted superiorly and secured to bone in the shifted position. Duncan and Savoie[32] showed satisfactory results in a small group of 10 patients with MDI. Others have also described capsular plication in patients with MDI.[91,141] Tauro[129] has described a modification of the capsular shift that involves a radial split at the 6 o'clock position. This procedure was used in combination with Bankart repair. A recurrence rate of 6.9% was demonstrated in a group of 34 patients with 2- to 5-year follow-up. Twenty-nine patients were treated with suture anchors and five patients were treated with transglenoid sutures. Two recurrences occurred in the suture anchor group and two in the transglenoid suture group. Given the recent studies showing high recurrence rates and significant potential complications associated with thermal capsulorrhaphy, capsular-ligament suture plication is the preferred arthroscopic treatment for capsular laxity.

Open Surgical Treatment

Indications for an open surgical procedure often depend on the experience of the surgeon. Advanced arthroscopic reconstruction requires technical expertise and special equipment. If appropriate technical experience or equipment is lacking, then an open procedure should be performed. Regardless of experience, relative indications for open surgical treatment exist. These indications include humeral avulsions of the glenohumeral ligaments, capsular rupture, previous failed open or arthroscopic repair,

prior failed thermal capsulorrhaphy, significant glenoid or humeral bone loss, and irreparable or chronic rotator cuff deficiencies, especially of the subscapularis.[95]

Although arthroscopic repair of anterior instability has seen good results in recent reports, open procedures are still the gold standard. A decision to perform surgery and the choice of procedure performed should be individualized based on the pathologic process creating the instability and on the training and experience of the surgeon. Patients with voluntary anterior instability are often poor surgical candidates; they require psychologic testing and may benefit from a rehabilitation program.[115] A trial of prolonged rehabilitation should also be used for patients with generalized ligamentous laxity or those with multidirectional instability. In addition, the presence of a Hill-Sachs lesion or glenoid deficiency may change the surgical approach.

Many open surgical procedures have been described as successful treatments for anterior glenohumeral instability. The major procedures can be divided into three different groups: capsular, subscapularis, and bony. Capsular reattachment procedures, such as the Bankart and Matsen repairs, are based on repairing the "essential lesion," as described by Perthes[108] and Bankart.[11] Subscapularis tightening procedures, such as the Magnuson-Stack and Putti-Platt procedures, are designed to realign and tighten the subscapularis and to limit external rotation. Bony procedures have been developed to augment the deficient anterior glenoid. The Bristow and Latarjet procedures provide a bony block and a musculotendinous sling across the anteroinferior glenohumeral joint by transferring the coracoid to the glenoid neck. Other conditions, such as a large Hill-Sachs lesion, may require an osteotomy or an infraspinatus transfer. In Sweden, osteotomies of the proximal humerus have been performed to realign the version of the glenohumeral joint.[138]

Glenoid deficiency may present a unique management problem. An acute glenoid fracture involving greater than one-fourth of the glenoid fossa and associated with shoulder instability is an indication for open reduction of the fragment with screw fixation.[16] A CT scan may be useful in determining fragment size and displacement.

Capsulolabral Repair

Bankart[11] and Perthes[108] described capsular repair by reattaching the detached anterior capsule to the anterior glenoid. Bankart[11] described the repair of the capsule to the bone of the anterior glenoid through the use of drill holes and sutures. The subscapularis muscle, which is carefully divided to expose the capsule, is reapproximated without any overlap or shortening. He reported no evidence of recurrence in 27 consecutive patients.

In describing long-term results with the Bankart procedure, Rowe et al.[114] developed a rating system for repair based on stability, motion, and function (Table 12-3). Over a 30-year period, they noted a 3.5% recurrence rate in 162 operatively managed shoulders, indicating that the results were excellent in 74% and good in 23%; 69% of the patients had full range of motion. They also noted that a fracture of the rim of the glenoid did not increase the risk of recurrence, but that a moderate to severe Hill-Sachs lesion increased the risk slightly. They performed primary repairs on patients with up to one-fourth of the glenoid fossa avulsed, achieving good to excellent results in all but 1 of 15 patients. They reported that 33% of the patients in throwing sports were able to return to their sports with the same level of proficiency, and that the other 67% could throw a baseball hard, but not with the same velocity as before surgery. There was no evidence of osteoarthritic changes in the 124 patients seen in follow-up.

Rosenberg et al.[112] reported that of 31 patients managed with the Bankart repair, only 13 were normal at follow-up; 14 had minimal degenerative changes, three had moderate changes, and one had severe degenerative changes (average follow-up, 15 years). Rowe et al.[117] also reported on recurrent anterior dislocation of the shoulder after surgical repair: Of 11 patients with lesions previously repaired by the Bankart procedure, three had intact repair at the time of reoperation. The underlying cause of the postoperative instability appeared to be severe laxity of the capsule. The other eight patients had disruption of the Bankart repair as well as capsular laxity.

Currently, suture anchors (which can simplify the procedure) are commonly used in open Bankart reconstructions. In a follow-up report by Levine et al.,[79] 32 patients underwent this procedure, with no complications from the suture anchor technique. However, there were two failures (recurrent anterior dislocations). Hovelius et al.[56] found a 2% redislocation rate after Bankart procedure compared with a 19% redislocation rate with the Putti-Platt procedure. More than one-third of the patients younger than age 25 were dissatisfied with the Putti-Platt procedure. The Bankart repair is frequently modified to adjust for capsular laxity, which makes this the procedure of choice for many orthopedic surgeons.

In 1989, Thomas and Matsen[131] described a procedure for repairing the anterior glenoid labrum without separating the subscapularis from the capsule (Fig. 12-9). They recommended detaching the capsule with the subscapularis tendon from the lesser tuberosity and reflecting it medially, exposing the anterior glenoid. A direct repair of the capsule can then be performed by placing the suture through the anterior glenoid. The subscapularis can then be reattached without advancement. For patients with capsular laxity, they recommended advancing the subscapularis. At 5.5 years average follow-up, good to excellent results were reported in 97% of patients. Range of motion was maintained with an average of 171 degrees of forward flexion and 84 degrees of external rotation in abduction.

TABLE 12-3
RATING SYSTEM FOR BANKART REPAIR

Scoring System	Units	Excellent (100–90)	Good (89–75)	Fair (74–51)	Poor (50 or less)
Stability					
No recurrence, subluxation, or apprehension	50	No recurrences	No recurrences	No recurrences	Recurrence of dislocation
Apprehension when placing arm in certain positions	30	No apprehension when placing arm in complete elevation and external rotation	Mild apprehension when placing arm in elevation and external rotation	Moderate apprehension during elevation and external rotation	Marked apprehension during elevation or extension
Subluxation (not requiring reduction)	10	No subluxations	No subluxations	No subluxations	
Recurrent dislocation	0				
Motion					
100% of normal external rotation, internal rotation, and elevation	20	100% of normal external rotation; complete elevation and internal rotation	75% of normal external rotation; complete elevation and internal rotation	50% of normal external rotation; 75% of elevation and internal rotation	No external rotation; 50% of elevation (can get hand only to face) and 50% of internal rotation
75% of normal external rotation, and normal elevation and internal rotation	15				
50% of normal external rotation and 75% of normal elevation and internal rotation	5				
50% of normal elevation and internal rotation; no external rotation	0				
Function					
No limitation in work or sports; little or no discomfort	30	Performs all work and sports; no limitation in overhead activities; shoulder strong in lifting, swimming, tennis, throwing; no discomfort	Mild limitation in work and sports; shoulder strong; minimum discomfort	Moderate limitation doing overhead work and heavy lifting; unable to throw, serve hard in tennis, or swim; moderate disabling pain	Marked limitation; unable to perform overhead work and lifting; cannot throw, play tennis, or swim; chronic discomfort
Mild limitation and minimum discomfort	25				
Moderate limitation and discomfort	10				
Marked limitation and pain	0				
Total units possible	100				

From Kim SH, Ha KI, Cho YB, Ryu BD, Oh I. Arthroscopic anterior stabilization of the shoulder: two to six-year follow-up. *J Bone Joint Surg Am* 2003;85-A(8):1511–1518, with permission.

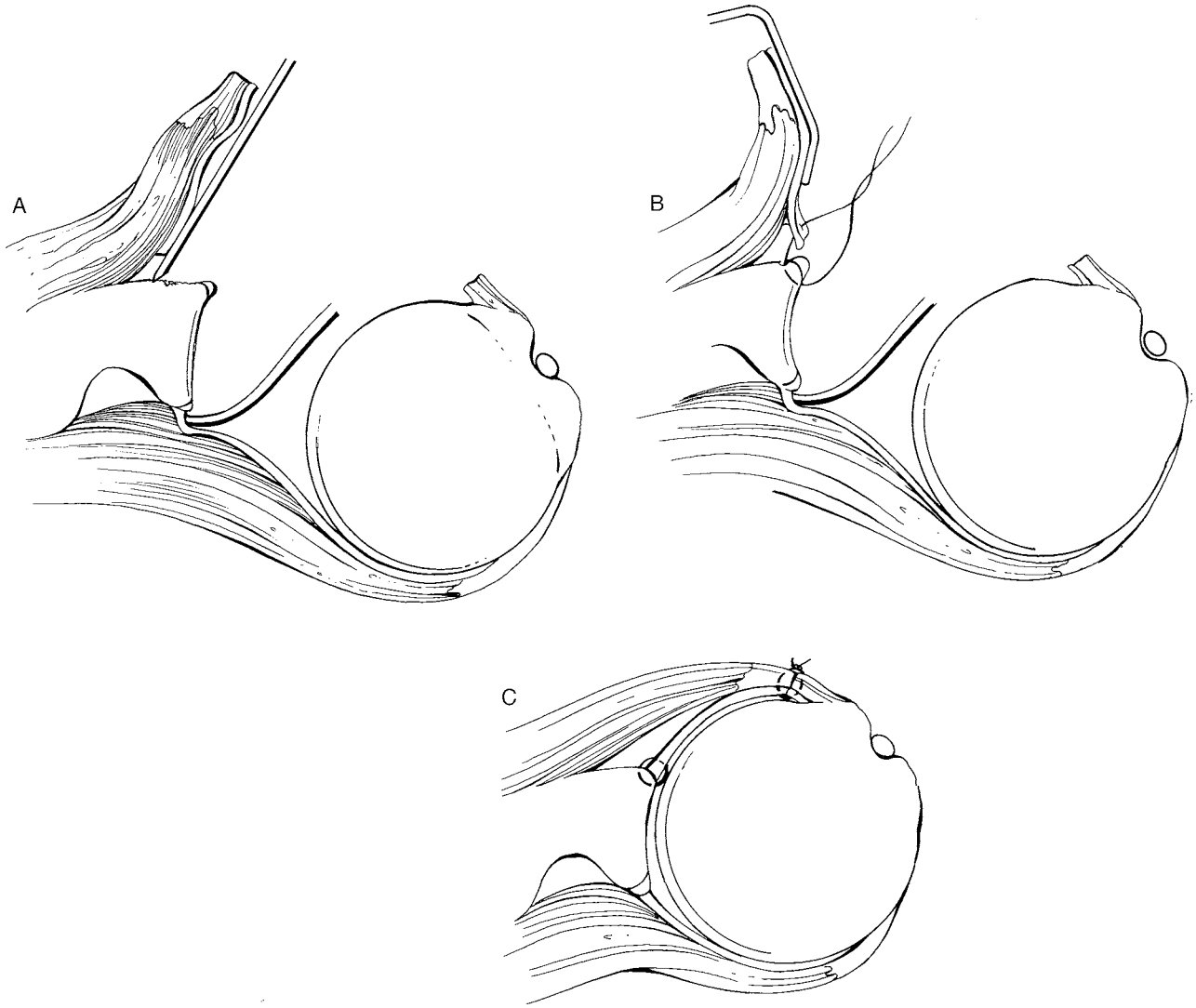

Figure 12-9 Matsen procedure. **(A)** The subscapularis is reflected with the capsule, exposing the Bankart lesion. **(B)** After the anterior glenoid neck is roughened with a curette, the Bankart lesion may be repaired. **(C)** The subscapularis is then reattached in its anatomic position to limit loss of external rotation. (From Thomas SC, Matsen FA 3rd. An approach to the repair of avulsion of the glenohumeral ligaments in the management of traumatic anterior glenohumeral instability. *J Bone Joint Surg Am* 1989;71(4):506–513, with permission.)

Capsular Shift Procedures

The effect of selective capsulorrhaphy has been documented. Gerber et al.[39] showed unique restrictions of passive motion with capsulorrhaphy of different areas of the capsule. Anterior plications restrict external rotation and posterior plications restrict internal rotation. Superior capsular plications restrict motion when the shoulder is in an adducted position and inferior plications restrict motion in abduction. A notable specific finding was that closure of the rotator interval decreased external rotation of the adducted arm 30.1 degrees, but caused no significant limitation of external rotation in abduction.

The Rockwood method of capsular imbrication (Fig. 12-10), as described by Wirth et al.[143] and Lusardi et al.,[84] addresses not only the repair of the capsule to the anterior labrum, but also the issue of capsular laxity. The subscapularis tendon is divided and reflected off the capsule. The lateral stump of the subscapularis tendon is also reflected from the capsule, exposing the entire capsule. The capsule is then divided vertically midway between its attachment on the glenoid rim and the humeral head. The joint is inspected for labral tear and stripping of the labrum, capsule, and periosteum off their normal attachments on the glenoid rim and the neck of the scapula. If stripping is noted, the capsule is reattached using sutures, followed by imbrication of the anterior capsule. The extent of the anteroinferior capsular laxity determines the degree of imbrication necessary to reduce the glenohumeral joint. In

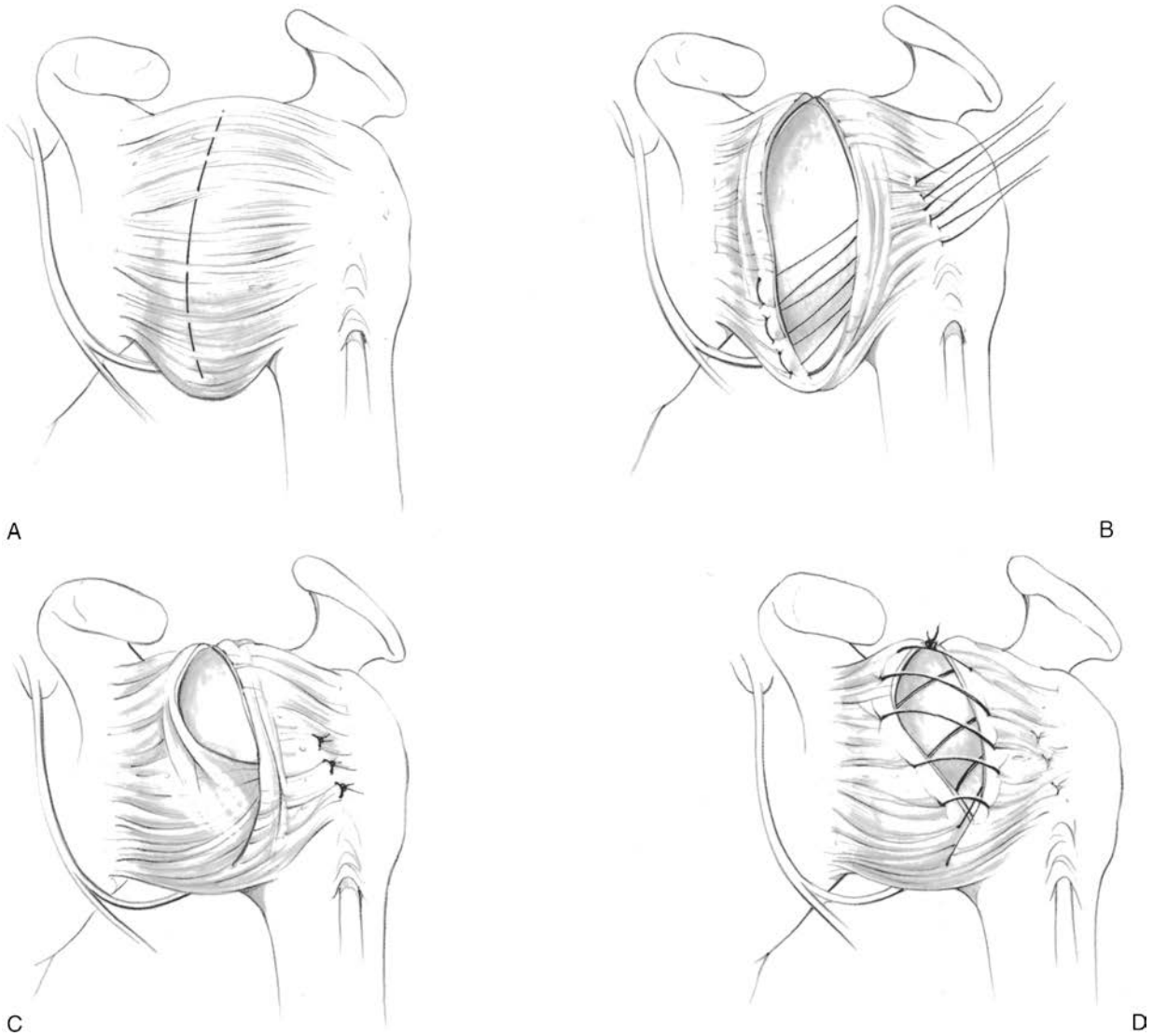

Figure 12-10 Rockwood method. **(A)** Division of the capsule vertically midway between the glenoid and humeral attachment. **(B)** The medial aspect of the capsule is overlapped laterally and superiorly under the lateral aspect of the capsule. **(C)** The medial aspect of the capsule is secured under the lateral aspect of the capsule with the arm held in 30 degrees of external rotation, and the lateral aspect of the capsule is advanced superiorly and medially over the medial aspect of the capsule, creating a double thickness of the weakened capsule. **(D)** If a capsular gap occurs, the defect may be repaired by lacing 1-mm–wide Dacron tape across the gap. (From Lusardi DA, Wirth MA, Wurtz D, Rockwood CA Jr. Loss of external rotation following anterior capsulorrhaphy of the shoulder. *J Bone Joint Surg Am* 1993;75(8):1185–1192, with permission.)

shoulders with a large degree of capsular laxity, the medial aspect of the capsule is often shifted 10 to 15 mm medially and superiorly and is imbricated for 15 to 20 mm by the lateral aspect of the capsule. After this procedure, the lateral aspect of the capsule is "double-breasted" by shifting the lax lateral capsule medially and superiorly and suturing it down to the anterior surface of the medial aspect of the capsule. Lusardi et al.[84] believed that this procedure eliminated laxity and provided added strength to the anterior capsule. Subsequently, the subscapularis tendon is anatomically repaired. In a study of 132 patients,[143] 93% had good or excellent results from this procedure at a minimum follow-up of 2 years. Patients sustained a loss of external rotation of 7 degrees compared with the contralateral side, and there was a 96% success rate on apprehension testing.

Neer and Foster[100] described a capsular shift procedure to address involuntary inferior and multidirectional instability related to capsular laxity (Fig. 12-11). The goal of this operation is to reduce joint volume. After thorough psychiatric evaluation, followed by initial rehabilitation, a capsular shift is performed, reducing the volume of the inferior portion of the capsule by detaching the capsule from the

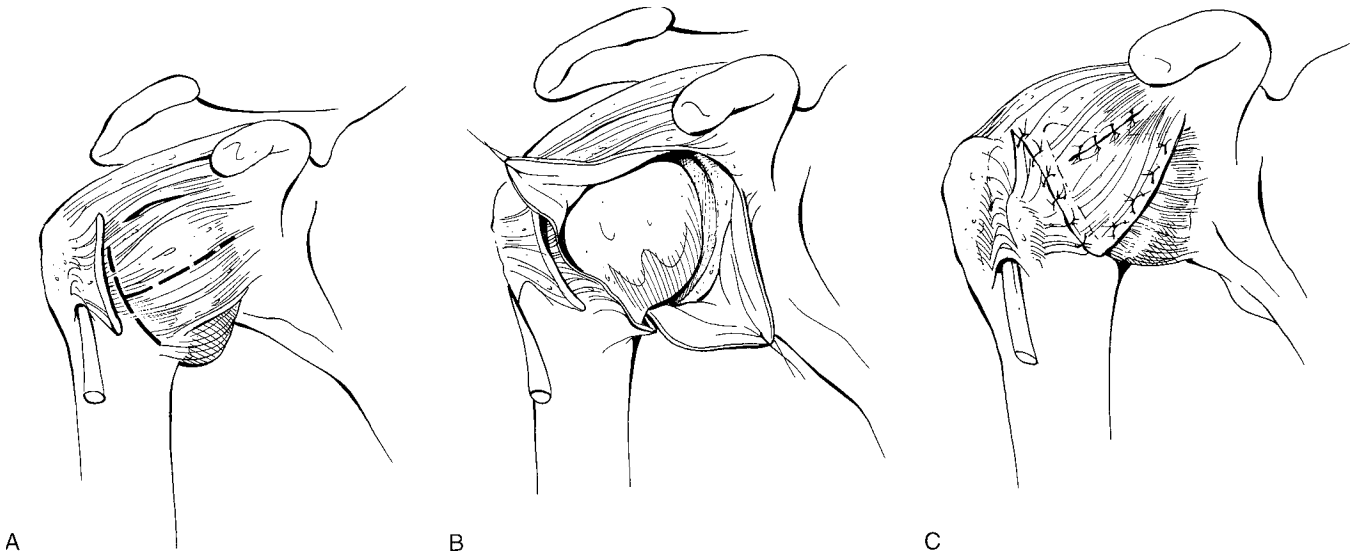

Figure 12-11 Neer inferior capsular shift. **(A)** Capsular incisions. The opening between the superior glenohumeral ligament and the middle glenohumeral ligament (which is almost constant) is closed with nonabsorbable sutures. A T-shaped opening is made by incising longitudinally between the middle glenohumeral ligament (MGHL) and the inferior glenohumeral ligament (IGHL) and detaching the capsule and ligaments from the neck of the humerus anteriorly, inferiorly, and to the posterior part of the neck. **(B)** Preparation of flaps and slot. The arm is externally rotated as the inferior flap is detached from the inferior part of the neck of the humerus, all the way back to the posterior aspect of the neck. During this step, a flat elevator is used to protect the axillary nerve. A shallow slot is made in the neck of the humerus anteriorly and inferiorly. **(C)** Relocating the flaps. The arm is held in slight flexion and 10 degrees of external rotation on the arm board. The inferior flap is relocated first. It is pulled forward to tighten the posterior part of the capsule until posterior subluxation no longer occurs, and it is pulled upward until the inferior capsular pouch is eliminated. The flap is then sutured to the stump of the subscapularis tendon and to the part of the capsule remaining on the humerus to hold it against the slot. The surplus portion of the flap is usually folded over to further reinforce the capsule. The superior flap, which contains the MGHL, is then brought down over the inferior flap so that it acts to suspend the humerus and also reinforces anteriorly. Note the sutures used to close the cleft between the MGHL and the IGHL before making the capsular incision. Finally, the subscapularis tendon is brought to its normal position and secured with nonabsorbable sutures so that it will remain a strong internal rotator. (Modified from Neer CS 2nd, Foster CR. Inferior capsular shift for involuntary inferior and multidirectional instability of the shoulder. A preliminary report. *J Bone Joint Surg Am* 1980;62(6):897–908.)

neck of the humerus and shifting it to the opposite side of the calcar. This maneuver not only obliterates the inferior pouch and capsular laxity on the side of the approach, but also reduces laxity on the opposite side. In a cadaveric study, Lubowitz et al.[83] showed that the inferior capsular shift reduced shoulder volume by 57%. Bigliani et al.[14] performed the Neer capsular shift procedure on 68 shoulders in 63 athletes. There was a 7-degree average loss of external rotation and a 2.9% incidence of recurrent postoperative dislocation. Of the 63 athletes, 92% returned to their sports and 75% of those returned to the same level of competitiveness. All 31 athletes involved in overhead sports returned to their sports (71% at the same level). Of the 10 overhead athletes who were involved primarily in throwing, five returned to the same level and five returned to throwing, but at a lower effectiveness rate than before surgery. Of the six professional or varsity athletes, only two returned to the same level of sports. In this series, 31% of the patients had a history of dislocation and had a Bankart lesion. The Bankart lesion is repaired at the time of the capsular shift. The series showed a 96% rate of good or excellent results and a redislocation rate of only 1.5%. No patient had recurrent subluxation.

The Jobe reconstruction[65,74,75] is a modified anterior capsulolabral shift, in which the subscapularis tendon is split transversely in line with its fibers at the junction of the upper two-thirds with the lower third (Fig. 12-12). A horizontal anterior capsulotomy is then made in line with the split of the subscapularis tendon. The labrum is left intact if still attached. If it is not, the anterior neck is decorticated and a superior flap is shifted inferiorly, overlapping and reinforcing the inferior flap that was previously shifted superiorly but not medially. Kvitne et al.[75] obtained 97% good or excellent results, with 81% of the patients returning to the same level of competition; 100% of the patients were subjectively satisfied with the operation. At the 2-year follow-up, no complications related to the use of fixation devices had occurred.

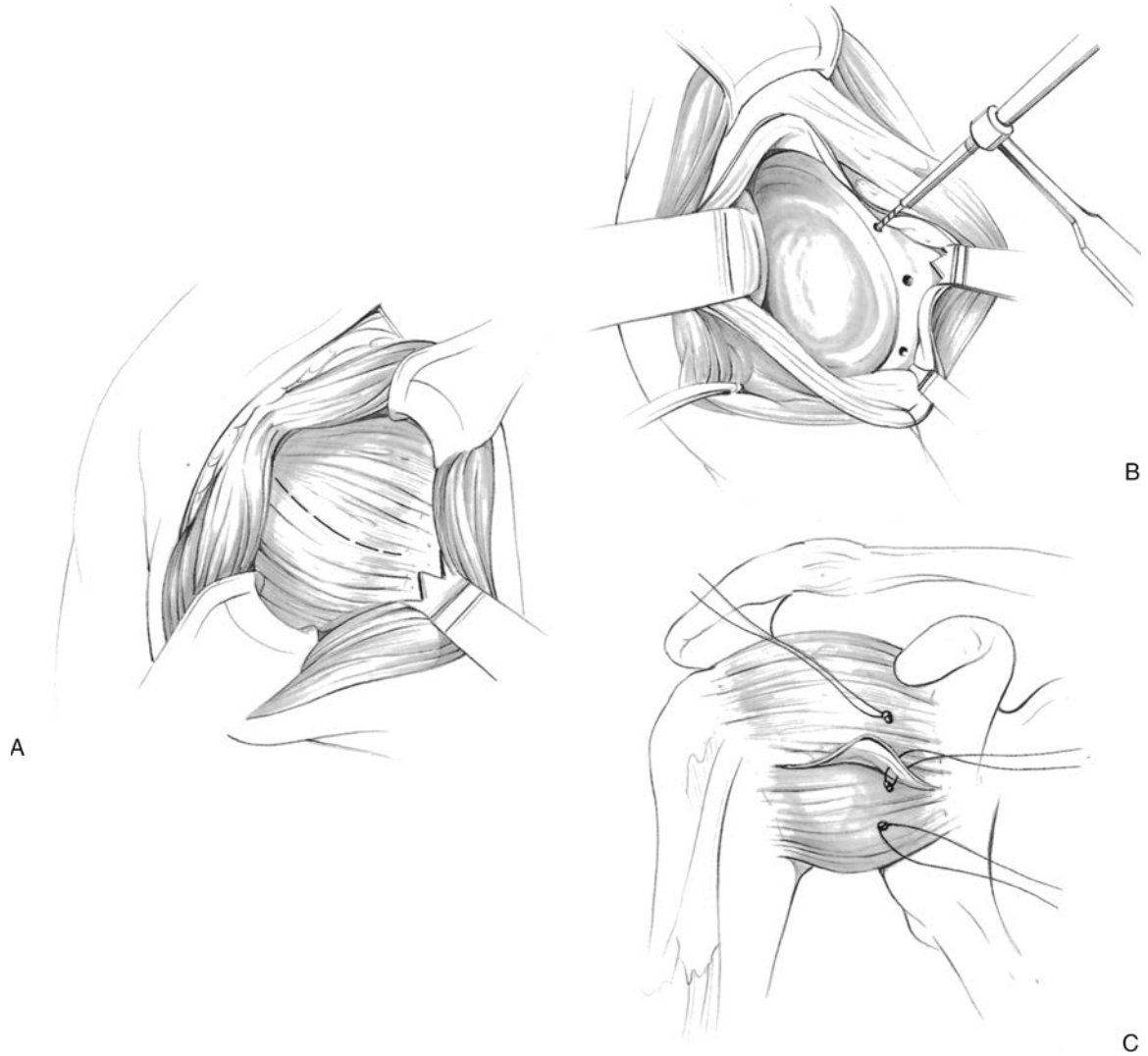

Figure 12-12 **(A)** Glenohumeral joint capsule exposure: *Dotted line* indicates site of capsulotomy. **(B)** Suture anchors are placed. **(C)** Superior capsular flap is shifted anteriorly, overlying the inferior flap. (From Kvitne RS, Jobe FW. The diagnosis and treatment of anterior instability in the throwing athlete. *Clin Orthop* 1993;291:107–123, with permission.)

Subscapularis Procedures

The current trend in orthopedics is away from subscapularis-based procedures. These procedures have a tendency to decrease external rotation and, although results are excellent in terms of redislocation rates, the patients have higher rates of dissatisfaction and osteoarthritis.

The Putti-Platt operation was described by Osmond-Clark in 1948.[105] This procedure shortens the subscapularis muscle unit by doubling it in a vest-over-pants fashion over the anterior glenohumeral joint. This limits external rotation and causes scarring in the anterior portion of the shoulder. Magnuson and Stack[85] described a procedure that involves transfer of the subscapularis tendon from the lesser to the greater tuberosity across the bicipital groove and 1 cm distally, which is believed to strengthen the anterior muscle barrier. As described by Magnuson and Stack, the operation results in 25% to 50% limitation of external rotation. Results of the Putti-Platt procedure show a long-term success rate of approximately 95%,[77,110] but it limits external rotation by 12 to 28.8 degrees. Regan et al.[110] reported that 22% of patients who had a Putti-Platt procedure had to modify throwing (compared with 38% in the Magnuson and Stack study[85]) and that 52% of patients with Magnuson-Stack procedures returned to their preinjury levels of competition.

Glenoid Bone Procedures

Significant glenoid bone loss, as described earlier in this chapter, is documented as approximately 25% of the anterior–posterior dimension.[15,21,22] Increased rates of recurrent instability with significant bone loss have revealed the need to address bony deficiencies.[21,22,38]

Glenoid Augmentation with Iliac Crest Bone Graft

Multiple authors have reported on reconstruction of the anterior glenoid with a free bone graft.[17,45,58,102] The goal of this technique is to restore the arc of the glenoid.[95] Techniques vary among different studies, but the most recent results have been good. Haaker et al.[45] and Hutchinson et al.[58] reported no recurrences in their series of 24 and 14 patients, respectively. In these two studies, patients were satisfied and had minimal loss of motion. Niskanen et al.[102] reported on a bone grafting procedure of the anterior glenoid that involved an iliac crest graft being press-fit into a trough created in the anterior glenoid. No other fixation was used. Fifty-two shoulders were included with a mean follow-up of 6 years. The recurrence rate was 21% and significant degenerative changes were present in 27 shoulders.

Coracoid Process Transfers

The Bristow procedure, originally described by Helfet[47] and later modified several times,[88] provides a bony block to dislocation by transferring the coracoid with the attached conjoined tendon to the neck of the scapula through an opening in the subscapularis tendon. This also reinforces the capsule with the conjoined tendon. Prevention of dislocation with this procedure has been very good (0% to 6%).[9,54,82,88,122] Nevertheless, Young and Rockwood[146] reported an overall good to excellent outcome in only 50% of 34 patients treated. A high rate of subluxation and hardware complications have been reported with the modified Bristow procedure; in addition, external rotation is frequently limited and overhead athletes usually cannot return to a highly competitive level.[9,82] Average loss of external rotation has been reported as high as 23 degrees.[134]

Burkhart and Debeer[21] contend that the Laterjet procedure with its larger coracoid bone block and soft tissue reinforcement with the attached conjoined tendon is more effective than the Bristow procedure, which has a smaller bone block. The Bristow procedure, they argue, relies more on the soft tissue restriction provided by the attached conjoined tendon when the shoulder is brought into abduction and external rotation. Allain et al.[2] demonstrated the Laterjet to be a stable reconstruction for recurrent instability. Of the 52 shoulders included in the study, none had redislocated at an average follow-up of 14.3 years. The same study showed osteoarthritis in 34 patients (58%) following the Laterjet procedure; however, the majority (25 patients) were only grade 1 changes. The factor most associated with osteoarthritis was placement of the coracoid too far laterally on the glenoid. Using the Rowe score as an outcome measure, 88% had a good or excellent result.

Hovelius et al.[55] recently showed results of the Laterjet procedure that are encouraging. In 118 shoulders with 15.2-year follow-up, only three experienced redislocation. Fourteen patients (12%) experienced dislocation or subluxation. Loss of external rotation was 12.4 degrees with the shoulder in abduction and 86% were able to return to their previous level of sports activity.

Humeral Bone Procedures

The interest in addressing large Hill-Sachs lesions is related to the increased recurrence rate in patients with these lesions noted by Rowe et al.[114] They noted that 100% of their patients with mild deficits had good to excellent results after Bankart repair, with no recurrences, but that those with moderate and severe defects had 5% poor results. Millett et al.[95] recommended addressing any glenoid bone deficiencies before surgical options for humeral defects are considered. These options include allograft reconstruction (Fig. 12-13) of the humeral defect (Hill-Sachs lesion) or rotational osteotomy of the proximal humerus.

Miniaci and Gish[97] recently reported on 18 patients with posterolateral defects that were greater than 25% of the humeral head. Average follow-up was 50 months. The average Constant-Murley score postoperatively was 78.5 and the Western Ontario Shoulder Instability Index showed significant improvement. There were no episodes of recurrent instability and 16 of 18 patients returned to work.

Derotation osteotomies of the humerus for instability have been described by Saha and Das.[119] They noted that the retrotorsion of the upper humerus is converted to relative antetorsion as the arm is raised overhead to 120 degrees. An osteotomy was performed that would align the articular surface of the humerus with the glenoid with the arm in 120 degrees of abduction. Although their early results showed no complications in the initial series, this technique has never become popular in the United States.

Postoperative Care

The timing of our postoperative rehabilitation protocol is described in Table 12-4. The patient returns for follow-up at 10 days, at which time the immobilizer is removed and rehabilitation is begun with pendulum exercises, isometric shoulder abduction, and internal rotation exercises. The patient should not proceed to regular activity before 3 months.

Authors' Preferred Surgical Technique: Anterior Capsulorrhaphy

Superficial Exposure

The anterior approach advocated by the authors is the same for both capsular repair and capsular plication and glenoid or humeral bone augmentation procedures. This unified anterior approach allows correction of any degree

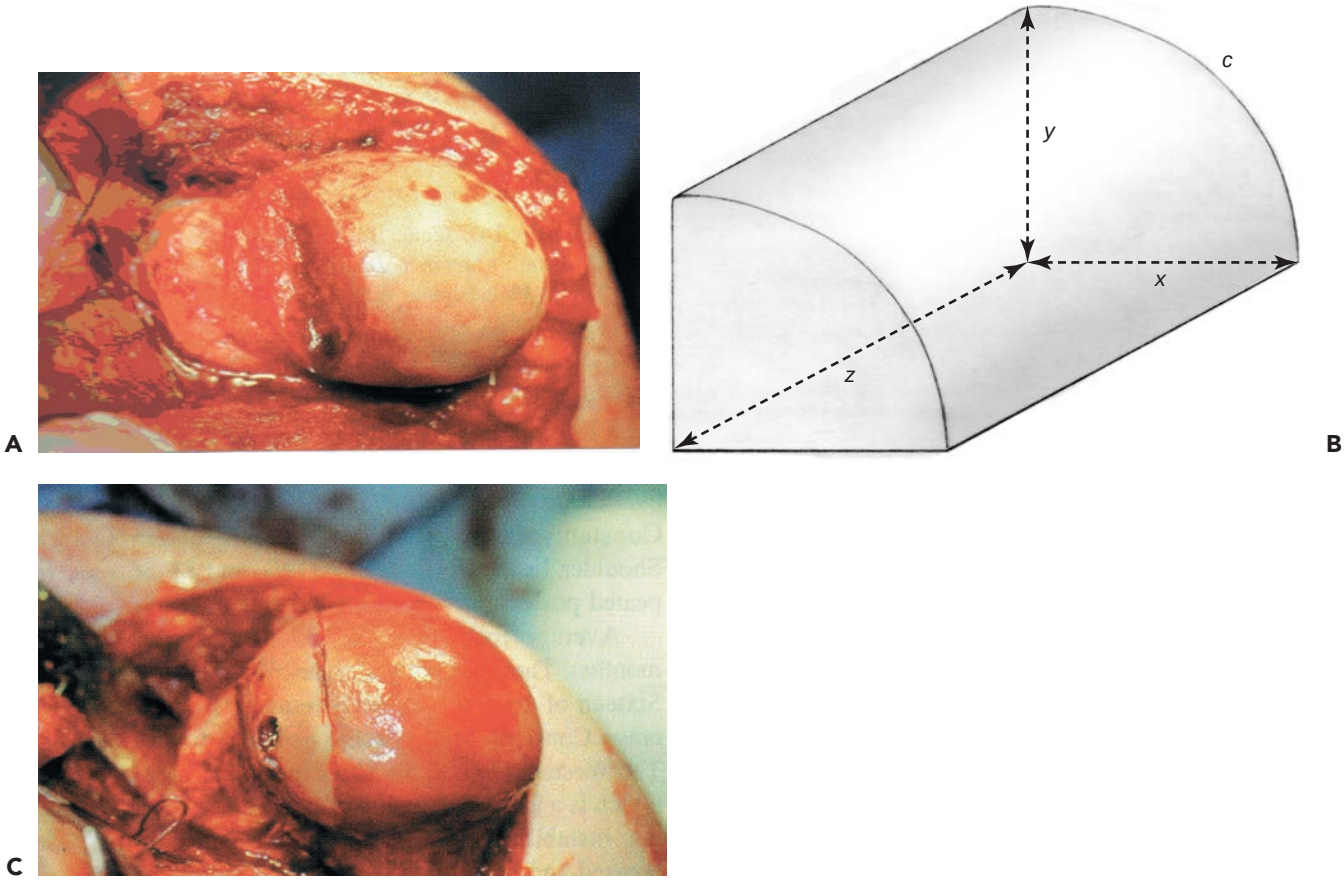

Figure 12-13 Humeral head allograft reconstruction. **(A)** Excised lesion. **(B)** Shaping the allograft. **(C)** Reconstructed humeral head.

of anterior capsular–labrum avulsion, capsular laxity, or bone loss that is defined intraoperatively.

The anterior axillary fold is identified by internal rotation of the arm. The incision begins in this anterior skin crease at the inferior border of the pectoralis major tendon and is extended superiorly approximately 5 cm. The subcutaneous plane is developed along the cephalic vein to the level of the clavicle and within 3 cm of the deltoid insertion site. The investing fascia on the medial side of the cephalic vein is incised over the extent of the subcutaneous dissection, the vein and deltoid muscle are retracted laterally, and the pectoralis major muscle is retracted medially. In heavily muscled patients, incising the upper 1 cm of pectoralis major tendon insertion can facilitate retraction. This is usually not necessary. In many patients, there is a leash of vessels superficial to the clavipectoral fascia that crosses over the deltopectoral interval at the level of the tip of the coracoid. These vessels, when necessary, can be ligated to improve exposure. The clavipectoral fascia is incised lateral to the coracobrachialis and the short head of the biceps (strap muscles), the musculocutaneous nerve is palpated on the undersurface of the muscle to define its location, and these muscles are then retracted medially.

Subscapularis Dissection

The structures to be identified are the subscapularis tendon from its bony insertion to its musculotendinous junction, the rotator interval, the long head of the biceps tendon within its groove, and the anterior humeral circumflex vessels. The axillary nerve can be palpated as it courses over the superficial inferior portion of the subscapularis muscle and enters the area under the inferior capsular pouch. The subscapularis tendon is incised with a coagulation Bovie cautery to the level of the capsule. The subscapularis incision begins 1 cm medial to the musculotendinous junction from the uppermost portion of the subscapularis, at the rotator interval, and extends inferiorly to the anterior circumflex vessels (Fig. 12-14A). These vessels are not generally ligated, but if more inferior exposure is required, then they can be. At the level of these vessels, the fibers of the subscapularis muscle are bluntly dissected toward the axillary nerve, taking care to protect the nerve from injury. Blunt dissection of the subscapularis at this level will identify the interval between the muscle and capsule. This is more easily accomplished at the inferior portion of the subscapularis than more superiorly where the tendon and capsule are more confluent.

TABLE 12-4
POSTOPERATIVE PHYSICAL THERAPY PROTOCOL

Time Frame	Procedures
0–2 days	Sling can be removed for bathing, dressing, and exercises Active exercises for elbow, wrist, and grip
7–21 days	Wean from sling Pendulum exercises Isometric shoulder abduction and internal rotation exercises
3 weeks	Wall climbing Active assisted ROM, abduction, and flexion Continue above
4 weeks	Progressive ROM Active assisted ROM, active ROM, and progressive ROM in flexion, abduction, and internal rotation Progressive resisted exercises Upper body ergometer
6 weeks	Progressive strengthening exercises Isokinetic strengthening when appropriate for patient Isolate external rotation and internal rotation
8–10 weeks	Progressive isokinetic strengthening of internal and external rotation in increasing ranges of abduction Add isotonic internal and external rotation strengthening
10–12 weeks	Plyoball
3 months	Patient returns to normal activity

ROM, range of motion.

After dissection of the interval between the muscle–capsule inferiorly, the plane of dissection is then bluntly carried superiorly with a narrow elevator. When it is ensured that the interval between the muscle and capsule is developed superiorly, sharp dissection can then be performed laterally between the tendon and capsule to meet the previously placed vertical incision in the tendon (Fig. 12-14B). The tendon is retracted medially with traction sutures and the subscapularis muscle is bluntly dissected medially from the capsule to a level 1 cm medial to the glenoid rim. The glenoid rim can then be easily palpated and the subscapularis tendon and muscle can be retracted medial to it with an anterior Bankart-type retractor or Hohmann retractor. The entire anterior capsule should now be exposed from the rotator interval to the anterior humeral circumflex vessels. The inferior capsular pouch is then exposed by blunt dissection deep to these vessels and deep to the most inferior portion of the subscapularis tendon. The axillary nerve can now be seen and palpated as it courses over the inferior capsule, and it can be retracted and protected by a blunt retractor if the surgeon prefers. We do not routinely expose it but are acutely aware of where it resides so as not to damage it. The entire anterior and inferior capsule is now exposed, and the interval between the capsule and lateral stump of the subscapularis tendon is sharply dissected a distance of 5 to 7 mm toward the lesser tuberosity (Fig. 12-14B).

Lateral Capsulotomy

The arm is placed in the plane of the scapula at 45 degrees abduction and 45 degrees of external rotation. In this position, the capsule is usually closely applied to the humeral head without any redundancy. If the capsule can be easily pulled away from the humeral head in this arm position, then there is redundancy of the anterior capsular pouch and a capsular shift will likely be necessary. In all anterior procedures, the capsule is incised 5 to 10 mm medial to its insertion on the humeral neck from the rotator interval to the anteroinferior capsular pouch (Fig. 12-14C). The humeral head is then retracted with a ring retractor and the capsule is retracted medially to inspect the glenoid rim and labrum–capsular attachments. With this exposure, the entire labrum can be examined along with the origin of the biceps tendon, the undersurface of the supraspinatus tendon, and the articular surfaces of the glenoid and humeral head. Hill-Sachs lesions can be palpated and, if large, can be seen with extension and external rotation of the arm.

Repair of the Bankart Lesion

If an avulsion of the capsule and labrum is noted (Bankart lesion), then the lesion is dissected from the glenoid rim medially to the glenoid neck, and the

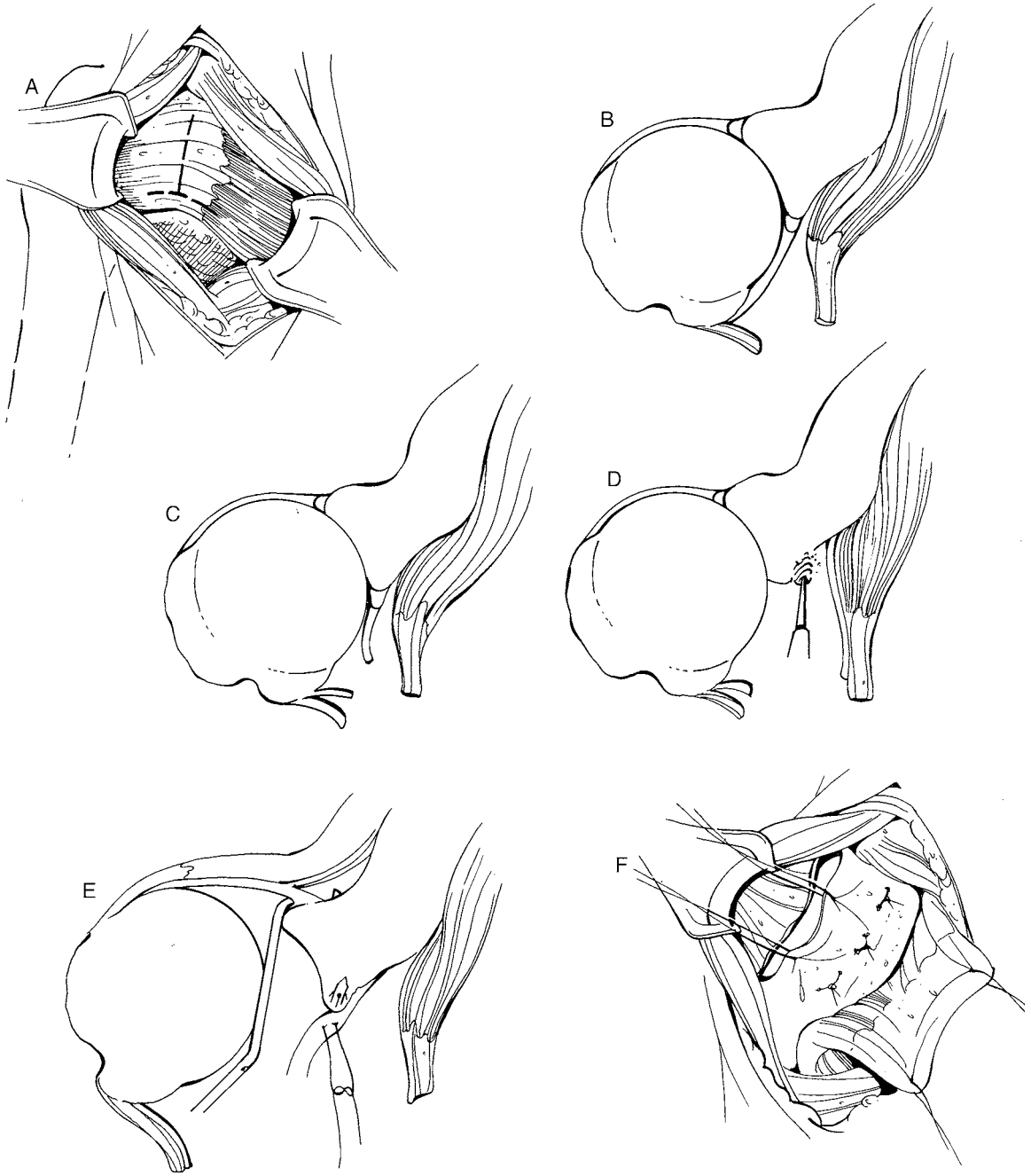

Figure 12-14 **(A)** The subscapularis incision begins 1 cm medial to the musculotendinous junction from the uppermost portion of the subscapularis at the rotator interval, and extends inferiorly to the anterior circumflex vessels. **(B)** The subscapularis is separated from the underlying capsule medially to the glenoid rim and laterally to the lesser tuberosity. The lateral stump of the subscapularis is defined to allow separated closure of the lateral capsule and subscapularis. **(C)** The capsule is then incised 7 to 10 mm from its humeral insertion site from the rotator interval to the inferior capsular pouch. **(D)** The capsule and subscapularis are reflected medially, the glenoid labrum is inspected, and, if a Bankart lesion is noted, it is reflected medially and the glenoid rim and neck are decorticated with a burr. **(E)** The suture anchors are placed exactly at the glenoid rim, and both limbs of the sutures are passed through the labrum and capsule. **(F)** The capsule is brought laterally and the limbs of the sutures are tied on the outside of the capsule.

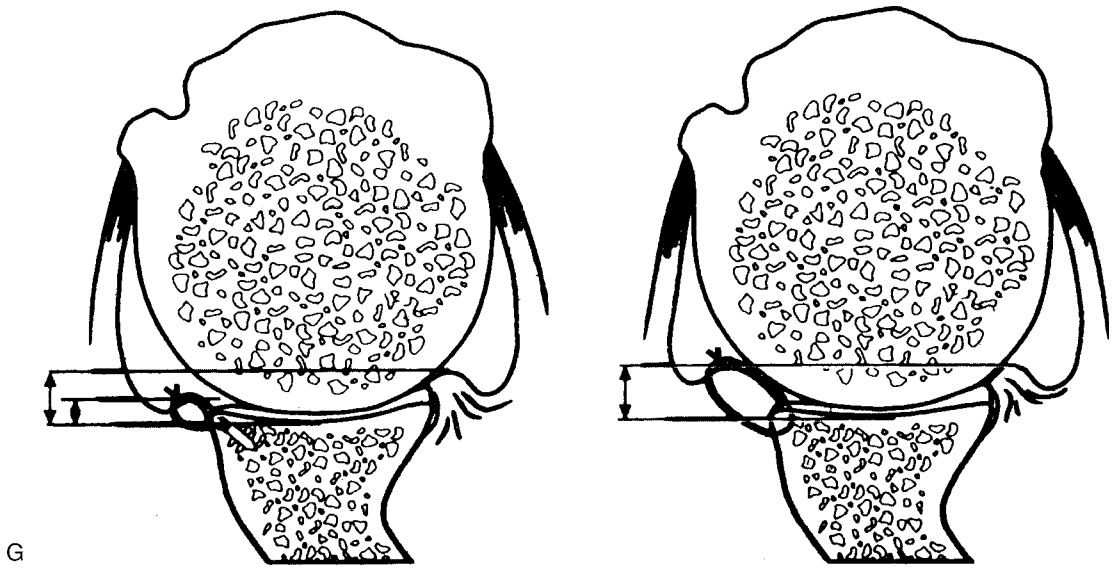

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Figure 12-14 *(continued)* **(G)** The goal of the surgery is to repair the capsule and labrum so that a "bumper" effect of the labrum is reconstructed onto the glenoid rim. It therefore is necessary to put the anchors at the rim of the glenoid, to pass the suture through the labrum, and, if the labrum is attenuated, to use a barrel stitch to gather up the capsule to form a thickened mass of tissue to reform an anterior bumper.

anterior Bankart-type retractor or Hohmann retractor is placed deep to the lesion, thereby exposing the entire anterior glenoid rim. If this exposure is limited by the superior and inferior attachments of the labrum, then additional exposure can be obtained by incision through the rotator interval capsule and labrum at the superior apex of the avulsed labrum. When this is performed, the entire anteroinferior capsule can be retracted medially and inferiorly, because it is hinged on the most inferior extent of the Bankart lesion. The anterior glenoid rim and glenoid are gently decorticated to bleeding bone (Fig. 12-14D).

Suture anchors are placed at the osteochondral rim of the glenoid. Care is taken not to place these anchors medial to this point (Fig. 12-14E). If the glenoid rim has been eroded to a surface that is rounded or flat and even with the glenoid neck, then placement of osseous tunnels is more difficult. When this occurs, but the bone loss is less than 20% of the anterior to posterior glenoid dimension, then bone augmentation is not performed. In this circumstance, the anchors are placed at the margin of the glenoid rim. Two to five suture anchors are placed, with the number of sutures dependent on the size of the lesion. The capsule is then pulled laterally, and the Bankart retractor is placed superficial to the capsule and deep to the subscapularis. The capsule–labrum junction is identified, and both limbs of each suture are passed through this tissue so that when the sutures are tied, the labrum is firmly and anatomically approximated to the glenoid rim (Fig. 12-14F). When the labrum and capsule are attenuated, the suture is passed through the labrum and capsule tissue using a "barrel stitch" (Fig. 12-14G). With the barrel stitch, the tissue is rolled up, creating a thickened tissue to act as a "new labrum" and as such provide a new "bumper" and restore the concavity of the glenoid fossa.

Lateral Capsular Closure

After completion of the repair of the Bankart lesion, the lateral capsulotomy is closed. The arm is placed in the plane of the scapula at 45 to 60 degrees of abduction and 45 to 60 degrees of external rotation. The arm is held with a sterile intraoperative arm positioner (McConnell, Ft. Worth, Texas). The capsule is then pulled, with a forceps, laterally and superiorly to simulate closure of the capsule to the capsule on the humeral neck. If there is no capsular redundancy, then the rotator interval and lateral capsule are closed anatomically with nonabsorbable sutures, without any intended shortening of the tissue. The position of the arm is determined by the patient's degree of external rotation in this arm position under anesthesia before making the skin incision, by the degree of humeral head anterior translation measured under anesthesia and compared with the normal shoulder, and by the degree of generalized ligamentous laxity. In the overhead-throwing athlete, with a Bankart lesion but without generalized ligamentous laxity, the arm is externally rotated to equal the opposite shoulder, and the lateral capsule is closed. When performed in this manner, intraoperative loss of external rotation is approximately

10 to 20 degrees. This intraoperative loss of motion is regained in the postoperative rehabilitation.

If there is mild capsular redundancy noted in the foregoing arm position, then the entire anterior–inferior capsule can be shifted superiorly at the rotator interval and sutured. Under the less common circumstance of excessive capsular redundancy and a Bankart lesion, the rotator interval is closed and the capsule is incised between the middle glenohumeral ligament (MGHL) and the anterior–superior band of the IGHL, taking care not to incise the glenoid labrum. This capsular incision now creates a superior flap (superior glenohumeral ligament and MGHL) and an inferior flap (IGHL and inferior pouch). The arm is positioned in the plane of the scapula at 45 to 60 degrees of abduction and 45 degrees of external rotation. In this arm position, the inferior flap is shifted superiorly, thereby obliterating the inferior capsular pouch. The arm is then placed in 20 degrees of abduction and 35 degrees of external rotation, and the superior flap is shifted inferiorly. The capsular closure is performed with nonabsorbable suture. The extent of the shift and the capsular tightening by this method is determined by the arm position, thereby ensuring that the intraoperative external rotation will be at least 30 degrees measured in neutral abduction and 70 to 90 degrees of external rotation in the abducted position. This technique will avoid overtightening the capsule and ensure reproducible results.

Capsular Shift without a Bankart Repair

The exposure, subscapularis dissection, and lateral capsulotomy for an isolated anterior–inferior capsular shift is as described previously. The degree of capsular redundancy is assessed as described, and the labrum attachment and intraarticular structures are evaluated. In most cases of atraumatic or repetitive microtraumatic anterior, anteroinferior, or multidirectional instability, there is minimal labral or articular cartilage pathology. In these patients, the extent of the inferior capsulotomy is determined by the degree of posterior humeral head translation. The greater the degree of asymmetrical posterior translation is, the greater the degree of posterior capsular tightening is necessary; therefore, the capsulotomy is carried further posterior along the humeral neck. After completion to the lateral capsulotomy, the rotator interval is closed with nonabsorbable sutures. The capsule is incised in the midaxial plane between the MGHL and the anterior–superior band of the IGHL to the labrum, thereby creating superior and inferior capsular flaps. The arm is positioned and the capsular flaps sutured as described previously.

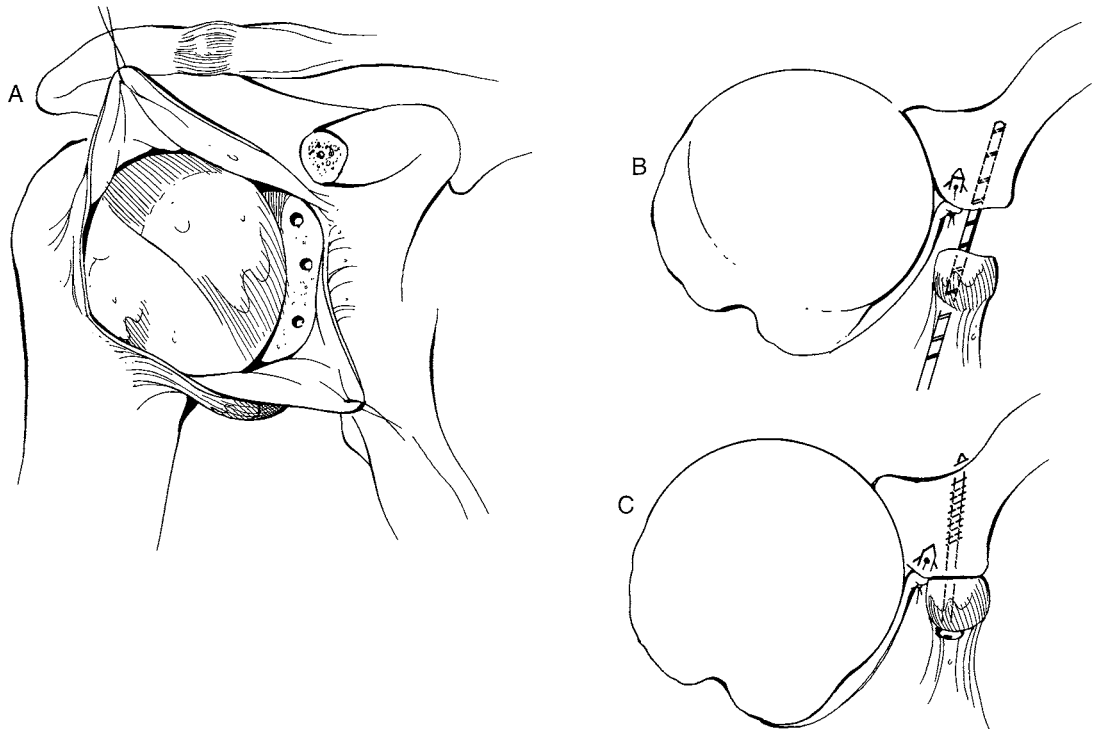

Figure 12-15 With moderate glenoid bone loss, an anterior capsulorrhaphy and coracoid transfer are performed. **(A)** The anchors are placed at the glenoid rim and the sutures are passed through the capsule and labrum, but not yet tied. **(B,C)** The glenoid defect is decorticated and the distal 1.5 cm of the coracoid are transferred to the defect and secured with a partially threaded screw.

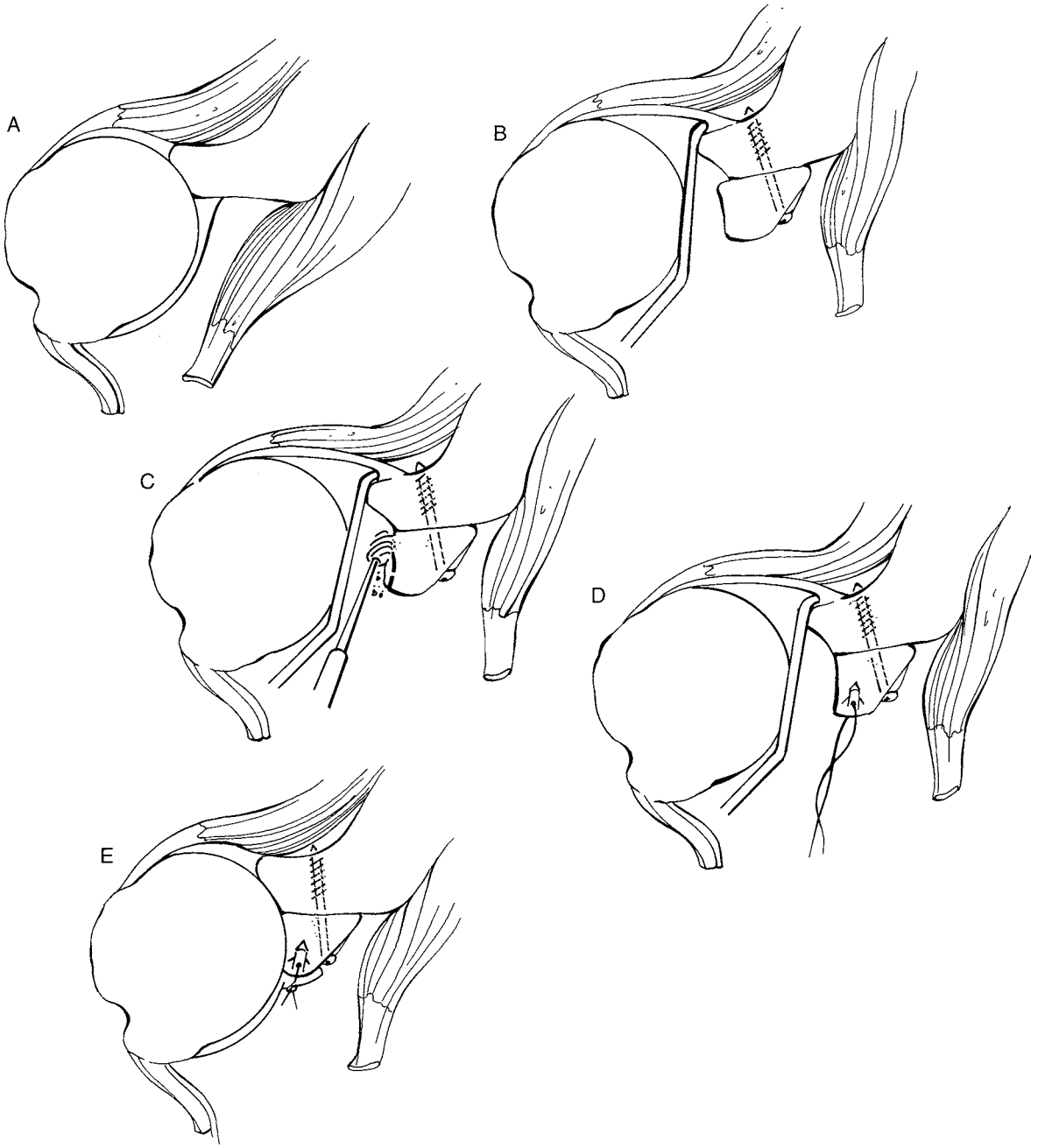

Figure 12-16 **(A)** For large anterior glenoid defects, an iliac crest graft is used to reconstruct the glenoid defect. **(B)** The defect is decorticated and made flat. A bicortical iliac crest graft is placed with its cancellous surface against the cancellous bone of the defect and secured with two partially threaded screws. **(C)** The position of the graft, which overhangs the face of the glenoid, is burred down to make a smooth contour with the native glenoid. **(D)** Suture anchors are placed into the rim of the graft. **(E)** The limbs of each suture are passed through the capsule and labrum, thereby repairing the ligaments to the new glenoid rim.

Subscapularis Closure

Closure of the subscapularis is with nonabsorbable suture without any shortening. In a similar way, the interval between the subscapularis and supraspinatus is closed. A drain is placed in the subdeltoid space. The subcutaneous tissues are closed with absorbable sutures, and the skin is closed with a nonabsorbable monofilament suture using a subcuticular technique. Steri-Strips, a sterile dressing, and a sling are applied, and the patient is reversed from the anesthetic.

Anterior Glenoid Deficiency

There is no consensus about the degree of glenoid bone loss that justifies bone reconstruction of the anterior glenoid. In our practice, we perform a bone reconstruction for cases with approximately 25% loss of the anterior-to-posterior glenoid dimension. In most people, this is approximately 6 mm of bone loss at the anterior–inferior aspect of the glenoid margin. Bone loss in the 20% to 30% range can be compensated by using a coracoid transfer; larger defects can be managed using an iliac crest graft.

The exposure, subscapularis dissection, and lateral capsulotomy for the bone augmentation procedures are as described previously, with the exception that the skin incision is extended superiorly to the tip of the coracoid. With significant glenoid bone loss, the glenoid defect is burred to a flat surface. Nonabsorbable sutures are placed with suture anchors at the osteochondral junction and passed through the labrum and capsule, but they are not tied (Fig. 12-15A). The periosteum over the osseous defect is then incised to expose the defect in an extraarticular fashion. The distal 1.5 cm of the coracoid with its attached strap muscles are osteotomized with a right-angle microsagittal saw. The tip of the coracoid is drilled with a 3.2-mm bit in a retrograde fashion and measured with a depth gauge. The drill bit is reversed, the tip of the coracoid is placed into the glenoid defect, the drill is advanced through the posterior glenoid cortex, and the depth of the glenoid hole is measured (Fig. 12-15B,C). A partially threaded 4.5-mm cancellous screw is then used to secure the coracoid tip into the glenoid defect. The previously passed capsular sutures are tied, thereby repairing the Bankart lesion to the rim; the coracoid transfer and screw remain extraarticular. The lateral capsular repair can be completed as just described; the technique is based on the degree of capsular redundancy. The subscapularis closure is as described.

For severe bone loss, an iliac crest graft is employed. The exposure, subscapularis dissection, and lateral capsulotomy are as described above. A bicortical iliac crest graft (appropriate size, 1.5 × 2.0 × 1.0 cm) is harvested. The anterior glenoid is decorticated and made flat to receive the flat cancellous surface of the iliac crest graft. The graft is placed so that the cortex of the iliac crest will constitute a new glenoid rim, and so that it slightly overhangs the articular surface of the glenoid. The graft is then secured with two 2.5-mm partially threaded cancellous screws (Fig. 12-16A,B). With a high-speed burr, the new articulating surface of the graft is contoured to match the level and radius of curvature of the remaining articular cartilage of the glenoid (Fig. 12-16C). Nonabsorbable sutures are placed at the rim of the iliac crest graft, as described for repair of a Bankart lesion. The lateral capsular and subscapularis closure is as described earlier (Fig. 12-16D,E).

Algorithm for Treatment

See Fig. 12-4.

CONCLUSION

The management of patients with anterior shoulder instability must be based on a thorough knowledge of the etiologic factors associated with the instability and the resulting pathology. The results of arthroscopic shoulder procedures continue to improve and in some cases have equaled the results of open procedures, with fewer complications. However, indications for open stabilization remain and the recurrence rates for open procedures are the gold standard.

Stability of the shoulder results from a marvelous harmony of static and dynamic factors. The challenge facing the physician treating anterior shoulder instability is to restore this often delicate balance. The choice of treatment necessary to accomplish this goal will vary according to host factors, the specific pathology, and the skill and experience of the surgeon.

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13 Posterior Instability: Open and Arthroscopic Management

Matthew L. Ramsey *Jeffrey S. Abrams*

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INTRODUCTION

Posterior instability of the shoulder is defined as the symptoms expressed as a result of excessive posterior glenohumeral translation. The symptoms associated with posterior instability may be due to *recurrent subluxation* or acute *dislocation*. Diagnosis depends on the clinical history of instability reported by the patient, reproduction of symptoms during physical examination, and the results of diagnostic evaluation.

Acute posterior dislocation is rare in comparison with its anterior counterpart. Recurrent posterior subluxation is the most common form of posterior instability and represents the principal topic of this chapter. Although posterior instability is uncommon in comparison with its anterior counterpart, it is being recognized with increased frequency.[27,32,81] Historically, the literature has been unclear on the distinction between recurrent posterior subluxation and posterior dislocation. This, combined with a limited understanding of the pathophysiology of recurrent posterior subluxation, has resulted in a lack of consensus on its diagnosis and management.

McLaughlin recognized the distinction between (locked) posterior dislocation and recurrent posterior subluxation.[49] Attempts to further classify recurrent posterior subluxation have employed the same terminology as anterior instability. This classification defines instability based on its degree (subluxation, dislocation), origin (traumatic, atraumatic), direction (anterior, posterior, inferior, multidirectional), or patient volition (voluntary, involuntary). In anterior instability, for which the origin and direction of instability often correlate with an underlying pathologic lesion and direct appropriate treatment, these terms are invaluable. The same is not true of posterior instability.

TABLE 13-1
CLASSIFICATION OF POSTERIOR INSTABILITY

Posterior dislocation
Acute posterior dislocation
Chronic (locked) posterior dislocation
Recurrent posterior subluxation
Volitional
Psychogenic
"Learned"
Dysplastic
Glenoid retroversion
Humeral head retrotorsion
Acquired
Soft tissue deficiency
Bony deficiency
Scapulothoracic dysfunction

Describing posterior instability based on a traumatic or atraumatic basis or as voluntary or involuntary does not necessarily define the underlying pathology or assist in treatment decisions. Regardless of the cause of posterior instability, the nature of the underlying pathology is not predicted based on current classification schemes. The absence of a classification system that unifies posterior instability has made it difficult to evaluate various treatments and their results.

An anatomically based classification of recurrent posterior subluxation, as opposed to the more traditional cause-based methods of classification, facilitates treatment by defining the pathologic process that produces the instability. The salient features of this anatomic-based classification system are summarized in Table 13-1.

POSTERIOR DISLOCATION

Acute Posterior Dislocation

Acute posterior dislocations are rare, accounting for approximately 5% of all dislocations. Direct trauma to the front of the shoulder, a posteriorly directed force on an adducted arm (fall on an outstretched hand), and indirect muscle forces (seizure or electrical shock) all can cause posterior dislocation.

Diagnosis

The diagnosis of posterior dislocation should be readily made through a careful history, physical examination, and supporting radiographic studies. Before the advent of radiographs, the distinctive presenting features and physical findings of posterior dislocation were described.[17] The routine use of radiography for injuries of the shoulder should increase the diagnostic accuracy. Unfortunately, nearly half of all posterior dislocations are missed.[36]

Patients with posterior dislocation typically present with the arm splinted at the side in adduction and internal rotation. The classic physical findings in posterior dislocation include the following: (a) limited external rotation of the shoulder with the arm at the side; (b) limited forward elevation of the arm; (c) a void in the anterior aspect of the shoulder; (d) prominence of the coracoid process; and (e) fullness of the posterior aspect of the shoulder. Appreciation of these features is facilitated by examining the shoulder from above. A thorough neurovascular examination before and after shoulder reduction is essential.

Radiographic evaluation for a suspected posterior dislocation includes at least an anteroposterior (AP), scapular lateral, and axillary view of the shoulder. If positioning the patient for a standard axillary radiograph is not possible because of painful abduction, a Velpeau axillary or a trauma axillary radiograph can be obtained. The views are sufficient to diagnose the position of the humeral head and any associated bony lesions (Fig. 13-1). The classic radiographic features of posterior dislocation include humeral head overlap on the glenoid rim on an AP radiograph, an empty glenoid on an axillary or a lateral radiograph, fracture of the lesser tuberosity, and a reverse Hill-Sachs lesion.

A computed tomography (CT) scan is recommended when a satisfactory trauma series is difficult to obtain or interpret. If there is any concern about an associated fracture that is not appreciated on the radiographic evaluation, a CT scan is helpful in characterizing the fracture pattern.

Treatment

The management of an acute posterior dislocation requires care to avoid further damage to the humeral head. Forceful reduction attempts in the face of a locked dislocation or a nondisplaced fracture risks displacing the humeral head. A closed reduction can be attempted if the reverse Hill-Sachs lesion involves 40% or less of the humeral head. The reduction maneuver may require the involvement of an assistant. The arm is flexed to 90 degrees and adducted to disimpact the humeral head from the glenoid rim. The arm should not be externally rotated until the humeral head has cleared the glenoid rim. Lateral traction on the arm assists in disimpacting the humeral head from the glenoid rim. Gentle pressure on the humeral head guides it into the glenoid fossa and provides tactile input on its location relative to the glenoid. Once the humeral head clears the glenoid, the arm is externally rotated and brought down to the side.

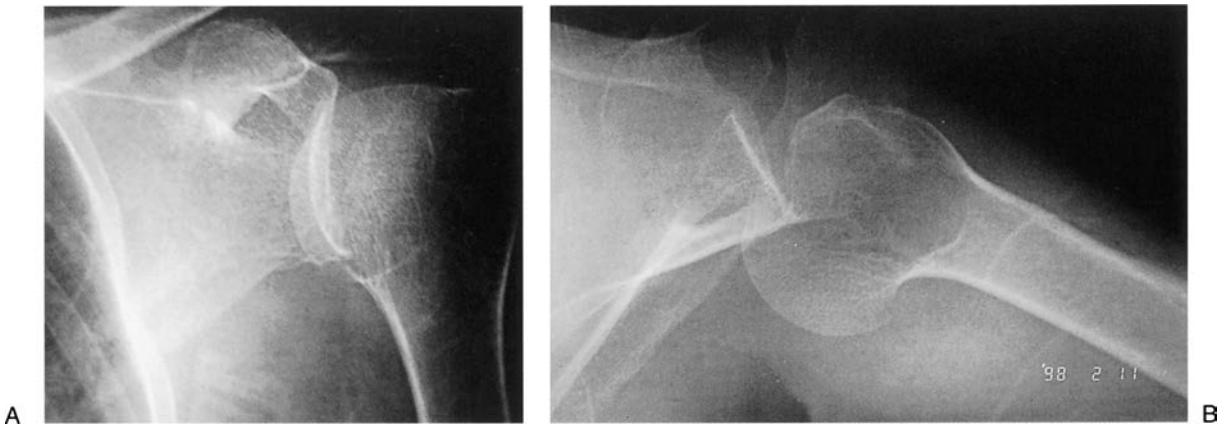

Figure 13-1 **(A)** Anteroposterior and **(B)** axillary radiograph of a locked posterior dislocation. Note the impression fracture of the humeral head and the nondisplaced anatomic neck fracture (*arrow*).

The arm is immobilized in a brace, with the arm in slight abduction and neutral to slight external rotation for 4 to 6 weeks. If there is a large (30% to 50%) humeral head defect and the humeral head dislocates with internal rotation when the arm is in neutral (0 degrees of abduction), then consideration should be given to transfer of the subscapularis, bone grafting, or humeral head replacement (see section on locked posterior dislocation in Chapter 15).

RECURRENT POSTERIOR SUBLUXATION

Volitional Recurrent Posterior Subluxation

Voluntary recurrent posterior subluxation describes a group of patients with an underlying conscious or unconscious ability to subluxate their shoulder by using abnormal patterns of muscular activity. In this group of patients there is no initial anatomic pathology in the glenohumeral joint. Over time, stretching of the glenohumeral ligaments can occur such that an involuntary component to the instability develops. Some of these patients have underlying psychiatric disorders as a cause for willful and voluntary posterior subluxation. Rowe et al. labeled these patients habitual dislocators.[65] Habitual dislocators are distinguished from other patients with posterior subluxation, who may have learned how to reproduce their instability, by their willful desire to subluxate their shoulders (Fig. 13-2). Despite the best intentions of the treating physician, habitual dislocators will frustrate all treatment efforts (operative and nonoperative) because of their abnormal psychologic need to subluxate their shoulder.[65] The overwhelming pathologic process in this group of patients is psychologic, and treatment should be directed according to their psychologic needs. Surgical intervention in this group is contraindicated.

A second group of patients can voluntarily reproduce their instability, but they have no underlying psychologic need to do so. This is a learned behavior that over time may develop an involuntary component. It is this involuntary component that is bothersome to the patient and often initiates evaluation by a physician.

Electromyographic evaluation of patients who can voluntarily subluxate their shoulders demonstrates selective inhibition of certain muscle groups that results in an unbalanced force couple, leading to posterior subluxation. Activation of the deltoid and pectoralis major without opposition from the posterior short rotators, resulting in the humeral head being pushed posteriorly, was identified in several patients.[65] Conversely, Pande et al. demonstrated unopposed activation of the posterior short rotators and posterior deltoid that in effect pulls the humeral head posteriorly.[60]

Dysplastic Recurrent Posterior Subluxation

Dysplastic bony architecture of the glenohumeral joint is another uncommon cause of recurrent posterior subluxation.[19] Localized posterior glenoid hypoplasia, increased glenoid retroversion, and increased humeral head retrotorsion are potential causes of recurrent posterior subluxation. Recent investigations have documented a low incidence of abnormal bony architecture in patients with instability and have postulated that developmental bony deformities are rare causes of recurrent posterior subluxation.[28,63,84] Edelson reported the incidence of posterior glenoid dysplasia in over 11,000 cadaveric specimens studied. In this group as many as 35% of the specimens had deficiencies in the posteroinferior aspect of the glenoid. Even though this condition may be more prevalent than previously thought, it is impossible to correlate these findings with clinical symptoms of instability.[19]

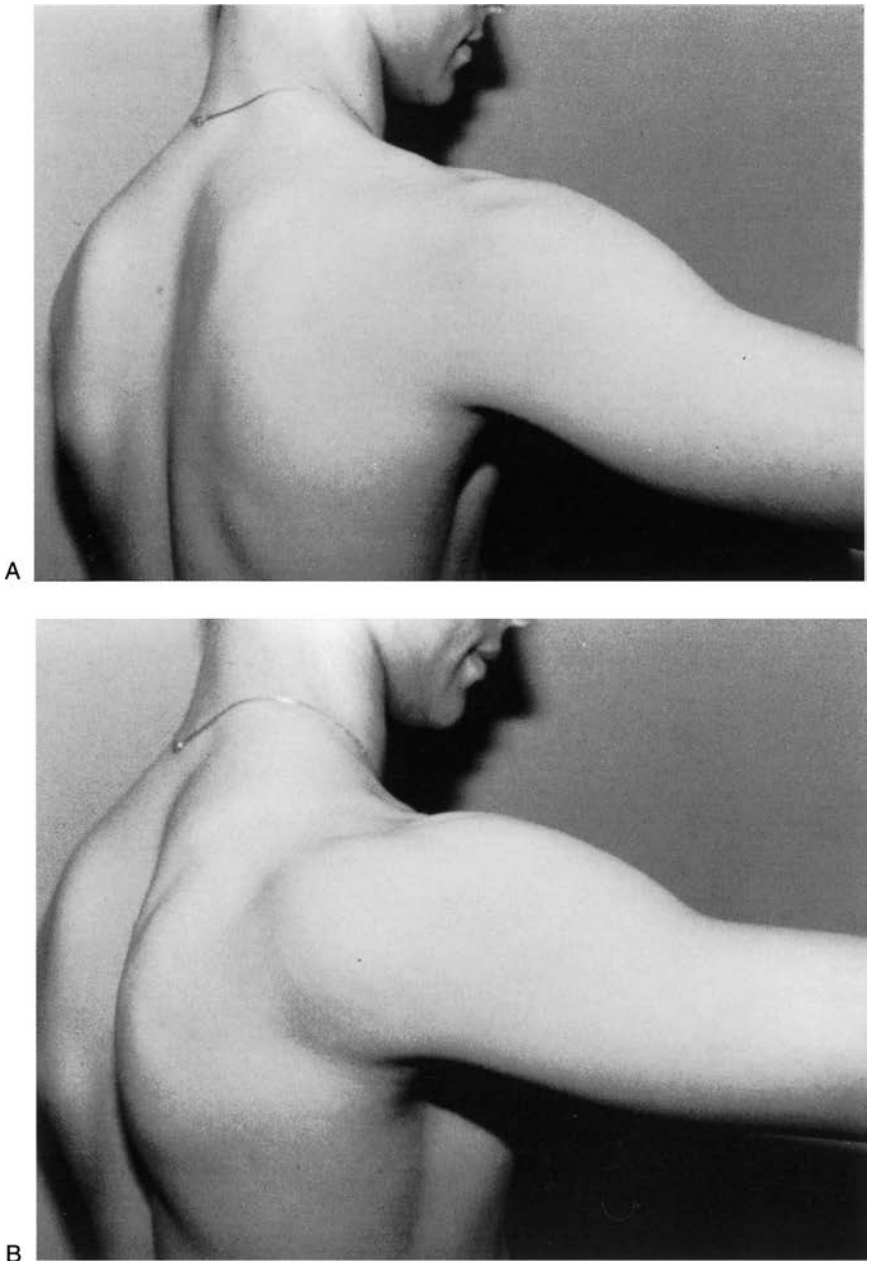

Figure 13-2 Photograph of a patient **(A)** before and **(B)** after dislocating his shoulder posteriorly with asymmetrical muscular contraction.

In the past, the theory that increased glenoid retroversion contributed to recurrent posterior subluxation was supported by radiographic techniques that indirectly measured glenoid retroversion, but are now felt to be inaccurate.[20] The advent of CT has allowed direct measurement of glenoid geometry and has renewed interest in this area. Recent studies based on CT scan assessment of glenoid version vary widely on the incidence of abnormal glenoid geometry and its contribution to instability. Gerber et al. and Randelli and Gambrioli found no correlation between altered glenoid version and instability.[28,63] Conversely, Hurley et al. and Wirth et al. have separately demonstrated increased glenoid retroversion and isolated posterior glenoid hypoplasia, respectively, in all patients with recurrent posterior subluxation reported in their respective series.[38,85] Although the incidence of increased glenoid retroversion or hypoplasia in patients with recurrent posterior subluxation is confused by these conflicting reports, it is clear that increased glenoid retroversion or hypoplasia can contribute in some cases to recurrent posterior instability.[84] The true incidence, however, is unclear.

Although the exact cause of glenoid hypoplasia is unknown, an abnormality in the formation or development of the proximal (subcoracoid) or inferior centers of ossification may be the cause. As these centers do not appear and ossify until well into the second decade of life,

these patients usually do not present with symptoms until their second or third decades.[64] Whereas a CT scan is useful in defining the severity of glenoid hypoplasia, several findings on routine shoulder radiographs have been associated with this condition. Among these are a shallow or irregular glenoid fossa, a prominent coracoid process, an enlarged acromion, hooking of the distal clavicle, an associated hypoplasia of the upper ribs, and flattening of the humeral head.[14,20,74] The appearance and the degree of these radiographic findings vary in patients with this condition.

Abnormalities of humeral torsion could lead to glenohumeral instability. Accurate measurement of humeral retrotorsion is difficult. Schutte et al. described a technique for measuring humeral retrotorsion by CT scan.[67] This allows direct measurements of humeral retrotorsion, thereby eliminating the inaccuracy of plain radiographs. Kronberg and Brostrom have documented a correlation between decreased humeral retrotorsion and anterior glenohumeral instability in some patients. However, a relation between increased humeral retrotorsion and recurrent posterior subluxation has not been established.[45]

Reports of proximal humerus rotation osteotomy for the treatment of recurrent posterior subluxation are not supported by radiographic data demonstrating increased humeral retrotorsion.[14,77] The rationale for osteotomy in these series is that patients with recurrent posterior subluxation can provoke symptoms of instability by internal rotation of the arm. Limiting internal rotation through osteotomy was believed to correct the problem by preventing the patient from placing the arm in a position that would incite dislocation.

Acquired Recurrent Posterior Subluxation

The largest group of patients with recurrent posterior subluxation acquires posterior instability as a result of repetitive microtrauma or as a result of a single traumatic event. Traumatic events leading to both osseous and soft tissue abnormalities can result in subsequent recurrent posterior instability. Because the cause of this instability is not as crucial to treatment as the underlying pathologic lesion that results in recurrent posterior subluxation, we define acquired recurrent posterior subluxation based upon the anatomic lesion. Lesions of the capsule, labrum, rotator cuff musculature, and glenoid can contribute to recurrent posterior subluxation. The most consistent deficiency relates to redundancy of the posterior capsule. Additionally, dysfunction of normal scapulothoracic mechanics can place the glenohumeral joint at risk for recurrent instability.

Unlike the anterior capsule, the posterior capsule is thin. The posterior capsule and the buttress provided by the posterior glenoid labrum are the primary static stabilizers to unidirectional posterior translation. Dynamic posterior stability is conferred by the rotator cuff musculature. The most consistent finding in patients with recurrent posterior subluxation is a patulous posterior capsule.[68] The posterior capsule either stretches over time or tears as a result of single-event trauma and heals in an elongated position, thereby increasing capsular volume. Posterior labral tears have been described with recurrent posterior subluxation; however, they are generally degenerative tears, rather than the rare capsular and labrum avulsion (i.e., reverse Bankart lesion; Fig. 13-3).[12,25,33,35]

The relation between the anterior soft tissues and posterior stability is referred to as the circle concept of capsuloligamentous stability.[68,69,73,76,82] Several biomechanical studies have investigated the contribution of anterior soft tissue structures to posterior stability. In a cadaver model, the soft tissue lesions caused by posterior dislocation have been identified.[52,59] In addition to posterior capsular avulsion and partial or complete tearing of the tendinous portion of the posterior rotator cuff, various lesions of the anterior soft tissue structures were identified. These included complete anterior capsular avulsion from the humeral neck and tears of the muscular portion of the subscapularis.

Selective cutting of soft tissue structures thought to contribute to posterior stability has further defined the role of these anterior and posterior soft tissues to static posterior stability. Increased posterior translation consistently requires a lesion of the posterior capsule, particularly the posterior band of the inferior glenohumeral ligament.[59] Isolated sectioning of the posterior rotator cuff musculature in the absence of a capsular lesion did not increase posterior translation.[58,68] However, when the posterior capsule was sectioned inferiorly, an increase in posterior translation was noted. Subluxation and dislocation occurred only after the anterior capsule and subscapularis tendon were cut.

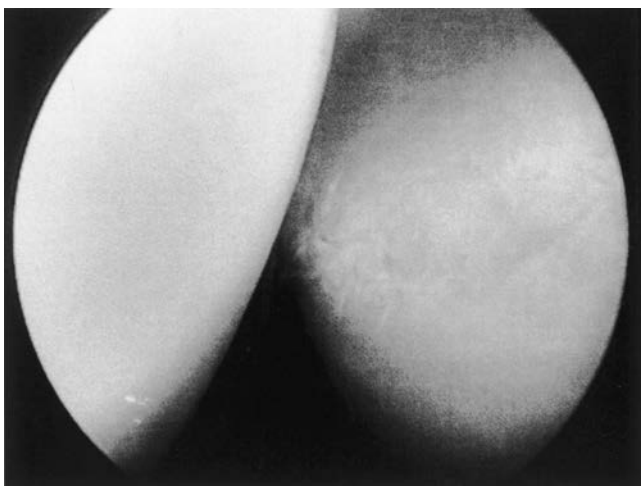

Figure 13-3 Arthroscopic photograph of a degenerative posterior labrum in a patient with recurrent posterior instability. Degenerative lesions of the labrum are typical for recurrent posterior instability. True reverse Bankart lesions are rare.

Several studies have found that the superior capsule (rotator interval capsule) plays an important role in posterior stability.[31,68,69] Sectioning the soft tissues of the rotator interval capsule increased posterior and inferior translation, often to the point of dislocation. Imbrication of the rotator interval increased resistance to posterior and inferior translation.[31]

Acquired posterior subluxation is less commonly caused by posterior glenoid rim deficiency. Although it is uncommon, it does exist and should be investigated with imaging studies if suspected.[25,57,68] The relation between the degree of posterior glenoid erosion and recurrent posterior subluxation has not been established. It seems reasonable to assume that a large posterior glenoid defect will compromise the buttress effect of the glenoid to posterior translation.

In the resting state, the scapula lies on the posterolateral thorax at an angle of 45 degrees.[46] This position on the thorax places the posterior glenoid behind the humeral head, buttressing it against posteriorly directed forces. With shoulder elevation the scapula rotates under the humeral head and provides a platform for glenohumeral motion.[64] A requisite for shoulder stability is that scapulothoracic and glenohumeral rhythm remain synchronous.[15,80]

Dysfunction of scapulothoracic rhythm may compromise the stability of the glenohumeral joint.[80] The serratus anterior muscle plays a key role in scapulothoracic rhythm. Paralysis of this muscle results in scapular winging and loss of power in elevation that potentially may influence glenohumeral stability.[22] Warner et al. used Moire topographic analysis to study patients with glenohumeral instability and demonstrated abnormal scapulothoracic mechanics compared with those of asymptomatic patients.[80] Although no patient demonstrated severe scapular winging, the degree of scapulothoracic dysfunction was variable. In patients with scapular winging from paralysis of the serratus anterior, glenohumeral instability may result from altered scapulothoracic mechanics. In patients with glenohumeral instability and lesser degrees of scapulothoracic dysfunction, it is unclear whether instability is the result of altered scapulothoracic mechanics or the cause of it.

Diagnosis

The most important components in diagnosing recurrent posterior subluxation are a meticulous history and physical examination. Patients typically present with complaints of pain or a sensation of the shoulder dislocating when the arm is placed in a provocative position. The provocative position is variable, but usually includes some degree of flexion, adduction, and internal rotation. An axial load may occasionally be required. In most patients pain is usually limited to episodes of subluxation. Persistent pain is unusual and may be associated with rotator cuff or biceps tendinitis and posterior capsule irritation.[35,78] Pain as a predominant complaint is more common in athletes and may indicate a predisposition to capsular or rotator cuff irritation with overuse during athletic activity.[25,77,78]

A traumatic event initiating recurrent posterior subluxation is not typical. Most often patients cannot recall an initiating event. Over time, with certain activities, they begin to notice shoulder subluxation that readily reduces when the shoulder is taken out of the provocative position. Many of these patients will ultimately learn the position where subluxation occurs and can reproduce the subluxation with specific arm positioning. However, unlike habitual dislocators, patients with nonpsychogenic instability do not voluntarily subluxate their shoulders, unless requested by an examiner, because of the discomfort associated with their instability. In these patients, the most prominent complaint is the involuntary component to the instability that is particularly bothersome and ultimately prompts the patient to seek medical attention.

The disability associated with posterior subluxation is variable and is dependent on the severity of the symptoms. As a general rule, activities of daily living and simple work activities are not limited by symptoms of recurrent posterior subluxation.[33–35] Participation in sports, however, is generally more troublesome, and often requires modification or complete elimination of activity. In more severe cases of posterior subluxation (i.e., patients ultimately requiring surgery), activities of daily living and work may be interrupted.[61]

Physical Examination

The physical examination is directed at reproducing the patient's symptoms and defining the character of instability. Range of motion is generally normal in patients who have not had prior surgery. Occasionally, internal and external rotation may be slightly limited. In athletes there is often activity-specific loss of motion. Overhead-throwing athletes often demonstrate increased external rotation, with an associated mild loss of internal rotation.[77,78] It is critical to evaluate scapulothoracic function during the physical examination for scapular winging or disruption of normal scapulothoracic rhythm.

Most patients can demonstrate their subluxation.[34] Once the position of subluxation is demonstrated by the patient, symptoms of instability can usually be recreated by the physician. In patients who cannot demonstrate their instability, the diagnosis of recurrent posterior instability is more difficult.

Testing for posterior subluxation should be performed with the patient in the sitting and supine position. Testing for increased posterior translation must be performed on the opposite side for comparison. It is important to realize that in a normal shoulder, the humeral head can subluxate posteriorly up to 50%.[30,55]

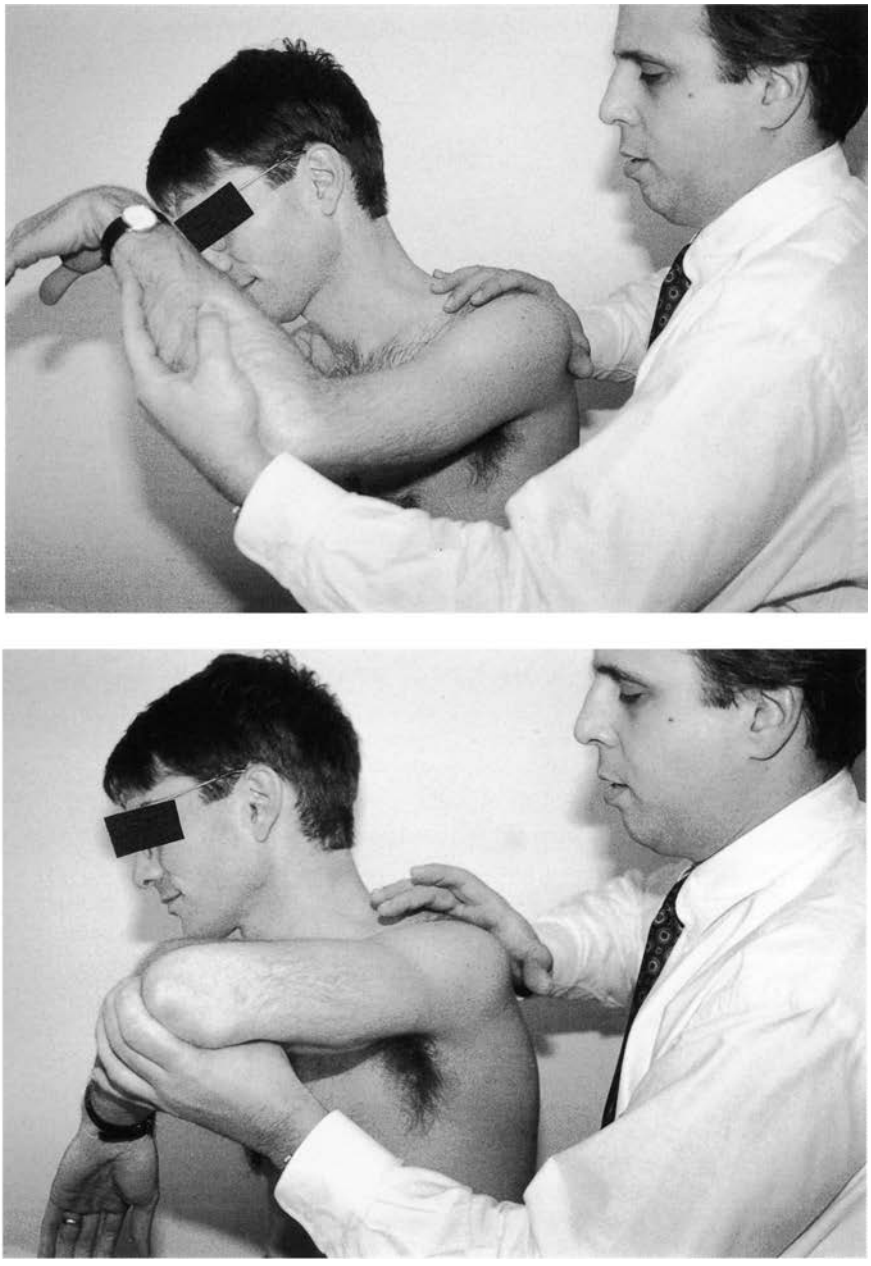

Figure 13-4 Seated posterior stress test: The examiner stands to the side of the patient and stabilizes the scapula. With the arm in 90 degrees of forward elevation in the plane of the scapula, a posteriorly directed force is applied with the arm in **(A)** external rotation and **(B)** internal rotation. The degree of posterior translation in each position is assessed.

The posterior stress test is performed with the patient seated and the examiner at the affected side (Fig. 13-4). The scapula is stabilized while the opposite hand positions the arm in flexion, adduction, and internal rotation and applies a posteriorly directed force. If the patient is able to demonstrate subluxation, replacing the arm in this provocative position should recreate the patient's symptoms with posterior stress testing. Otherwise, testing must be performed in varying degrees of flexion (between 90 and 120 degrees), adduction, and internal rotation to determine the arm position where subluxation occurs. With coronal plane extension, the humeral head will relocate into the glenoid fossa. The relocation is sudden and can be felt by the patient and examiner. Most patients with recurrent posterior subluxation have a positive posterior stress test.[25,61] However, apprehension typical of anterior instability is unusual.[33,34] The predominant symptoms are pain or reproduction of instability symptoms.

The load-and-shift test should be performed with the patient in the sitting and supine positions.[64] In the seated position, with the examiner behind the patient, the scapula is stabilized to minimize scapulothoracic motion (Fig. 13-5). With the opposite hand, the humeral head is grasped and a centering force is applied. Anterior and posterior translation is assessed and compared with that of the opposite side. We prefer the load-and-shift test to be

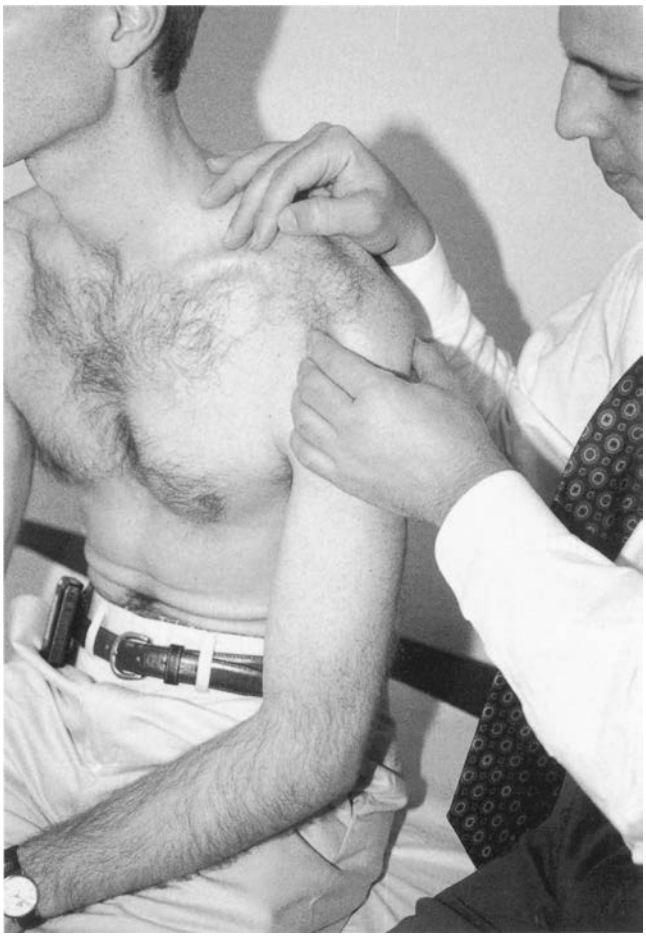

Figure 13-5 Seated load-and-shift test. The examiner is seated at the side of the patient. The scapula is stabilized with opposite hand. A centering force is applied to the glenohumeral joint, and the amount of anterior and posterior translation is assessed.

performed in the supine position with the arm in the plane of the scapula, in 45 to 60 degrees of abduction, with varying degrees of rotation from full external to full internal rotation (Fig. 13-6). Positive testing will result in a reproduction of the patient's symptoms of instability, pain, and crepitation. Side-to-side difference in the amount of internal rotation necessary to obliterate or minimize posterior translation is a clinical measure of residual pathologic capsular laxity.

Inferior translation is assessed next by grasping the elbow, with the arm at the side, and applying an inferiorly directed force. Attention to the region below the acromion will show an indentation, indicating a sulcus sign if inferior instability exists, and should be estimated and recorded in centimeter increments (Fig. 13-7).[64] In most normal patients, passive external rotation will cause a decrease in the sulcus sign, which indicates an intact and functioning rotator interval capsule. Asymmetrical loss of this finding supports the diagnosis of a rotator interval lesion and, therefore, would help explain the cause of the instability.

The zone of instability is most often posteroinferior, but can also be straight posterior or multidirectional. It is important to remember that increased laxity in one direction does not always correlate with, or mean, instability. In patients in whom an isolated posterior component of instability exists, the posterior stress test and load-and-shift test demonstrate subluxation at approximately 80 to 90 degrees of forward elevation. In the more common instance of a posteroinferior instability, subluxation occurs with more forward elevation (110 to 120 degrees). Additionally, a positive sulcus sign may exist. A sulcus sign that is asymmetrically positive identifies the inferior component of the instability and should raise the suspicion for insufficiency of the rotator interval capsule. In both instances, the degree of subluxation may be exaggerated by simultaneous adduction and external rotation. Rotator interval capsular insufficiency may be an isolated process or, more commonly, a component of multidirectional laxity. In cases of multidirectional laxity with instability primarily manifested in the posteroinferior zone, physical signs of generalized ligamentous laxity (i.e., hyperextension of the elbows, knees, metacarpophalangeal joints, or other) are often present. In addition, there is increased humeral head translation in all directions, but symptoms occur primarily with posterior translation. Distinguishing between these cases of isolated posterior, posteroinferior, and multidirectional laxity with a primarily posteroinferior component is important in determining treatment options. Depending on the cause and pattern of instability and the anatomic lesions, the most appropriate treatment may be isolated posterior capsulorrhaphy (reverse Bankart procedure; posteroinferior capsular shift), a bony procedure (bone block or posterior glenoid osteotomy), or an anteroinferior capsular shift combined with rotator interval plication.

Imaging Studies

Routine radiographs of the shoulder should be obtained and include a true AP view obtained in the plane of the scapula, a lateral scapular or Y view, and an axillary view. These radiographs may not demonstrate any abnormalities. However, particular attention should be directed toward the axillary radiograph for evidence of calcification of the posterior capsule, fracture or erosion of the posterior glenoid (Fig. 13-8), or reverse Hill-Sachs defects (Fig. 13-9).[25,56,57]

Stress axillary radiographs or fluoroscopy are generally not necessary, for the history and physical examination usually clarify the diagnosis. However, in the small group of patients for whom doubt of the diagnosis remains, some authors have found these imaging modalities quite helpful.[20,51,55] Comparison with the unaffected side is recommended, for posterior glenohumeral translation of 50% has been demonstrated in normal shoulders.[30,56]

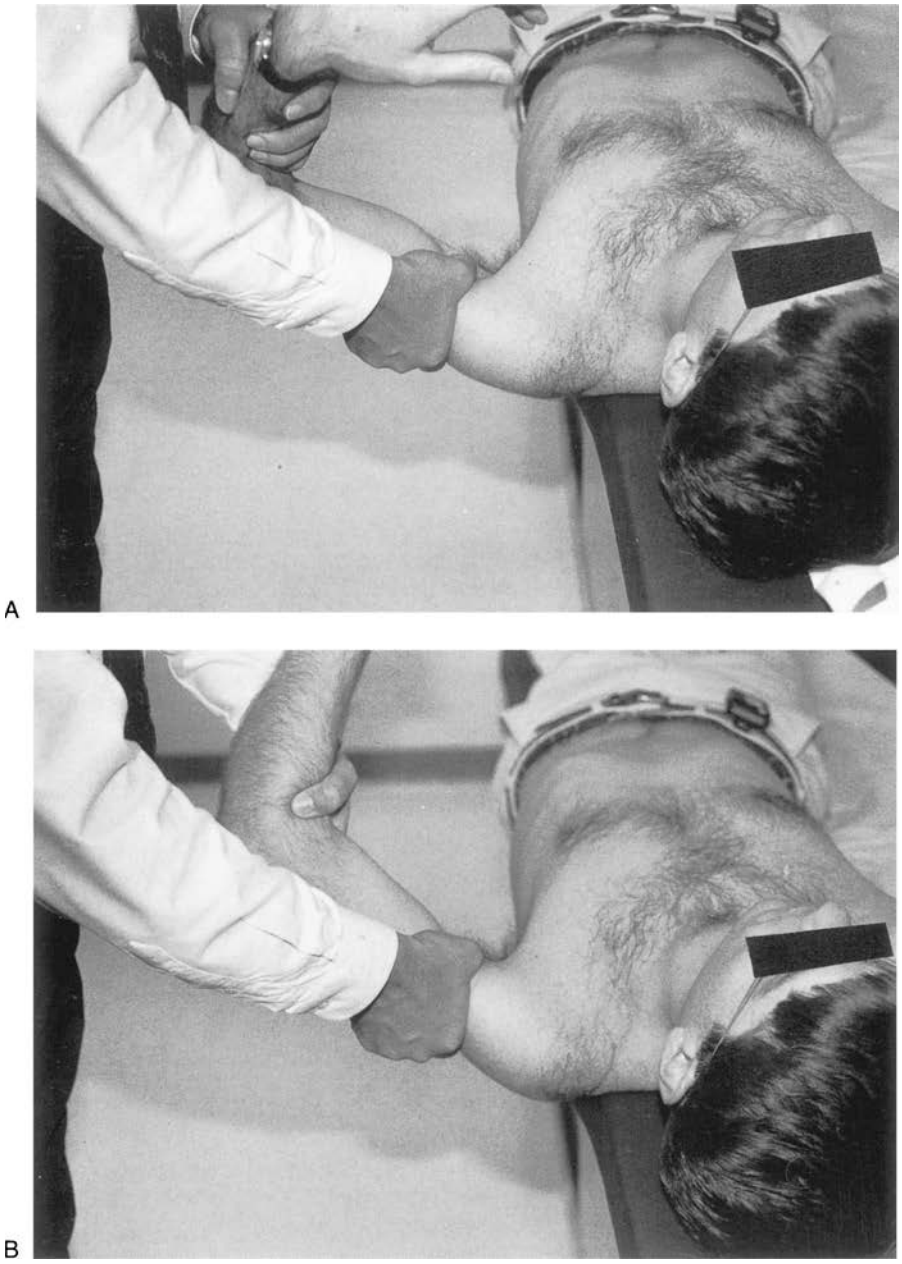

Figure 13-6 Supine load-and-shift test: The patient is supine on the examining table. The arm is brought into approximately 90 degrees of forward elevation in the plane of the scapula. A posteriorly directed force is applied to the humerus with the arm in varying degrees of rotation from **(A)** external rotation to **(B)** internal rotation.

Advanced-imaging studies are not routinely used, but are considered when the specific pathologic lesion underlying recurrent posterior subluxation is unclear. CT excels in its ability to define bony detail. If plain radiographs suggest abnormalities of glenoid version, glenoid hypoplasia, or posterior glenoid erosion, CT or magnetic resonance imaging (MRI) is useful (Fig. 13-10).[28,63]

Computed arthrotomography was developed to better define intraarticular soft tissue pathology and to allow assessment of capsular volume. Whereas Bigliani et al. found one-third of CT arthrotomograms to be over- or underread for labral pathology when correlated with findings at surgery, Callaghan found CT arthrotomography to be 100% accurate, sensitive, and specific for posterior labral defects (Fig. 13-11).[5,11]

MRI has improved our ability to assess soft tissue pathology about the shoulder. The advantages of MRI over plain radiographs and CT include no exposure to ionizing radiation, excellent soft tissue resolution, noninvasiveness, and ability to image in multiple planes. Numerous studies have shown that MRI is superior to other imaging studies at defining labral and capsuloligamentous pathology.[29,37,39,40,42,71] The posterior capsule inserts directly onto the posterior glenoid labrum, not the bony glenoid (Fig. 13-12). This anatomic relation of the capsulolabral complex often confuses accurate interpretation of MRIs. The capsulolabral complex is best

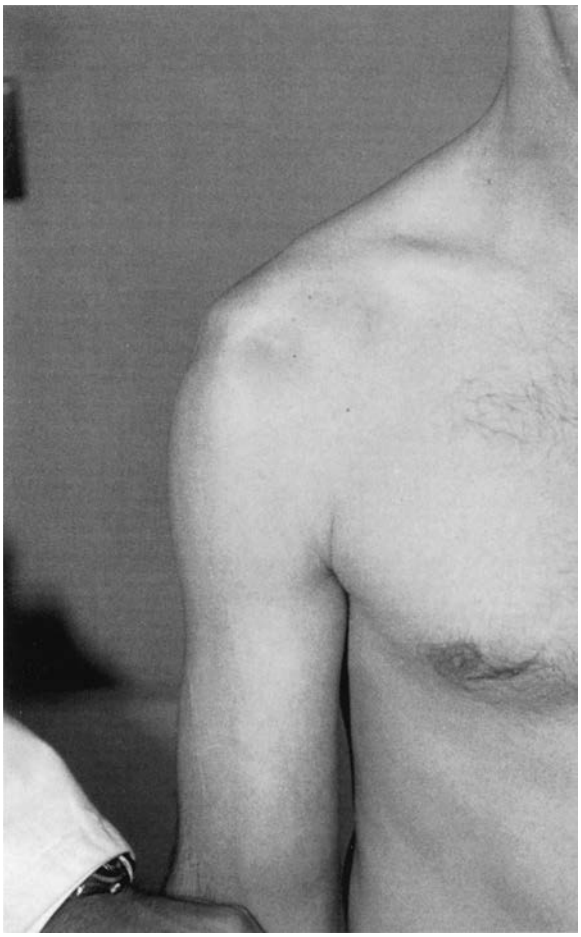

Figure 13-7 Sulcus sign: The examiner is seated next to the patient. The forearm is grasped and an inferiorly directed force is applied to the arm in neutral glenohumeral rotation. Attention is directed to the region immediately inferior to the acromion. A positive sulcus sign will reduce with external rotation of the shoulder in a patient with a competent rotator interval capsule.

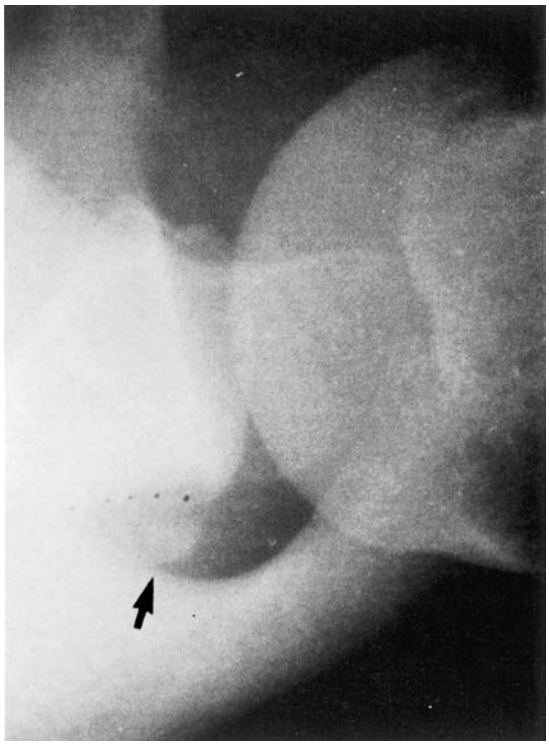

Figure 13-8 Axillary radiograph showing fracture of the posterior glenoid rim (*arrow*). The fracture fragment has healed to the posterior glenoid neck.

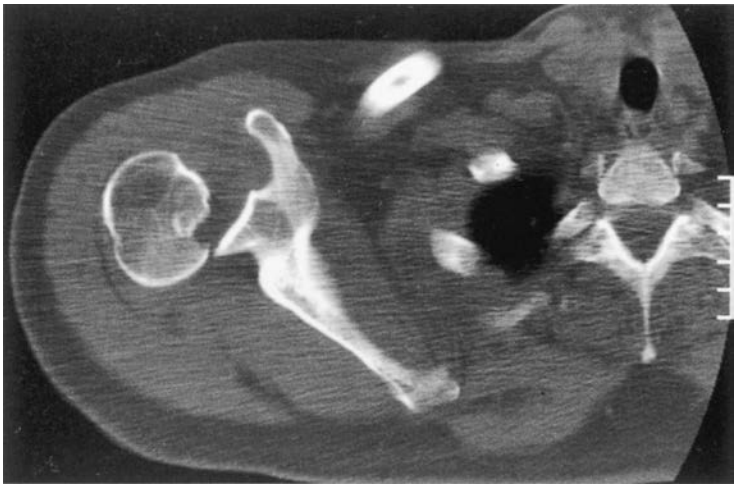

Figure 13-9 A computed tomography scan of a large reverse Hill-Sachs lesion.

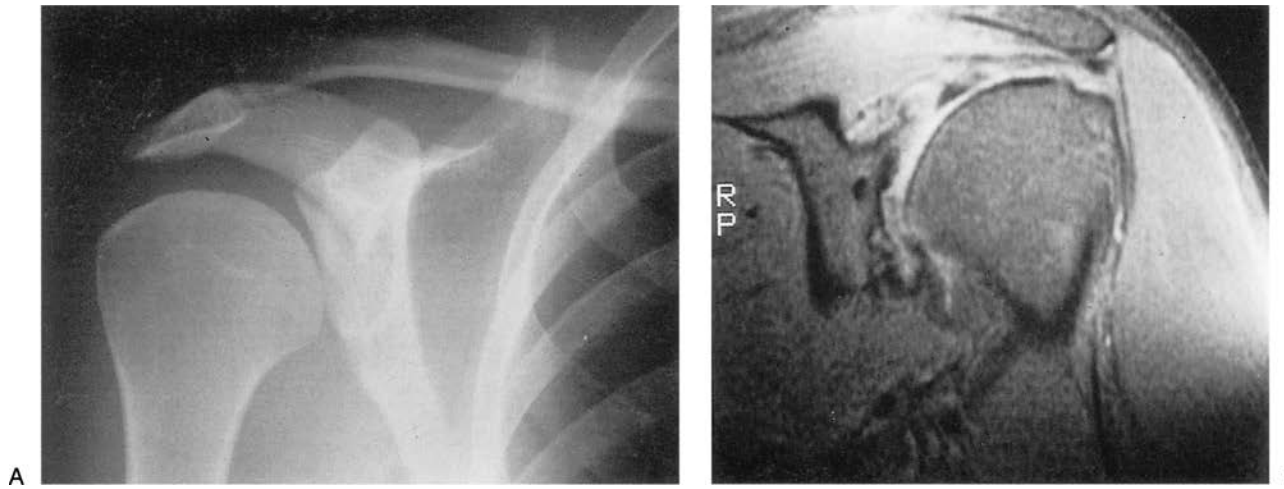

Figure 13-10 **(A)** Anteroposterior radiograph and **(B)** magnetic resonance image of a patient with posterior glenoid hypoplasia with increased glenoid retroversion.

demonstrated with the arm in the neutral position. Imaging in external rotation can simulate a labral tear by creating posterior capsular redundancy at the capsulolabral junction.[74] Additionally, in the nondistended joint, the close proximity of the glenohumeral ligaments to the posterior glenoid labrum can be mistaken for a labral tear.[47,53]

Magnetic resonance arthrography (MRA) has been more sensitive at detecting labral pathology than MRI.[13,23] Distention of the joint with contrast affords better visualization of the glenoid labrum and glenohumeral ligaments. Chandnani et al. found that MRA more consistently detects labral tears, detached labral fragments, and labral degeneration than conventional MRI and CT arthrography.[13]

Advanced-imaging techniques have evolved rapidly and afford better definition of intraarticular pathology than do standard radiographic techniques. However, the information obtained can be misleading if not considered in context with the history and physical examination. These studies should be used to confirm the presence of suspected, specific pathologic lesions, rather than as a screening tool.

Examination Under Anesthesia

Before beginning definitive surgical stabilization, examination under anesthesia (EUA) is useful to confirm one's clinical suspicions, even in the most clinically obvious cases of recurrent posterior instability. The sensitivity and specificity of EUA is improved by examining the shoulder in various positions of shoulder elevation and rotation.[16] By placing the arm in 45 to 60 degrees of abduction and varying the degree of humeral rotation, the posterior band of the inferior glenohumeral ligament and posterior capsule are placed under varying degrees of tension. With the

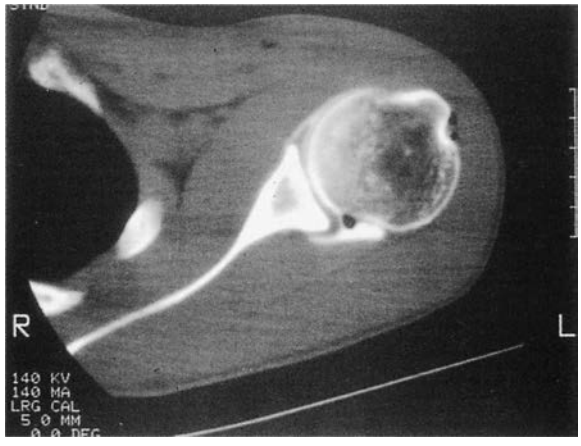

Figure 13-11 A computed tomographic arthrogram of the shoulder. Intraarticular contrast improves visualization of the glenoid labrum.

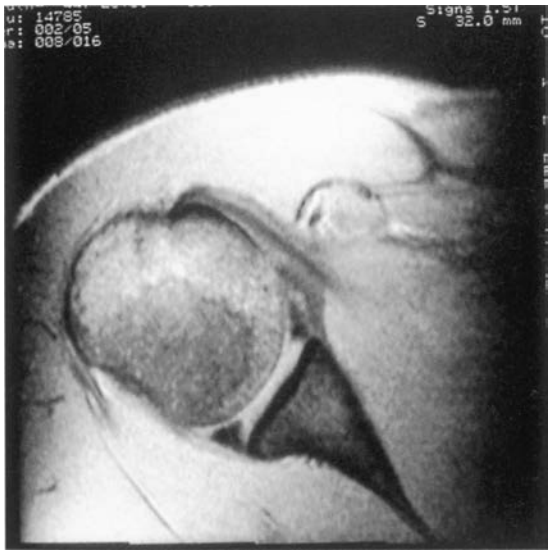

Figure 13-12 A magnetic resonance image demonstrating the posterior capsule inserting on the labrum, not the glenoid.

arm in external rotation, the posterior capsular structures are lax, allowing posterior translation of the humeral head against a posteriorly directed force. Progressive internal rotation tightens these structures, resulting in capture of the humeral head and elimination of posterior translation against a posteriorly directed force. It is important to note the degree of internal rotation at which posterior translation is minimized. Differences in posterior translation in different arm positions when compared with the opposite shoulder are indicative of posterior capsular insufficiency. With the patient in the supine position, the superior capsular structures are tested by applying inferior traction while loading the joint. A sulcus sign is demonstrated and quantitated using the acromion as a reference. Progressive external rotation tightens the superior capsular structures and should eliminate any sulcus sign present in internal rotation if the superior capsular structures are competent.

In those circumstances under which a high degree of suspicion exists for posterior subluxation, one may further consider diagnostic arthroscopy after an equivocal EUA. Although not as helpful in detecting specific pathologic lesions, such as anterior instability, observation of an incompetent posterior inferior glenohumeral ligament or an excessively redundant posterior capsule may assist one in defining the pathology present before attempting posterior stabilization in special situations.[16]

TREATMENT

The diagnostic workup along with the treatment algorithm employed by our Shoulder and Elbow Service for recurrent posterior subluxation is summarized in Fig. 13-13.

Nonoperative

The recommended initial treatment by most authors for symptomatic recurrent posterior subluxation is nonoperative.[10,25,34,48,57,61,78] Nonoperative treatment should include activity modification; psychologic counseling for patients with voluntary, psychogenic recurrent posterior subluxation; and a shoulder-strengthening program for the dynamic muscular stabilizers, including the rotator cuff (especially infraspinatus and teres minor), posterior deltoid, and scapular stabilizing muscles. Activity modification is aimed at preventing further injury or stress to the posterior capsule, labrum, or rotator cuff. Activities that place the shoulder in the provocative position of forward elevation, adduction, and internal rotation are best avoided during rehabilitation.

Strengthening is accomplished through resisted external rotation exercises with rubber bands of increasing resistance or free weights, and in time, may progress to isokinetic exercises. It is important to balance the strengthening program with internal rotation exercises. Additionally, periscapular strengthening is vital to reestablish synchronous scapulohumeral rhythm.[50]

Patients who fail a prolonged trial of nonoperative therapy and remain symptomatic should be considered for surgical stabilization. Some authors have found that patients with incapacitating symptoms do poorly with nonoperative management compared with patients with moderate symptoms and suggest early surgical intervention.[7,25] Surgery should be directed at correcting the underlying pathologic lesion causing recurrent subluxation. This underscores the value of an anatomically based classification system, rather than the cause-based system currently favored.

Surgical Options and Rationale for Surgical Treatment

Numerous posterior procedures have been described for recurrent posterior subluxation. These procedures include posterior capsulorrhaphy, with or without a bone block; posterior bone block alone; glenoid osteotomy; posterior infraspinatus capsular tenodesis; and posteroinferior capsular shift.[3,5,8,9,24,25,41,44,49,51,52,54,57,61,70] Historically, the results of surgery have been poor, with recurrence rates of up to 50% with significant complications.[34,77] The use of procedures when the pathology of recurrent posterior subluxation was not well understood preoperatively or not completely addressed at surgery no doubt contributed to these poor results. Recently, surgical techniques have been employed that anatomically correct the underlying pathology. This has resulted in more-encouraging results.[6,25] Consequently, the importance of accurately defining the pathologic lesion preoperatively cannot be overstated. The specific surgical techniques are determined by the underlying pathology in each particular case.

Arthroscopic evaluation of shoulders with recurrent posterior instability has the added benefit of identifying anterior joint pathology that would not be visible from an open posterior approach of the shoulder. Technologic advances have permitted all soft tissue pathology identified arthroscopically to be managed arthroscopically. Bone involvement contributing to recurrent posterior instability is not as successfully managed arthroscopically.

Acquired recurrent posterior subluxation is most often caused by soft tissue deficiency (capsule, labrum, or both), erosion or deficiency of the posterior glenoid, or scapulothoracic dysfunction. A patulous posterior capsule is usually the primary pathologic lesion. If a reverse Bankart lesion coexists with a redundant posterior capsule, a combined repair of the reverse Bankart lesion and posterior capsular plication or shift is necessary. When posterior capsular redundancy or detachment is combined with erosion of the posterior glenoid rim or increased glenoid retroversion (i.e., hypoplasia), the posterior capsular procedure is combined with posterior glenoid bone graft or posterior opening wedge osteotomy, respectively. Posterior bone block may

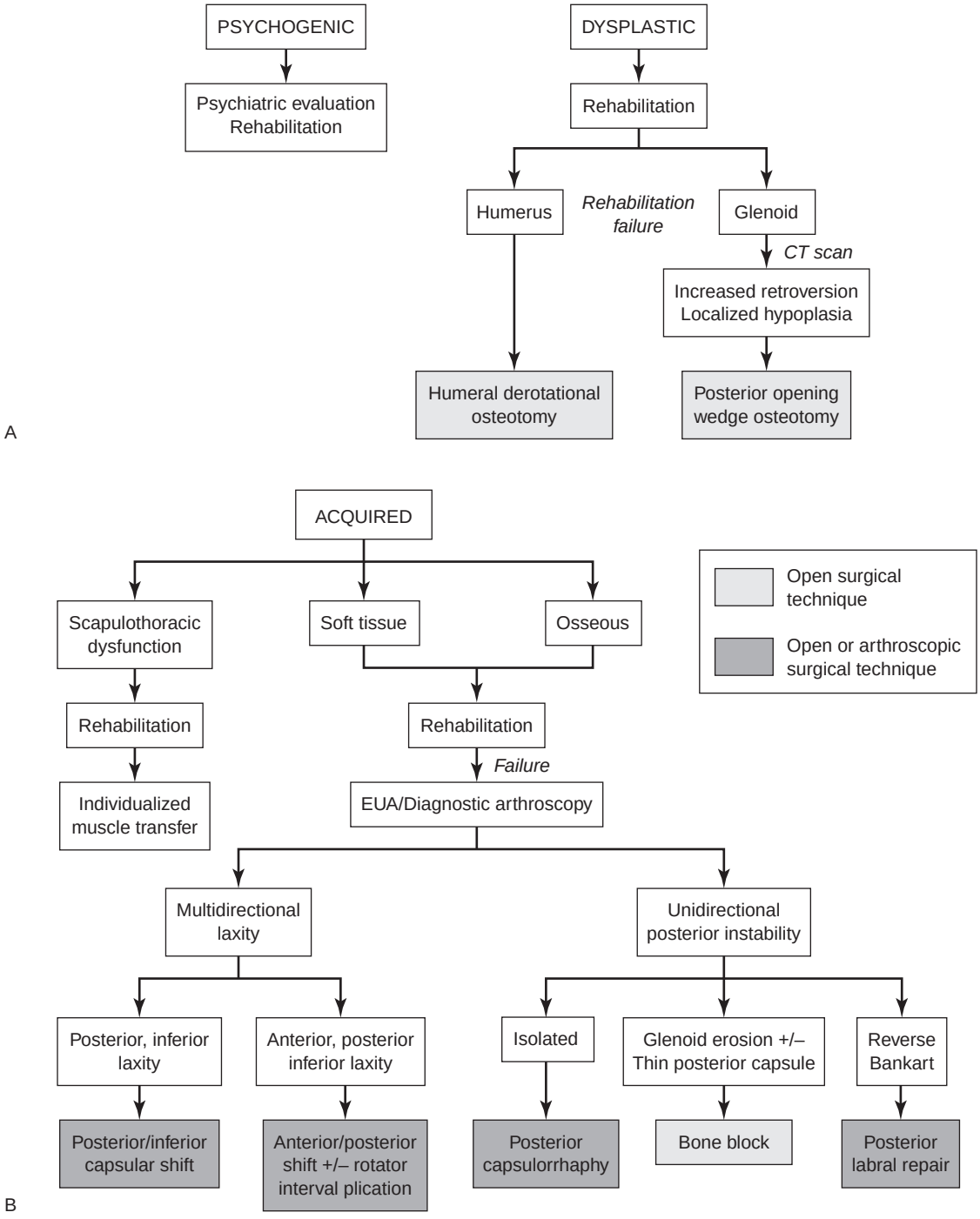

Figure 13-13 University of Pennsylvania treatment algorithm for recurrent posterior subluxation. **(A)** Psychogenic and dysplastic recurrent posterior subluxation and **(B)** acquired posterior subluxation.

also be indicated for properly performed but failed capsular procedures, even if the glenoid architecture is normal.

Patients with recurrent posterior subluxation due to loss of soft tissue restraint are considered for arthroscopic repair. Contraindications for arthroscopic posterior repair include bone abnormalities and volition. Patients with excessive glenoid retroversion may need bone alteration (i.e., osteotomy) to correct posterior subluxations. This includes patients with traumatic unidirectional posterior instability due to a tear or detached restraint.

Patients that have multidirectional instability have symptomatic inferior subluxation and either anterior or posterior symptoms, or both. Many patients with posterior and inferior symptomatic subluxation are termed "bidirectional" or posterior symptomatic multidirectionally unstable patients.

It is critical to determine the direction of instability before surgery. If instability is only posterior, a procedure addressing the redundant posterior capsule is warranted. However, posterior subluxation is often accompanied by varying degrees of inferior and multidirectional laxity. In cases of posteroinferior subluxation when there is no anterior component to the instability pattern and the rotator interval capsule is functionally intact, a posteroinferior capsular shift from a posterior approach should be performed. In patients with multidirectional laxity, the surgical approach depends on the primary location of their symptoms. If their symptoms are directed to the posterior aspect of the shoulder, we prefer to approach these posteriorly and perform a posteroinferior capsular shift. Conversely, anterior symptoms are addressed with an anteroinferior capsular shift. In the rare patient with true multidirectional instability with anterior and posterior symptoms, combined anterior and posterior capsular procedures may be necessary.

Surgical treatment of scapular winging is beyond the scope of this chapter. However, the importance of restoring scapulothoracic mechanics in controlling posterior instability cannot be overemphasized. Scapular winging can be an important contributor to recurrent posterior subluxation. When caused by long thoracic nerve injury, pectoralis major transfer, alone or in combination with posterior capsulorrhaphy, may be indicated.[62] In these cases, it is often difficult to determine the need for posterior capsulorrhaphy in addition to the pectoralis major transfer. In most instances, pectoralis major muscle transfer alone is sufficient to correct the posterior instability symptoms.

Glenoid or humeral dysplasia is an uncommon cause of posterior subluxation. However, in the presence of glenoid hypoplasia or increased glenoid retroversion, a glenoid osteotomy should be considered.[70] Humeral rotational osteotomy is considered only when there is documented abnormal humeral retrotorsion.[14,75]

Patients with a psychologic cause for recurrent posterior subluxation should be managed with an exercise program combined with psychologic counseling.[65] Surgery is contraindicated as long as the underlying psychologic needs leading the patient to voluntarily subluxate his or her shoulder exist. Surgery should be considered only after the underlying psychologic problems are resolved and the patient still demonstrates symptomatic involuntary recurrent posterior subluxation. Extreme caution is still warranted in performing surgery in this group of patients. If any element of psychogenic posterior subluxation remains, surgery is doomed to failure.

Open Surgical Technique

Patient Positioning

The patient is positioned in the lateral decubitus position and secured with anterior and posterior posts or a bean bag. The head and neck should be supported in neutral position and the knees and ankles padded. Impervious drapes are placed at the base of the neck and around the axilla, medial to the vertebral border of the scapula, and a minimum of 6 cm medial to the anterior axillary crease (Fig. 13-14). The arm is prepared and draped free. The arm may be supported anteriorly on a Mayo stand, or an intraoperative sterile shoulder positioner (McConnell Orthopedic Manufacturing Co., Greenville, TX) may be used.

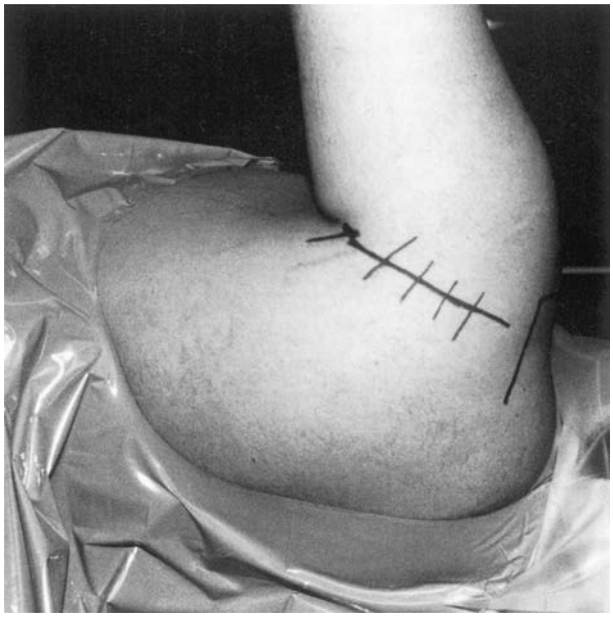

Figure 13-14 Intraoperative photograph demonstrating patient positioning and draping in the lateral decubitus position. The shoulder should be draped widely to allow palpation and visualization of topographic structures that will aid in dissection.

Surgical Approach

The incision begins at the posterior axillary crease and extends superiorly to the spine of the scapula (Fig. 13-15). Medial subcutaneous dissection is carried out to the border of the posterior deltoid. The lateral subcutaneous dissection is to the lateral border of the acromion. The fibers of the posterior deltoid are split in that portion of the deltoid that overlies the posterior glenohumeral joint line, from the spine of the scapula distally for a distance of 4 to 5 cm. Blunt dissection of the posterior deltoid fibers is carried deep to the subdeltoid bursa, exposing the underlying infraspinatus and teres minor muscles (Fig. 13-16). An alternative approach for exposure of the infraspinatus and teres minor muscles is to elevate the posterior deltoid along its inferior margin. Identification of the inferior border of the posterior deltoid allows this margin to be defined. Dissection to the subdeltoid space and abduction of the shoulder facilitate superior retraction of the posterior deltoid. Deep retractors are placed to expose the infraspinatus and teres minor tendons (Fig. 13-17). Inferior to the teres minor is the quadrangular space that

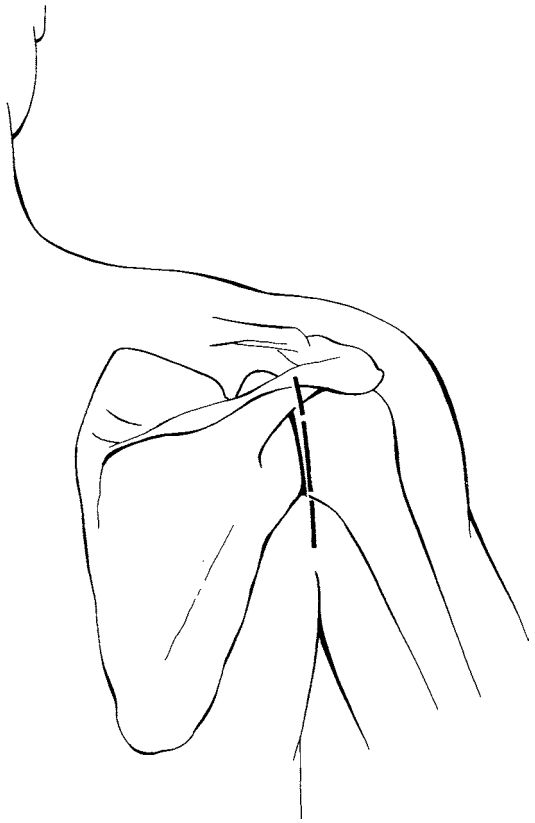

Figure 13-15 Drawing of incision for posterior shoulder surgery. A vertical incision is preferred for cosmetic reasons. The incision begins at the spine of the scapula and extends to the posterior axillary crease.

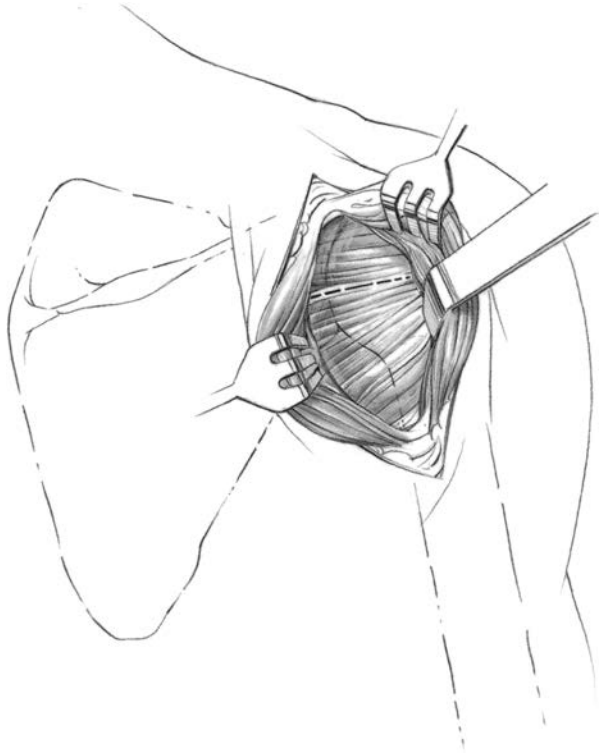

Figure 13-16 The longitudinal split in the deltoid should be performed so that the operative field is over the center of the glenohumeral joint, exposing the infraspinatus and teres minor muscles.

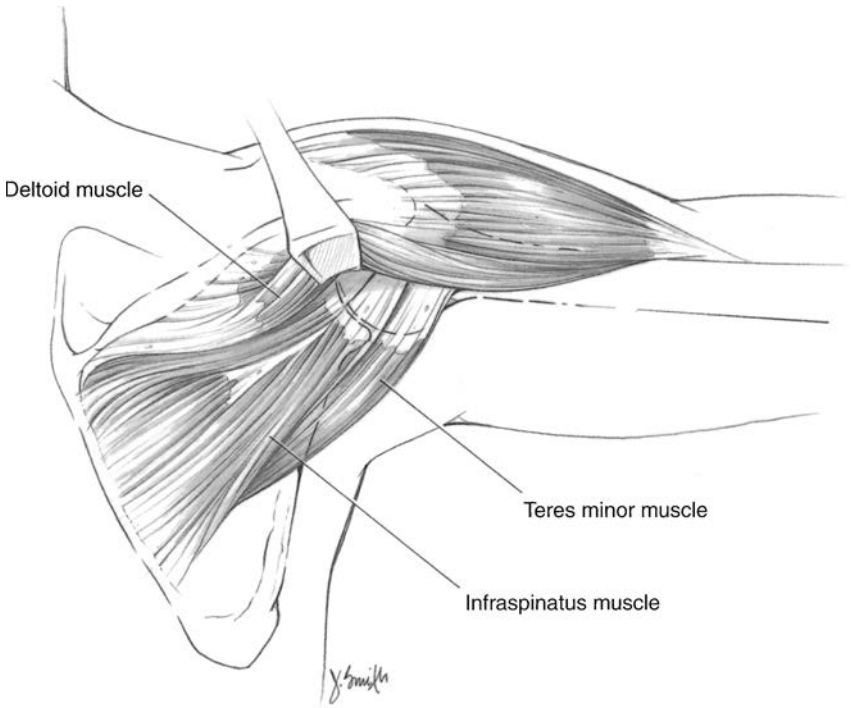

Figure 13-17 Superior elevation of the deltoid is an alternative to a deltoid split. The arm must be placed in 90 degrees or more of elevation to allow the deltoid to be retracted cephalad.

contains the axillary nerve and posterior humeral circumflex vessels.

Exposure of the posterior capsule can be accomplished either in the internervous plane between the infraspinatus (suprascapular nerve) and teres minor (axillary nerve) or within the posterior fat stripe that separates the upper and lower portions of the infraspinatus muscle.[72,83] The fat stripe commonly seen between the upper and lower halves of the infraspinatus muscle is located at the midequator of the glenohumeral joint. Dissection through the infraspinatus fat stripe will place the operative procedure more central within the glenohumeral joint and, thereby, facilitate better exposure of the upper portion of the joint. It is possible to perform posterior capsulorrhaphy through this interval without detaching the infraspinatus insertion, and it is our preferred technique. In this approach, this interval is developed to the posterior capsule (Fig. 13-18). Dissection superiorly to the supraspinatus tendon and inferiorly to the posteroinferior aspect of the joint is undertaken, and reverse Homans retractors are placed to expose the capsule. Medial dissection along the infraspinatus muscle is limited to 1.5 cm medial to the glenoid margin to avoid injury to the inferior branch of the suprascapular nerve that would denervate the inferior portion of the infraspinatus muscle. Medial exposure is the primary limitation of this approach.

The most extensile exposure of the posterior capsule is obtained by dissecting the interval between the infraspinatus and teres minor. This interval can be extended medially without fear of denervating the infraspinatus (Fig. 13-19). The infraspinatus muscle and tendon are then bluntly dissected from the underlying capsule. The thickness of the infraspinatus tendon can then be assessed. The midtendinous portion of the infraspinatus tendon is then incised to the level of the capsule, approximately 1 cm medial to its humeral attachment. Further blunt dissection is performed to separate the infraspinatus tendon from the capsule superiorly, medial to the glenoid rim and laterally toward the greater tuberosity. The infraspinatus is then reflected medially. Caution should be used in retracting the infraspinatus medial to the glenoid rim to avoid excessive traction on the suprascapular nerve at the spinoglenoid notch. Using a narrow blunt elevator, the posteroinferior capsule is dissected from the underlying teres minor. A narrow, deep blunt retractor is placed deep to the teres minor to expose the posteroinferior capsule.

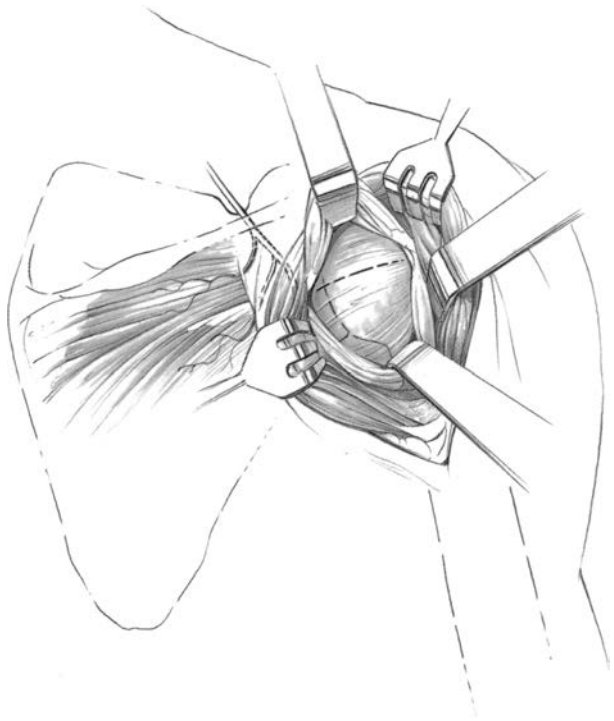

Figure 13-18 Posterior capsular exposure through a fat stripe split between the upper and lower portion of the infraspinatus. Medial dissection is limited to 1.5 cm medial to the glenoid so that injury to the inferior branch of the suprascapular nerve is avoided.

Posterior Capsulorrhaphy (Author's Preferred Technique)

The capsule is incised in a medial to lateral direction midway between the superior and inferior poles of the glenoid. Traction sutures may be placed in the superoinferior portions of the capsule. A humeral head retractor is inserted, and the glenoid rim is inspected. When a capsular avulsion is not identified, then the vertical capsular incision is performed approximately 5 mm medial to the humeral insertion site of the capsule (Fig. 13-20). The capsulotomy is performed superiorly to the posterior insertion site of the supraspinatus tendon. The inferior extent of the lateral capsulotomy is dependent on the degree of inferior capsular redundancy. In general, the lateral capsulotomy is carried out past the inferiormost portion of the inferior capsular pouch to the 6 o'clock position. The arm is then placed in the plane of the scapula in 45 degrees of abduction and neutral rotation to approximately 15 degrees of external rotation. The inferior leaflet is then shifted superiorly to obliterate the inferior capsular pouch and place the inferior leaflet under slight tension. Lateral capsular closure is accomplished with nonabsorbable sutures. The superior capsular flap is then shifted inferiorly to the point of slight tissue tension.[52] Lateral capsular closure is achieved with nonabsorbable sutures. The interval between the superior and inferior flaps is then closed and reinforced with nonabsorbable sutures (Fig. 13-21).

If the infraspinatus tendon was cut, it is then closed with slight overlap of the tissue using nonabsorbable sutures. If it was not incised, the infraspinatus is allowed to retract to its normal position and the fascia is closed with absorbable sutures. The deltoid is allowed to retract to its normal position. Absorbable suture is placed in the superficial fascia, and a drain is inserted in the depth of the wound. The subcutaneous tissues are closed with absorbable suture and the

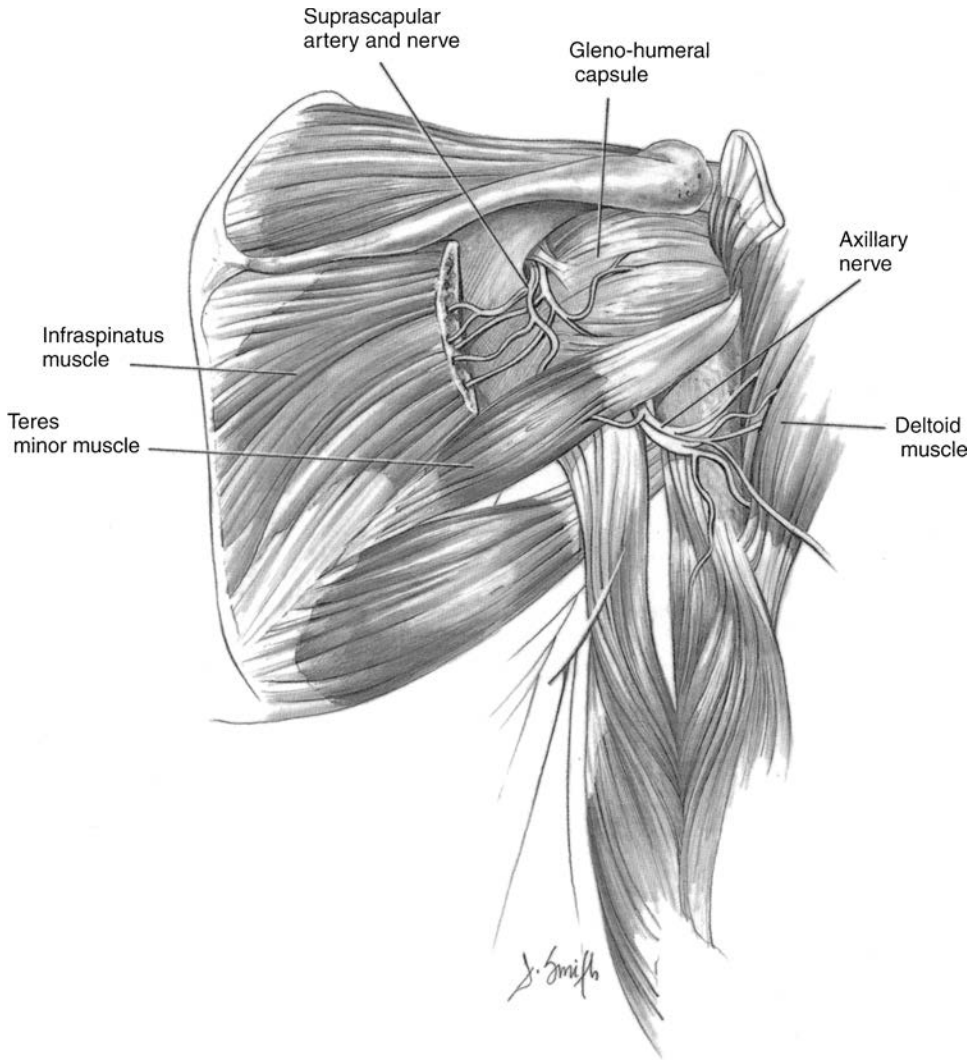

Figure 13-19 Posterior capsular exposure through the interval between the infraspinatus and teres minor muscles. This is an extensile exposure to the posterior aspect of the shoulder. Vigorous medial retraction of the infraspinatus should be avoided to prevent traction on the suprascapular nerve at the spinoglenoid notch.

skin with nonabsorbable suture using the subcuticular technique. Steri-Strips and a sterile dressing are placed and the arm is positioned in a prefabricated prefitted thoracobrachial orthosis. The arm is positioned slightly posterior to the coronal plane of the thorax in 20 degrees of abduction and 10 degrees of external rotation.

Posterior Labral Repair

The patient positioning and surgical exposure are described above. The horizontal capsulotomy is performed, a humeral head retractor is inserted, and the joint is inspected. If a reverse Bankart lesion is identified, the scapular neck must be prepared and the capsule reattached to the glenoid rim. The vertical capsular exposure is made by dissection of the Bankart lesion, thereby reflecting the capsulolabral tissue from the glenoid margin. The posterior glenoid and scapular neck are then decorticated with a curette or power burr. The labrum is reattached to the articular margin of the posterior glenoid using commercially available suture anchors or transosseous tunnels (Fig. 13-22).

A posteroinferior capsular shift is performed, with repair of the reverse Bankart lesion if excessive posteroinferior capsular redundancy exists. The capsular shift can be performed on the glenoid side when the labrum is repaired or on the humeral side after the labrum is repaired. The humerus is positioned in less external rotation (10 degrees) to avoid overtightening. If the shift is performed on the glenoid side, the capsule and labrum are cut at the midglenoid, creating both a superior and inferior flap. The inferior capsular leaflet is then shifted superiorly to obliterate the inferior

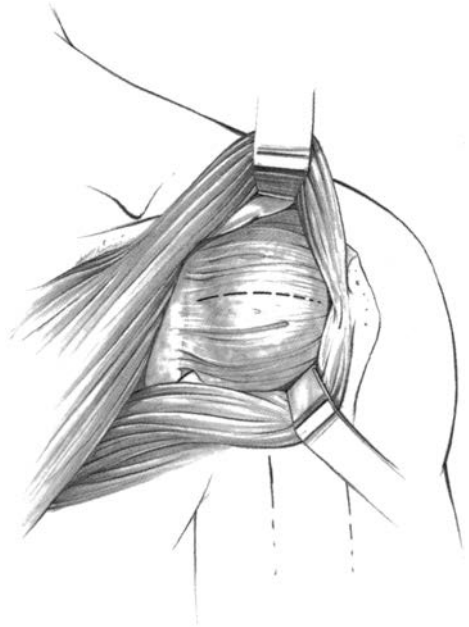

Figure 13-20 A horizontal capsulotomy is performed at the mid-equator of the joint, allowing intraarticular inspection. The lateral vertical limb of the capsulotomy is performed 5 mm medial to the humeral attachment when no posterior labral pathology is noted.

capsular pouch and place the inferior leaflet under slight tension. The inferior leaflet is repaired to the glenoid margin with suture anchors placed at the glenoid margin. The superior leaflet is subsequently shifted inferiorly to a point of slight tissue tension. The sutures used to secure the inferior leaflet to the glenoid margin are used to secure the superior leaflet (Fig. 13-23). Infraspinatus closure, skin closure, and bracing are performed as discussed in the foregoing section.

If the capsular shift is performed on the humeral side in the presence of a reverse Bankart lesion, the capsule is incised at the midglenoid level to, but not through, the labrum. The labrum is repaired anatomically (without shift) to the glenoid margin using suture anchors or transosseous tunnels (see Fig. 13-22). The capsule is then incised vertically 5 mm medial to its humeral insertion site. The arm is positioned in the position of 45 degrees of abduction, and neutral to 10 degrees of external rotation, and a posteroinferior capsular shift procedure is performed, as previously described (see Figs. 13-20 and 13-21). This is our preferred technique when a capsular shift is required in the presence of a reverse Bankart lesion.

Posterior Infraspinatus Capsular Tenodesis

Patient positioning and exposure are described previously in this chapter. Once the deltoid is split or elevated, the underlying infraspinatus is exposed. The arm is then placed in neutral position and a 2.5-cm vertical incision is made through the infraspinatus and underlying capsule, centered on the superior and inferior glenoid margins. This is medial

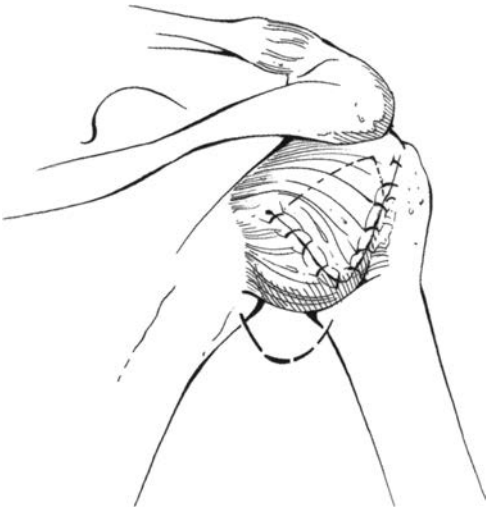

Figure 13-21 Posteroinferior capsular shift: The inferior leaflet is shifted superiorly to slight tissue tension with the arm positioned in approximately 45 degrees of abduction and neutral to slight external rotation. The superior leaflet is subsequently shifted inferiorly.

to the insertion of infraspinatus and leaves a lateral cuff of infraspinatus and underlying capsule for tenodesis (Fig. 13-24). The posterior labral tissue is then identified. If a posterior labral detachment (i.e., reverse Bankart) is found, it is repaired with sutures or a suture-anchoring system, as previously described. The lateral flap of infraspinatus and capsule are then mobilized and secured to the posterior glenoid

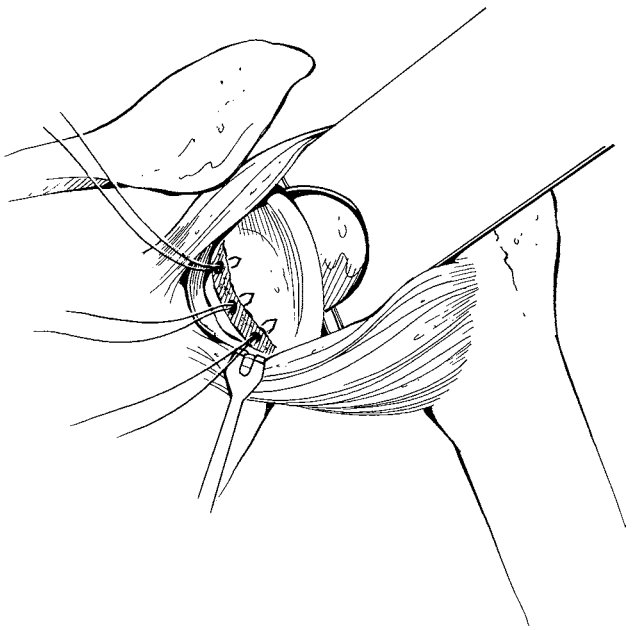

Figure 13-22 Posterior labral repair: The horizontal capsulotomy is performed. A humeral head retractor and forked retractor provide exposure to the posterior glenoid neck. The glenoid neck is decorticated and suture anchors or transosseous sutures are placed along the glenoid margin, passed through the detached labral tissue, and tied, thereby reestablishing the normal anatomy of the posterior labrum and glenoid.

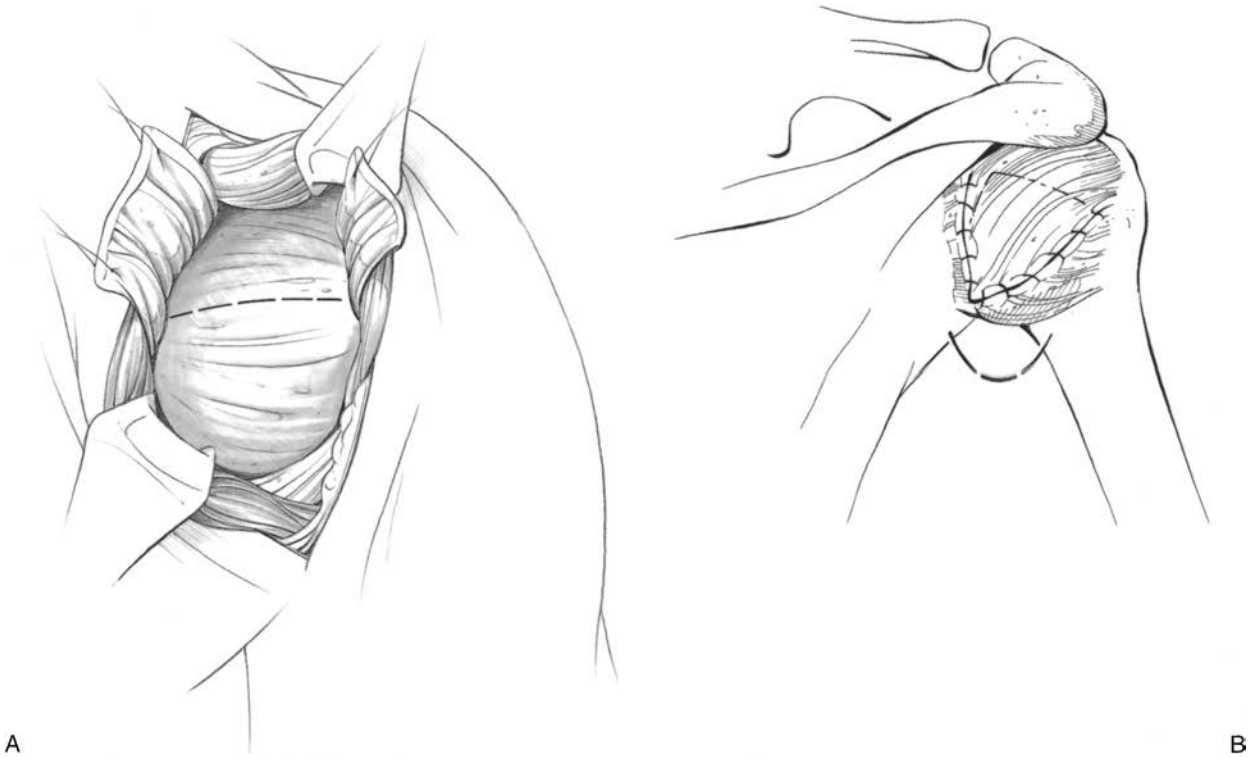

Figure 13-23 Posterior labral repair with medial-based capsular shift: **(A)** The horizontal capsulotomy continues through the capsule and labrum. **(B)** The inferior capsular leaflet and labrum are shifted superiorly to slight tissue tension with the arm in 45 degrees of abduction and neutral to slight external rotation. The superior capsular leaflet and labrum are then shifted inferiorly.

labrum with nonabsorbable suture, with the arm in slight external rotation (Fig. 13-25). The remaining portion of infraspinatus and underlying capsule is overlapped over the primary repair with the arm in neutral to slight internal rotation. The deltoid is then allowed to retract back to its normal resting position and the subcutaneous tissue is approximated with absorbable suture. The skin is closed with a nonabsorbable suture in a running subcuticular fashion.[32]

Posterior Bone Block

Patient positioning and exposure are as described previously. The capsule is incised in the medial lateral direction at the midlevel of the capsule and the joint is inspected. A 2 × 2-cm, 7- to 10-mm–thick bone graft is obtained from the posterior iliac crest or posterior scapular spine (Fig. 13-26). The posterior glenoid neck is exposed and the inferior half is decorticated. The capsular–labral attachment

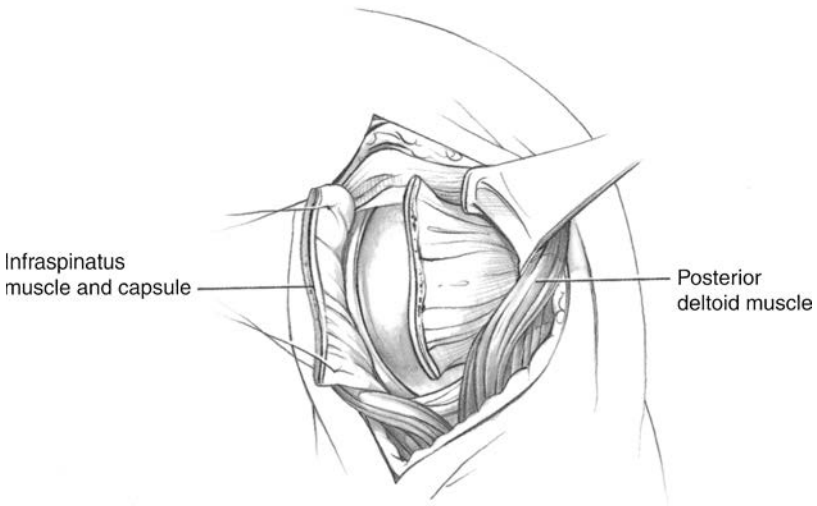

Figure 13-24 Posterior infraspinatus capsular tenodesis: The arm is positioned in neutral rotation and the infraspinatus muscle and underlying posterior capsule are incised at, and parallel to, the glenoid rim.

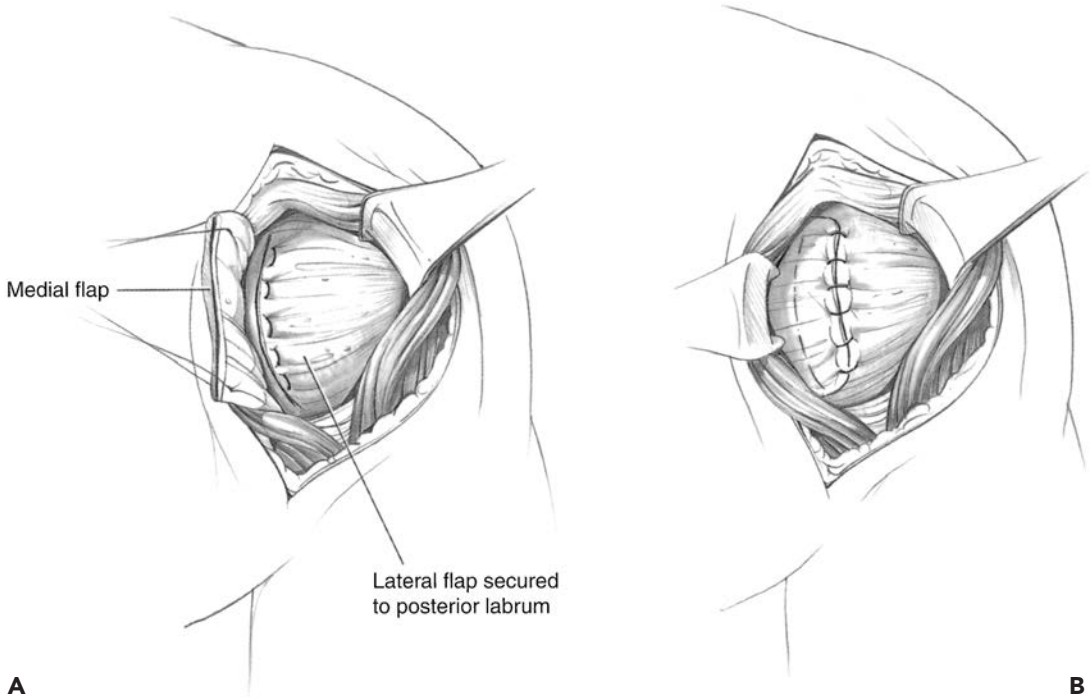

Figure 13-25 **(A)** The arm is placed in slight external rotation and the lateral flap of infraspinatus, and the capsule is secured to the posterior labrum. **(B)** The medial flap of infraspinatus and capsule overlaps the lateral flap.

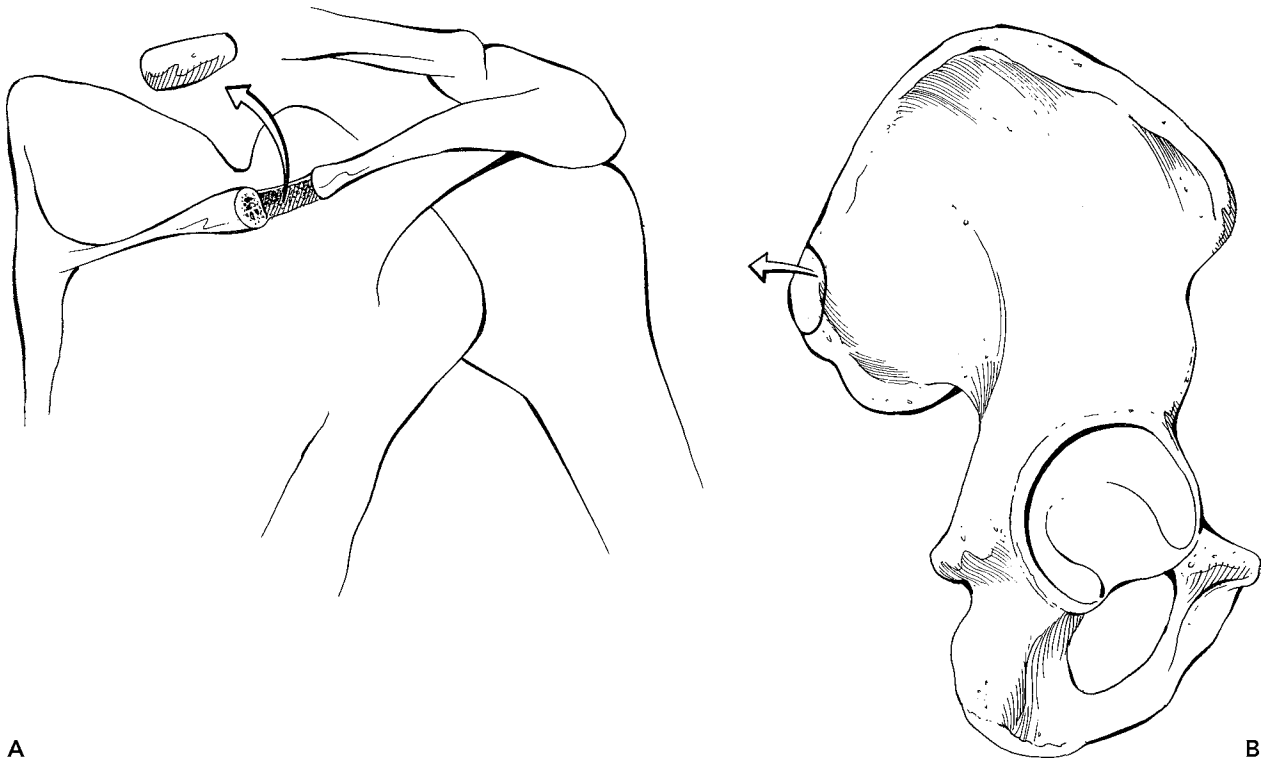

Figure 13-26 Bone graft is harvested from the **(A)** scapular spine or **(B)** posterior iliac crest.

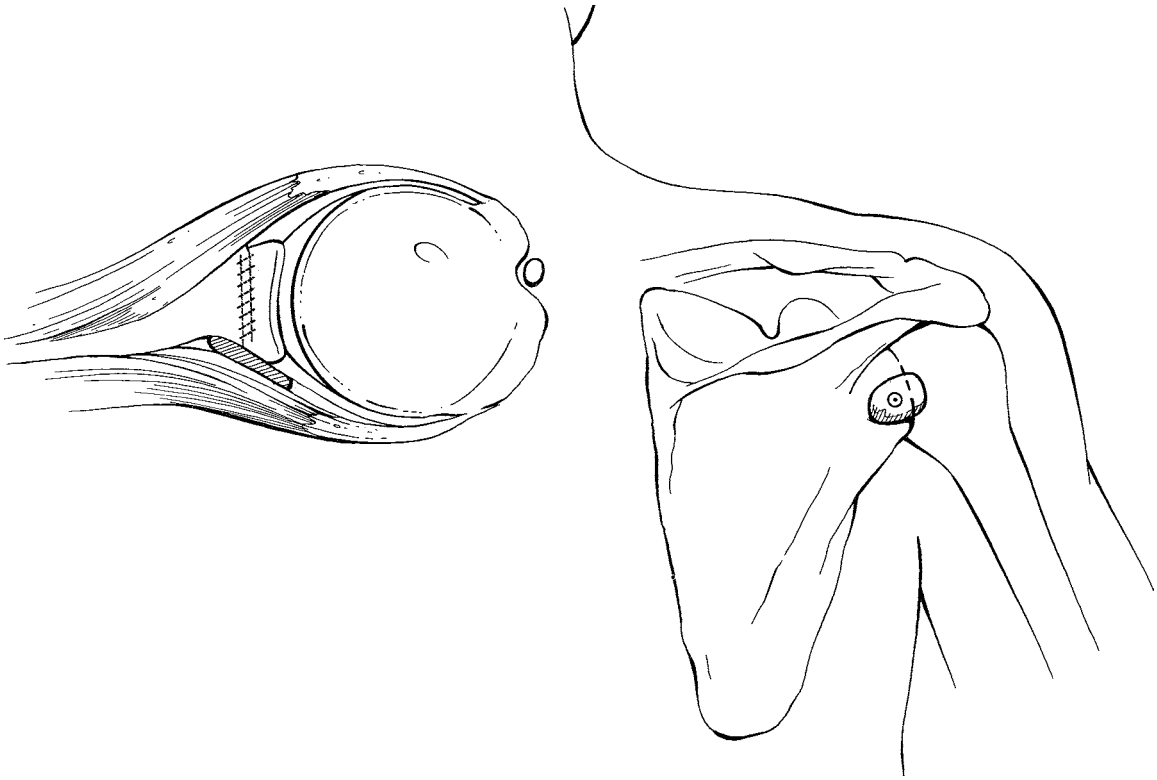

Figure 13-27 Posterior bone block: Bone graft is placed just inferior to the equator of the glenoid. Note the graft does not extend posterior to the posterior continuation of the glenoid rim.

to the inferior glenoid is left intact. The cancellous side of the bone graft is placed against the posterior glenoid neck so that it extents to, but not beyond, the posterior glenoid rim (Fig. 13-27). The bone graft is secured with one or two partially threaded cancellous screws. A burr may be used to contour and trim the bone graft to the level of the posterior glenoid rim after it is secured. The capsule, infraspinatus, and wound are closed as described earlier. If a capsular plication or shift is performed at the time of the bone block procedure and a reverse Bankart lesion is not present, then a humeral-sided vertical capsulotomy is performed and a capsular shift is carried out as previously described.

Posterior Opening Wedge Glenoid Osteotomy

Patient positioning and exposure are as described previously. The capsule is incised in a superior-to-inferior direction at the midlevel of the capsule, and the joint is inspected. A straight, flat instrument, such as an osteotome, is placed along the surface of the glenoid fossa. The osteotomy is made parallel to the face of the glenoid surface 10 mm medial to the posterior glenoid rim. The glenoid osteotomy is taken to, but not completely through, the anterior glenoid cortex. Keeping the anterior cortex and periosteum intact is important in keeping the osteotomy stable. The osteotomy is then opened and a cortical bone graft harvested from the spine of the scapula is used to maintain the osteotomy in the open position (Fig. 13-28). Internal fixation is generally not required, but a small staple may be used if the osteotomy is unstable. If capsular redundancy is present, a horizontal capsular incision in the medial-to-lateral direction is performed and a medial-based capsular shift is performed. Infraspinatus and wound closure are performed in standard fashion.

Arthroscopic Surgical Techniques

Examination Under Anesthesia

Examination under anesthesia was discussed previously in this chapter. The goal of examination under anesthesia is the same whether the surgery is to be performed open or arthroscopically.

Patient Positioning and Portal Placement

Arthroscopic shoulder stabilization can be done either in the lateral decubitus or the beach-chair positions. For those choosing the lateral decubitus position, the patient is approximately 20 degrees rolled back from perpendicular to the operating room table. An axillary roll is placed

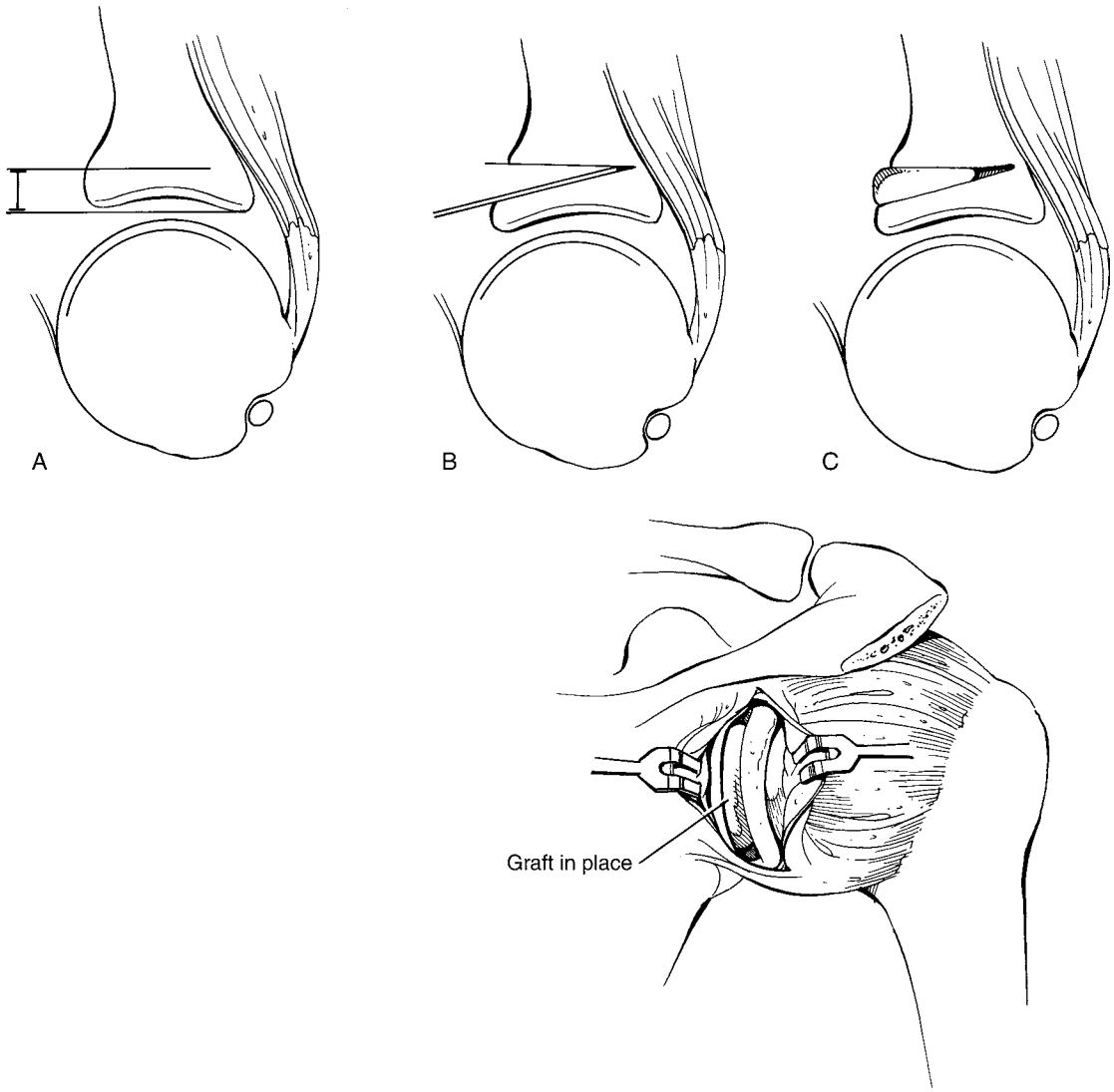

Figure 13-28 Posterior opening wedge osteotomy. **(A)** An osteotomy is performed 10 mm medial to the glenoid margin, parallel to the face of the articular surface. **(B)** The osteotomy extends to, but not through, the anterior cortex of the glenoid. **(C)** Bone graft is placed in the opened osteotomy to maintain the relocated position of the articular surface.

under the dependent axilla, and a supportive bean bag is used along with tape to support the torso and chest wall. The patient's arm is abducted approximately 20 degrees with 7 to 12 lb of longitudinal traction applied. The table should be rotated so that the anesthesiologist is in front of the patient, allowing for the surgeons to have access to the front, top, and posterior quadrants of the affected shoulder. If the beach-chair position is selected, the affected shoulder needs to be free from the edge of the table, allowing for posterior, anterior, and superior exposure to the affected shoulder. This may require additional support to the head and neck of the anesthetized patient.

The majority of shoulders undergoing arthroscopic stabilization will require a standard posterior viewing portal, anterosuperior portal, and occasionally an additional posteroinferior portal. The initial portal is developed 2 cm inferior to the spine of the scapula at the junction with the lateral margin of the acromion. This is slightly lateral to the usual posterior viewing portal. The joint is often inflated with fluid through a spinal needle, followed by introducing a blunt trochar and cannula, which allows the scope to be placed in the posterior viewing portal. An anterior portal is then developed from outside in, using a spinal needle placed anterior to the acromion and entering the joint midway between the biceps and subscapularis superior border. Transferring the scope to the anterior portal, the original posterior portal can be inspected from inside of the joint to see whether it is appropriate for anchor placement. Often these portals are parallel to the glenoid and do not always allow for favorable angulation

to the glenoid to allow for anchor penetration. A third portal inferior and lateral can be demonstrated by an outside-to-inside technique using a spinal needle (Fig. 13-29). The spinal needle is directed at the posteroinferior glenoid margin and can be further developed should anchor placement be indicated. This portal does not require an additional cannula and can be used as a percutaneous portal for suture anchor placement.

Diagnostic Arthroscopy

Pathologic findings associated with posterior and posteroinferior shoulder instability can be appreciated arthroscopically.[2,86] The most common finding is an enlarged posteroinferior capsule, into which the humeral head is allowed to sublux (Fig. 13-30). Posterior labral changes found in shoulders with recurrent subluxation vary from intact labrum to tears.[4,43] Labral pathology is different than what is normally associated with recurrent anterior instability. The author's review of arthroscopic posterior labral pathology identified 22% posterior labral detachments (reverse Bankart lesions), 18% capsule tears at the junction of labrum, 43% labral tears with intact capsules, and 17% absent glenoid labral or capsule tears (Fig. 13-31). Additional capsular problems include midsubstance capsule tears, as well as lateral capsule avulsions from the humeral head (Fig. 13-32).[1] Articular findings associated with recurrent glenohumeral instability may include glenoid chondral injuries, loose bodies, and articular-side rotator cuff tears (Fig. 13-33).

There may be additional labral pathology opposite the site of posterior glenohumeral subluxation.[2] Patients may have associated type II superior labral avulsions, anterior labral detachment, and enlarged rotator intervals (Fig. 13-34). The rotator interval viewed arthroscopically includes the space between the superior and middle anterior glenohumeral ligaments. Normally this space allows for visualization of the superior aspect of the subscapularis tendon. As this interval enlarges, a greater amount of the subscapularis tendon is easily seen from the posterior viewing portal (Fig. 13-35). Dilation of the rotator interval is determined by clinical testing as well as arthroscopic appearance. Excessive sulcus signs in neutral and external rotation, combined with the arthroscopic appearance of widening of this interval and exposure of the subscapularis tendon, would suggest that this is a pathologic finding associated with recurrent posterior subluxation. This compromise of the anterosuperior supporting structures is felt to be an important stabilizer in the adducted shoulder.[31]

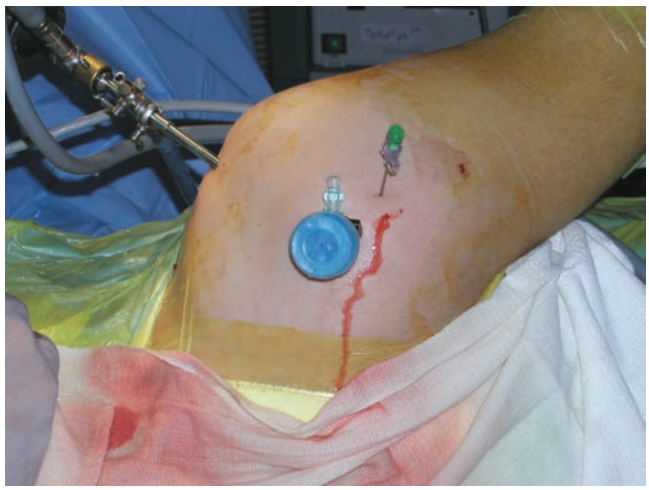

Figure 13-29 Arthroscopic portals for posterior repair in the right shoulder. Scope is anterior; working portal is posterior. An optional portal is lateral and inferior for anchor placement.

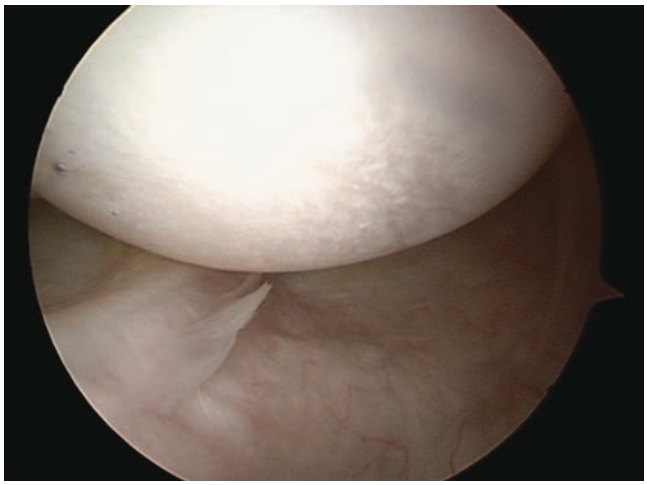

Figure 13-30 Enlarged posterior pouch: Right shoulder visualized from posterior portal demonstrates enlarged capsular pouch and intact labrum.

Suture Anchor Repair

A suture anchor repair is indicated in cases where the labrum is detached from the glenoid, the labrum is insufficient to anchor capsular sutures, and the glenoid articular surface has been disrupted along the margin of the labrum attachment. In these situations, the glenoid can be roughened with a burr or rasp and the labrum mobilized onto the articular edge with or without a posteroinferior capsular shift.

After visualization from the posterior portal, a scope is placed in the anterior portal. The posterior portal is replaced with a large cannula that allows for introduction of suture hooks and instrumentation. Through this portal, the glenoid and labrum can be prepared by shaving the junction and removing devitalized tissue. Additional abrasion can be applied to the capsular ligaments to create a tissue response at the site of repair.

Through the optional accessory posteroinferior portal, a drill hole is made on the inferior glenoid articular margin, and a suture anchor is placed percutaneously (Fig. 13-36).

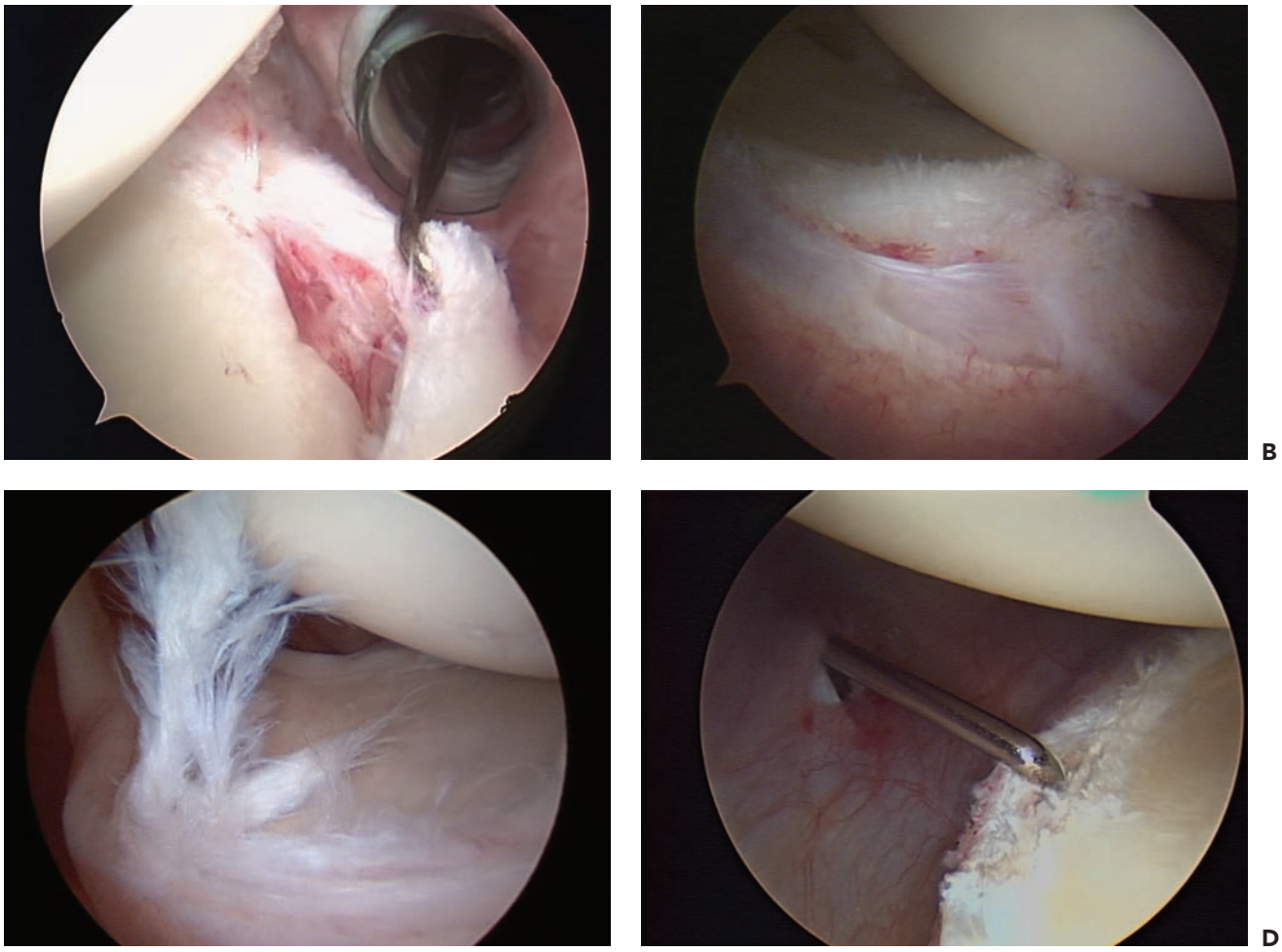

Figure 13-31 Classification of posterior capsule labrum tears. **(A)** Posterior labral detachment from glenoid will require a suture anchor repair. **(B)** Posterior capsule detached from labrum. Capsule plication to labrum can reduce posterior translation. **(C)** Posterior labral tear with intact capsule. Suture anchor may be necessary if inadequate labrum. **(D)** Posterior labral scuffing. Capsule plication to intact labrum.

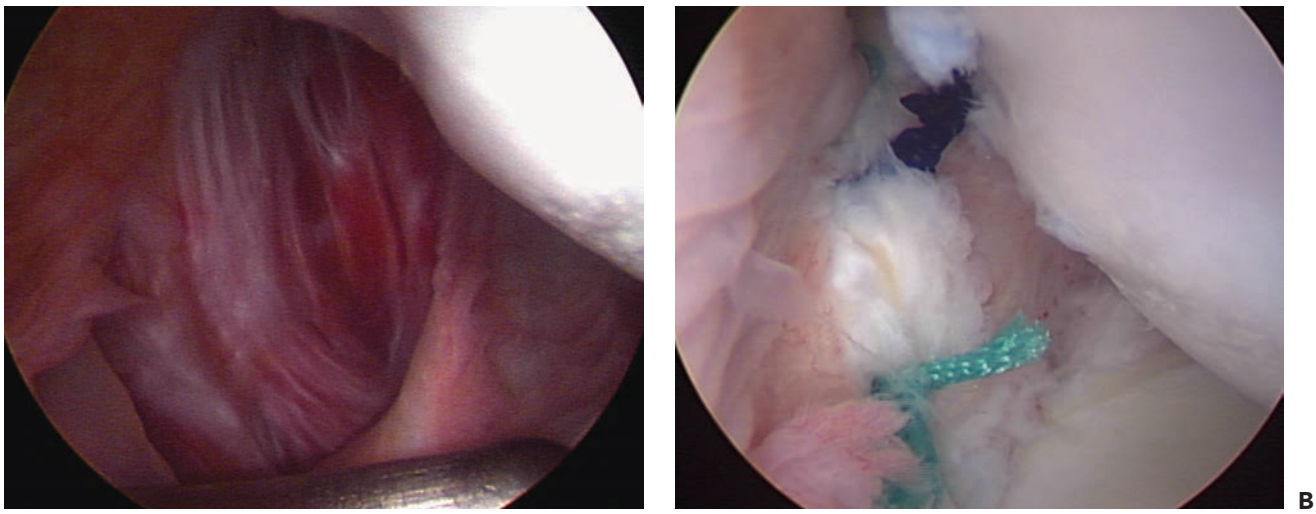

Figure 13-32 **(A)** Posterior capsule tear and humeral avulsion. **(B)** Anchor repair to humeral head and capsule tear repair.

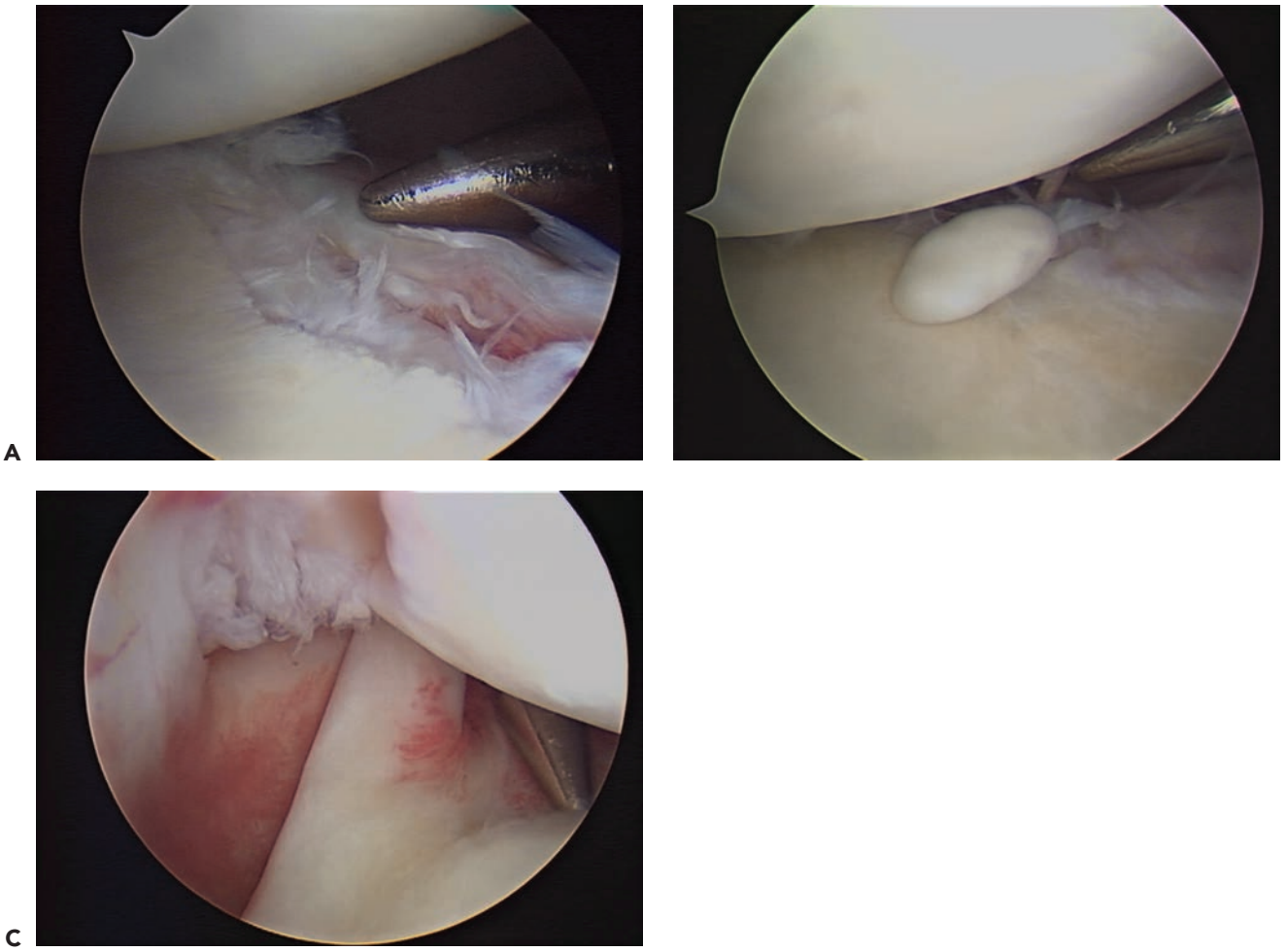

Figure 13-33 Associated articular findings: **(A)** Glenoid articular defect: Suture anchors may allow labral repositioning over the defect. **(B)** Loose body. **(C)** Articular partial-thickness rotator cuff tear.

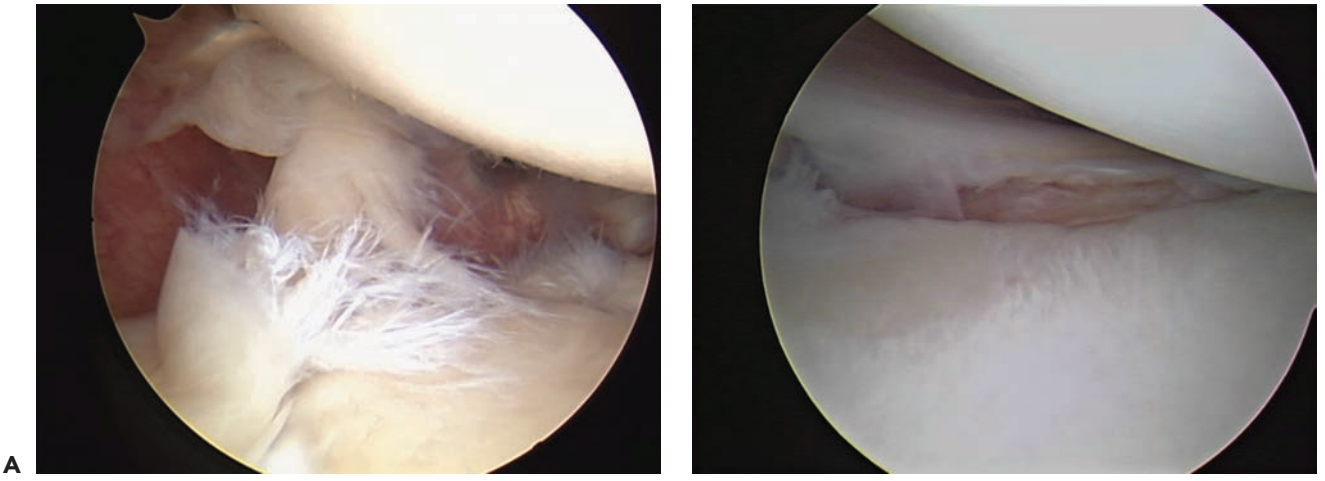

Figure 13-34 Labral tears in addition to posterior pathology in posterior subluxators. **(A)** Superior labrum from anterior to posterior (SLAP) tear. **(B)** Anterior labral tear without capsule changes.

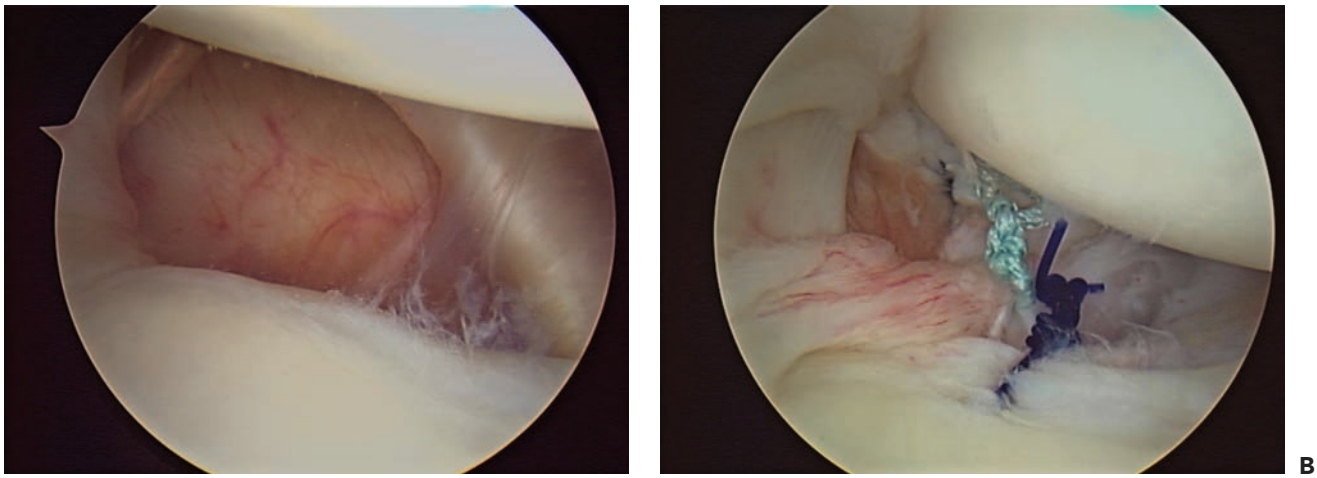

Figure 13-35 Rotator interval enlargement. **(A)** Stretched interval between superior and middle glenohumeral ligaments exposes a greater amount of subscapularis. **(B)** Reduction of the interval is performed by advancing the superior border of the middle glenohumeral ligament to the superior ligament.

A Spectrum suture hook (Linvatec, Largo, FL) is introduced through the posterior portal, grasping the labrum at the inferiormost aspect of the labral tear adjacent to the suture anchor. A suture shuttle (Linvatec, Largo, FL) is introduced and retrieves one arm of the braided suture through the labrum. As the suture is tied, the labrum is reapproximated to the posterior glenoid rim (Fig. 13-37). If there is concomitant capsular redundancy that needs to be addressed as the labrum is repaired, a suture hook is used to grasp the posteroinferior capsule, reposition it more superiorly, and pass the hook a second time underneath the labrum. This creates a capsular pleat that is incorporated into the labral repair, resulting in a superior shift of the posteroinferior capsular tissue (Fig. 13-38). Additional suture anchors are used as needed throughout the length of labral deficiency. Additional capsule plication sutures can be used to further reduce the posterior capsular pouch.

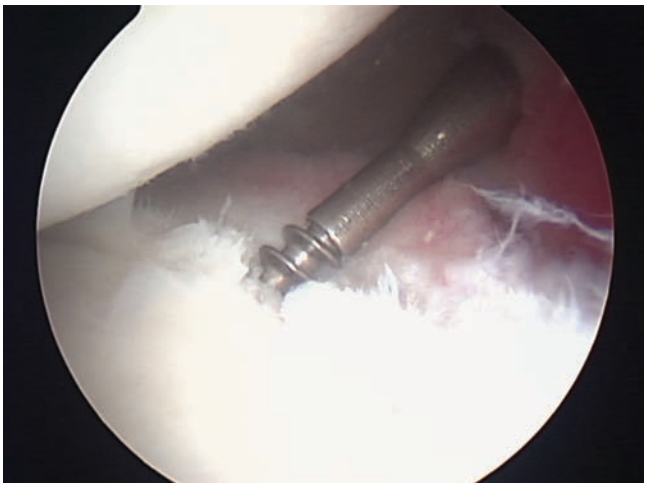

Figure 13-36 Suture anchor placement. An anchor is placed percutaneously using needle guidance into the posterior inferior quadrant.

Capsular Plication

In cases where the labrum is attached to the glenoid margin, plication sutures can be used to reduce the posteroinferior capsular pouch. A shaver is placed through the posterior portal, and the capsule is abraded with the suction off in the areas of anticipated repair. A curved suture hook is introduced into the inferior pouch and a full-thickness capsular bite is taken. A second pass through the intact inferior labrum completes the capsular plication (Fig. 13-39). Either a monofilament suture or suture shuttle can be introduced for later substitution with a braided suture. The definitive suture is tied beginning inferiorly. The capsule plication continues, grasping the inferior pouch with full thickness and again through the labrum, moving superiorly along the posterior glenoid. The sutures are tied sequentially below the cannula. The pouch is reduced and the capsule is secured in a superior direction. The reverse hooks can be used to further tension the capsule above the cannula. Once all the sutures are secured, the plastic cannula is removed, and the suture hook can be percutaneously passed through the skin portal, grasping the superior edge of the capsule and closing down the portal hole with an absorbable monofilament suture.

Anterior and Superior Repair Including Rotator Interval

There are a number of patients with type II superior labrum from anterior to posterior (SLAP) lesions as well as anterior labral avulsions, associated with recurrent posterior shoulder subluxation. In cases of significant labral pathology, additional suture anchor fixation is suggested. This may include a superior labral anchor repair or selective anterior labral suture anchor repair. It is important not to ignore these potential additional lesions, to minimize

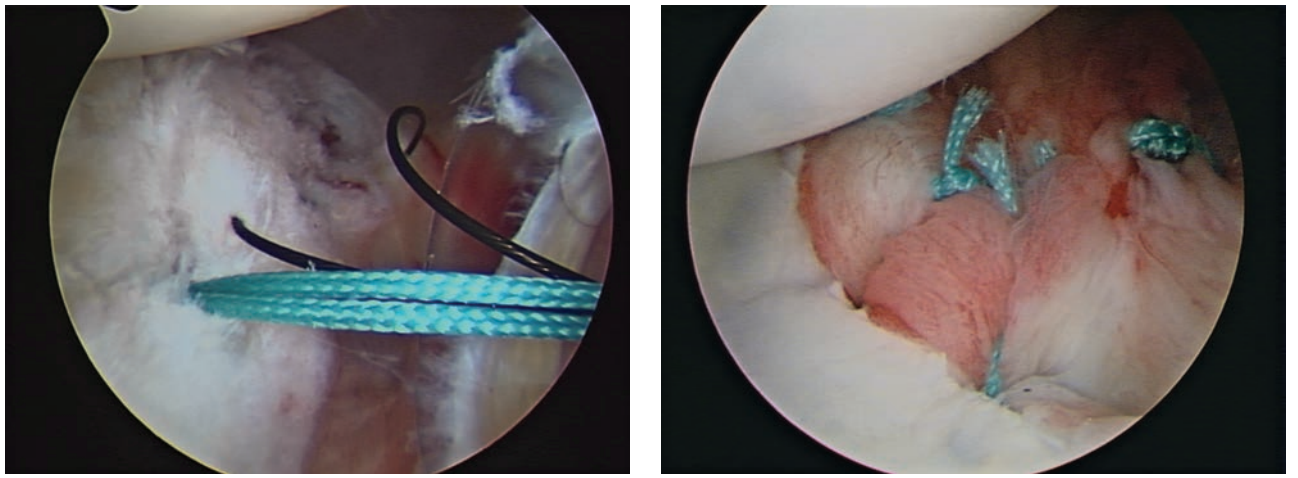

Figure 13-37 Suture anchor repair. **(A)** Anchor has been placed along glenoid rim. Shuttle is passed through inferior capsule and under labrum to retrieve braided sutures. **(B)** Completed capsule-to-glenoid repair with suture anchors.

risk of recurrence as well as avoid the potential for coracoid impingement syndrome.[2] With the posterior structures stabilized, the shoulder may be predisposed to increased anterior translation, reducing the space between the rotator cuff and the coracoid process. Anterior suture anchor repairs are designed to reattach the labrum and not create significant capsular shifts. A suture anchor is placed on the articular margin and sutures attach the labrum without significant capsular advancement.

The rotator interval has gained recognition as being important in shoulders that have increased posterior and posteroinferior translation. In selective shoulders, this interval may be closed by reapproximating the middle and superior glenohumeral ligaments (see Fig. 13-35). With the arthroscope in the posterior portal, curved suture hooks are used to grasp the superior edge of the middle glenohumeral ligament and introduce a suture that could be retrieved through the superior glenohumeral ligament. This may be further assisted by a second suture with the reverse curved suture hook, grasping the full thickness of the superior glenohumeral ligament and reapproximating to the advanced middle capsular ligament. No attempt to incorporate tendinous structures is made. By grasping the full thickness of the superior glenohumeral ligament, portions of the coracohumeral ligament are incorporated in this repair. The repair begins adjacent to the glenoid, and subsequent sutures are placed from medial to lateral. The most lateral sutures may be difficult to see and are tied in the subacromial space. Blind knot-cutters may be helpful in dividing the sutures after the interval has been securely closed.

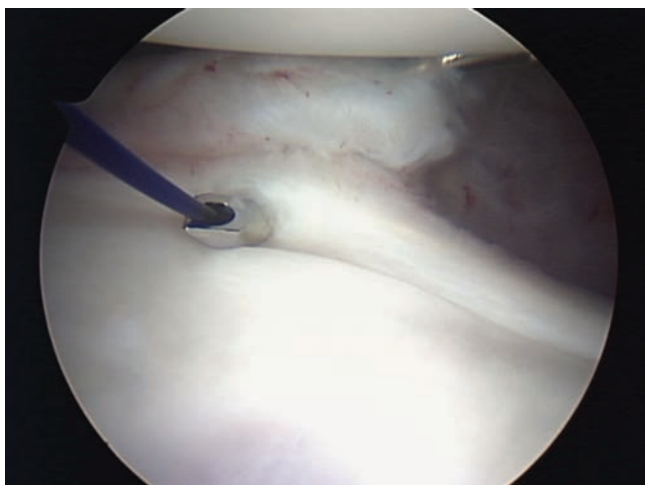

Figure 13-38 Inferior quadrant stabilizing stitch is made by using a suture hook, grasping capsular pouch, and plicating to inferior labrum.

POSTOPERATIVE CONSIDERATIONS

Postoperative management after open or arthroscopic posterior shoulder surgery requires the use of a thoracobrachial orthosis or external rotation brace. Prior to surgery, patients are fitted for the orthosis or brace with the arm positioned in slight abduction in or slightly posterior to the coronal plane of the body and in neutral to 10 degrees of external rotation (Fig. 13-40). The prefitted orthosis is applied in the operating room at the conclusion of the surgical procedure.

Patients are begun immediately on active assisted external rotation beyond the brace with the elbow at the side, elbow flexion and extension exercises, and shoulder shrugs. No inferior traction or lifting weights is allowed. Immobilization is maintained for a 4- to 6-week period. The time period is determined by the degree of passive motion measured 4 weeks postoperatively. If the shoulder demonstrates inability to internally rotate past the neutral position, the brace is discontinued at 4 weeks. The arm is

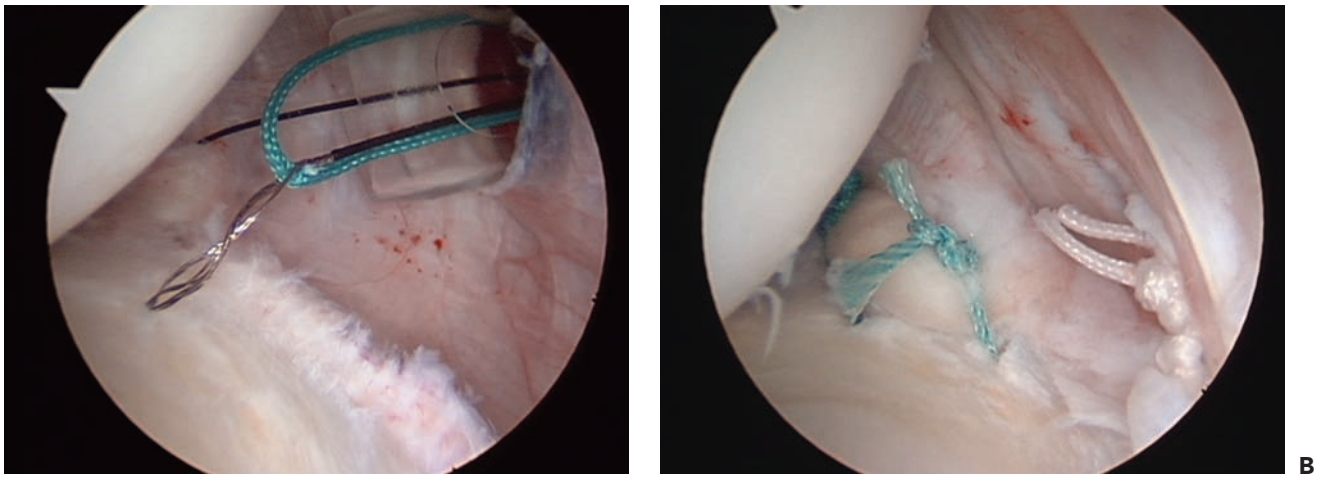

Figure 13-39 Capsular plication. **(A)** A suture shuttle is introduced with a suture hook and a braided suture is retrieved. **(B)** Capsular plication repair: Posterior capsular pleats created, eliminating the posterior pouch.

then kept in a sling for an additional 2 weeks, during which time the patient starts a gentle exercise program. Patients with generalized ligamentous laxity are generally treated with a brace for 6 weeks. During the time that the brace is used, the patient is allowed to remove the brace to dress and bathe, as long as the arm is not allowed to internally rotate beyond the neutral position.

After the brace is removed the patient starts active assisted supine forward flexion, external rotation, and internal rotation. With the scapula well supported by the table, the neuromuscular coordination of forward elevation without winging is more easily achieved. Once the arm approaches near full forward flexion, the patient is allowed to sit and stand and continue to perform active assisted

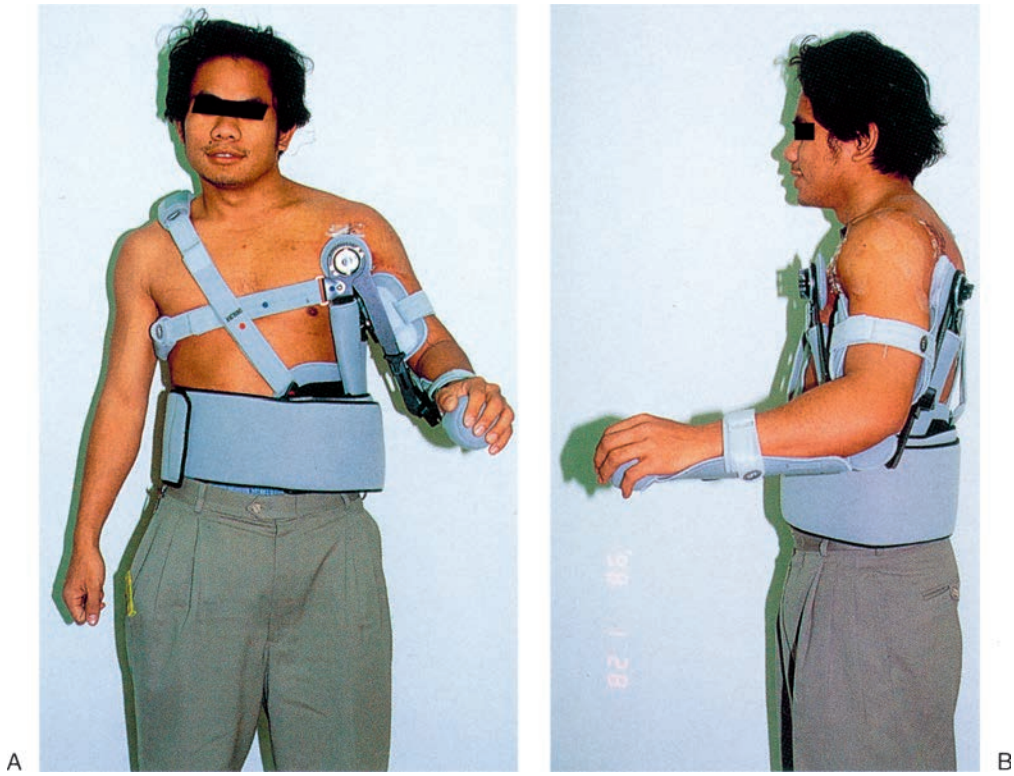

Figure 13-40 **(A)** Frontal and **(B)** side view of postoperative brace positioning after posterior capsulorrhaphy. The arm is placed in slight abduction and neutral to slight external rotation. The arm should be in the plane of the body or slightly posterior to this plane.

forward elevation. Visualizing the scapula from behind, one can determine when it is safe to begin independent forward arm elevation without the assistance of the other extremity. At 8 weeks postoperatively, the patient is begun on gentle resistive exercises. This will include external rotation resistive exercises and scapular stabilizing exercises. The scapular stabilizing exercises include strengthening of the latissimus, rhomboids, and trapezius. This can be achieved with bent-over rows, seated rows, and latissimus pull-downs to the chest in front. Wall pushups are not attempted until 3 months, and then a widened-grip pushup and/or bench press with light weights and high repetition is begun.

An anticipated return to athletics includes noncontact sports at 4 months and contact sports at 6 months. Sports that run the risk of a forward fall on an outstretched arm are considered contact sports. Patients may consider returning to work depending on the nature of their job. Sedentary work can often be achieved early on, depending on the ability for transportation to and from the job. More strenuous activities may require 8 to 16 weeks, depending on the nature of the job and the level of recovery that has been achieved. Total rehabilitation time is generally 16 to 24 weeks for primary capsulorrhaphy surgery. Revision surgery and complex situations, including scapulothoracic reconstruction, may require longer periods for rehabilitation.

RESULTS

The lack of a universally accepted and applied classification system for posterior instability makes interpretation of treatment results presented in the literature more difficult.

Success in treating recurrent posterior subluxation of the shoulder using a nonoperative exercise program is dependent on the amount of disability the patient is experiencing at the time of presentation, as well as on how one judges treatment success or failure. Fronek et al. reported a 63% success rate using nonoperative measures in patients with moderate disability when performing strenuous activities, but who had no interference with activities of daily living on initial presentation.[25] Hurley et al. demonstrated similar improvement in 68% of the patients treated under a similar treatment protocol.[38] Success in both of these clinical investigations was defined as clinical improvement that satisfied the patient to the extent that no further treatment other than maintenance muscular conditioning was required. These patients often demonstrate persistent posterior instability. However, the involuntary subluxations that in many cases prompted them to seek treatment are significantly improved. These studies combined with the mixed results of surgical intervention warrant the inclusion of nonoperative measures as an initial form of treatment in any algorithm.

At first glance, the results of the surgical treatment of recurrent posterior subluxation are discouraging. This has led some authors to conclude that recurrent posterior subluxation should not be treated surgically.[34] However, a more detailed examination of the literature shows cause for guarded optimism. Historically, confusion over the classification of posterior instability, a poor understanding of the underlying pathophysiology, and the routine use of surgical procedures that failed to address the pathology of the underlying instability resulted in high surgical failure rates.

The surgical management of recurrent posterior subluxation can be divided into soft tissue and osseous procedures. Because the cause of posterior instability usually resides in the posterior soft tissue structures, repairs aimed at reinforcing these deficient structures are more commonly employed. More recently, identification and correction of the specific pathologic lesion has gained favor over so-called nonanatomic repairs.

The most common finding in patients with recurrent posterior subluxation is a patulous posterior capsule. Although treatment of this pathologic lesion depends on the direction of the instability, all anatomic repairs stress a rebalancing of this patulous capsule to restore glenohumeral stability. Fronek and colleagues reported on 24 patients with isolated posterior subluxation treated with posterior capsulorrhaphy, with a 91% success rate.[25] The capsular repair is reinforced by the infraspinatus tendon and a posterior bone block if the posterior soft tissues are deficient. Hawkins et al. also favor utilization of the infraspinatus tendon to reinforce the capsular repair posteriorly and reported an 85% success rate.[32,35] In 1980, Neer and Foster introduced the inferior capsular shift in patients for whom there are inferior and posterior components to the instability.[52] Bigliani et al. reported the early results with this procedure in 25 patients with recurrent posterior subluxation, with 88% satisfactory results.[5] Pollock and Bigliani reported longer-term follow-up of this procedure, with an overall satisfactory rate of 80%.[61] Interestingly, several of these failures occurred in patients with revision surgery. Excluding revision cases, the success rate improved to 96%, highlighting the importance of meticulous soft tissue repair at the first surgery.

Although labral detachment from the posterior glenoid rim is rare in recurrent posterior subluxation, several authors have successfully treated recurrent posterior subluxation with an anatomic labral repair when a reverse Bankart lesion is present. Rowe and Yee performed reverse Bankart repairs on two patients with recurrent posterior subluxation with no recurrence of instability.[66]

Arthroscopic repairs for posterior instability typically group isolated labral repairs, capsular plication, and combined labral repairs and capsular plication together.[4,43] This makes it more difficult to determine the effectiveness of repairs for each specific pathology. However, these combined reports indicate satisfactory results when all identified contributing pathologies are addressed.

Several nonanatomic procedures have been previously described, with mixed results. Among these is the reverse

Putti-Platt operation. Dugas et al. treated 18 patients with recurrent posterior subluxation with a reverse Putti-Platt procedure.[18] Satisfactory results were obtained in 16 of 17 patients available for follow-up. Six patients experienced mild loss of motion. Similarly, Hawkins et al. reported good results in their patients undergoing this procedure, but in a separate group of patients, who were originally operated on by other surgeons, they reported a recurrence rate of 83% using this repair. This suggests that success with this procedure may depend on the surgeon's experience.[34] Boyd and Sisk reported nine nonanatomic soft tissue repairs that were augmented by posterior transfer of the long head of the biceps tendon.[8] All patients reportedly did well without recurrence of instability. The second group of procedures for recurrent posterior subluxation addressed bony pathology by either redirecting abnormal osseous anatomy (glenoid osteotomy or proximal humerus rotational osteotomy) or by augmenting deficient glenoid bone stock or incompetent posterior soft tissues (bone block procedures).

Glenoid osteotomy (glenoplasty), first reported by Scott, is a posterior opening wedge osteotomy of the glenoid neck with interposition of bone graft, thereby redirecting the glenoid more anteriorly.[70] In his original report of three cases, one patient dislocated anteriorly in the early postoperative period and another had recurrent posterior subluxation. Norwood and Terry reported 19 patients with recurrent posterior subluxation, from various causes, treated by glenoid osteotomy.[57] Three patients (16%) continued to experience isolated posterior instability, four patients (21%) developed isolated anterior instability, and two patients (12%) had multidirectional instability in the postoperative period.

English and McNab advocated an anatomic approach to recurrent posterior subluxation.[21] The surgical results of eight patients were reviewed. All demonstrated increased glenoid retroversion on preoperative radiographs. Four of eight patients were treated with posterior glenoid osteotomy without recurrence. Interestingly, they noted that in patients with ligamentous laxity, there was a tendency for the humerus to subluxate anteriorly. More recently, glenoid osteotomy has been successfully employed in patients with localized posterior glenoid hypoplasia and recurrent posterior subluxation.[85]

The role of posterior glenoid opening wedge osteotomy in the treatment of recurrent posterior subluxation is open to many criticisms. Gerber et al. have documented coracoid impingement following posterior glenoid osteotomy.[28] There has been a spectrum of anterior instabilities reported following glenoid osteotomy that range from coracoid impingement to anterior dislocation.[57,70] Posterior glenoid osteotomy is a technically demanding procedure, with the potential for significant complications. Hawkins et al. reported a 41% complication rate with this procedure, including subsequent glenohumeral arthritis in two patients, one caused by intraarticular extension of the osteotomy.[34] Finally, earlier studies that justify glenoid osteotomy based on radiographic evidence of increased glenoid retroversion may have overstated this problem.[20] In the past, the ability to document increased glenoid retroversion by plain radiographs has been questioned. Galinat et al. have recently reported a reproducible means of determining glenoid version from plain radiographs.[26] However, this method was not used in previous studies. Currently, CT scan is the most accurate method of determining glenoid version.

Increased proximal humeral retrotorsion has been implicated as a cause for recurrent posterior subluxation. Rotational osteotomy has been used to treat recurrent posterior subluxation, based on the assumption that increased humeral retrotorsion contributes to posterior instability. However, the relation between humeral retrotorsion and posterior instability has not been established. By limiting internal rotation through proximal humeral rotational osteotomy, it was theorized that posterior instability would subside. Surin et al. reported 12 cases of recurrent posterior instability treated with external rotation osteotomy of the proximal humerus.[75] One patient had pain after osteotomy that was attributed to anterior impingement. A second patient developed recurrent instability. Most patients had significant restriction of external rotation postoperatively. Chaudhuri et al. reported a series of patients who had rotational osteotomy for glenohumeral instability.[14] Only 1 of 16 cases underwent osteotomy for recurrent posterior subluxation. This patient developed postoperative anterior instability requiring muscle transfer.

Another group of bony procedures act to buttress the posterior glenoid with bone graft from the iliac crest or spine of the scapula. Several authors have reported the use of posterior bone block procedures for recurrent posterior instability.[3,41] Ahlgren and colleagues treated five patients with a posterior bone block procedure.[3] Two of the five patients had normal shoulders postoperatively, whereas three demonstrated varying degrees of recurrent posterior instability. More commonly, posterior bone block procedures are combined with a posterior capsulorrhaphy or performed for failed posterior soft tissue procedures.[41,51,79]

CONCLUSIONS

Recurrent posterior subluxation is less common than anterior subluxation, but it is being diagnosed more frequently than in the past. The treatment of recurrent posterior subluxation is dependent on the underlying pathology. An anatomically based classification system facilitates the diagnosis and appropriate treatment plan. The earlier literature on surgical treatment has yielded inconsistent results. More recently, improved recognition of the underlying pathology coupled with more anatomic surgical approaches have resulted in consistently improved results. An algorithm for

the evaluation and treatment of posterior instability based upon an anatomic classification is presented.

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14 Multidirectional Instability: Open and Arthroscopic Management

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INTRODUCTION

Although Neer's original description of multidirectional instability was published more than 20 years ago, our collective understanding of the cause and optimal treatment protocols remain incomplete. Reviewing more historical publications, we learn that although the term "multidirectional" had yet to be applied, several authors recognized that there were patients who had instability patterns that did not fit into the known classification schemes of the time.[8,22,23,80] Multidirectional shoulder instability is a complex entity, and even 20 years after its first discussion in contemporary literature, relatively few series of patients with this condition have been reported. An accurate and encompassing definition of this clinical entity also remains elusive. The pathognomonic anatomic finding is redundancy of the inferior capsule allowing the shoulder to sublux, not only anteriorly but inferiorly and posteriorly as well.[21,63,65,71] Patients with multidirectional instability (MDI) possess two clinical features. First, most symptoms are experienced in the midrange positions of the glenohumeral motion, such as those that occur

during activities of daily living. These symptoms may be incapacitating enough that patients tend to avoid the extremes of glenohumeral motion. Second, the physical examination demonstrates the ability to dislocate or sublux-ate the glenohumeral joint in three directions (anteriorly, posteriorly, and inferiorly) with concurrent reproduction of symptoms in one of these directions. Importantly, both features are thought to be necessary for a diagnosis of multidirectional instability and are useful in distinguishing this instability pattern from other types of instability.

More recent investigations and analysis of this unique type of instability pattern suggest that pathology of the rotator interval may be implicated in the cause of this clinical syndrome.[24,29] Despite the increased awareness of MDI and the increased attention in research efforts to better understand its cause, understanding of this disorder remains woefully incomplete. Patient presentations are quite variable, the cause multifactorial, and treatment algorithms incomplete. In addition, MDI still lacks a uniform definition accepted by the body of orthopaedic surgeons.[61,78] Because of these ambiguities, the diagnosis of multidirectional instability is somewhat subjective, with the current tendency being to overdiagnose this once overlooked complex condition. Treatment of multidirectional instability employs nonoperative, open

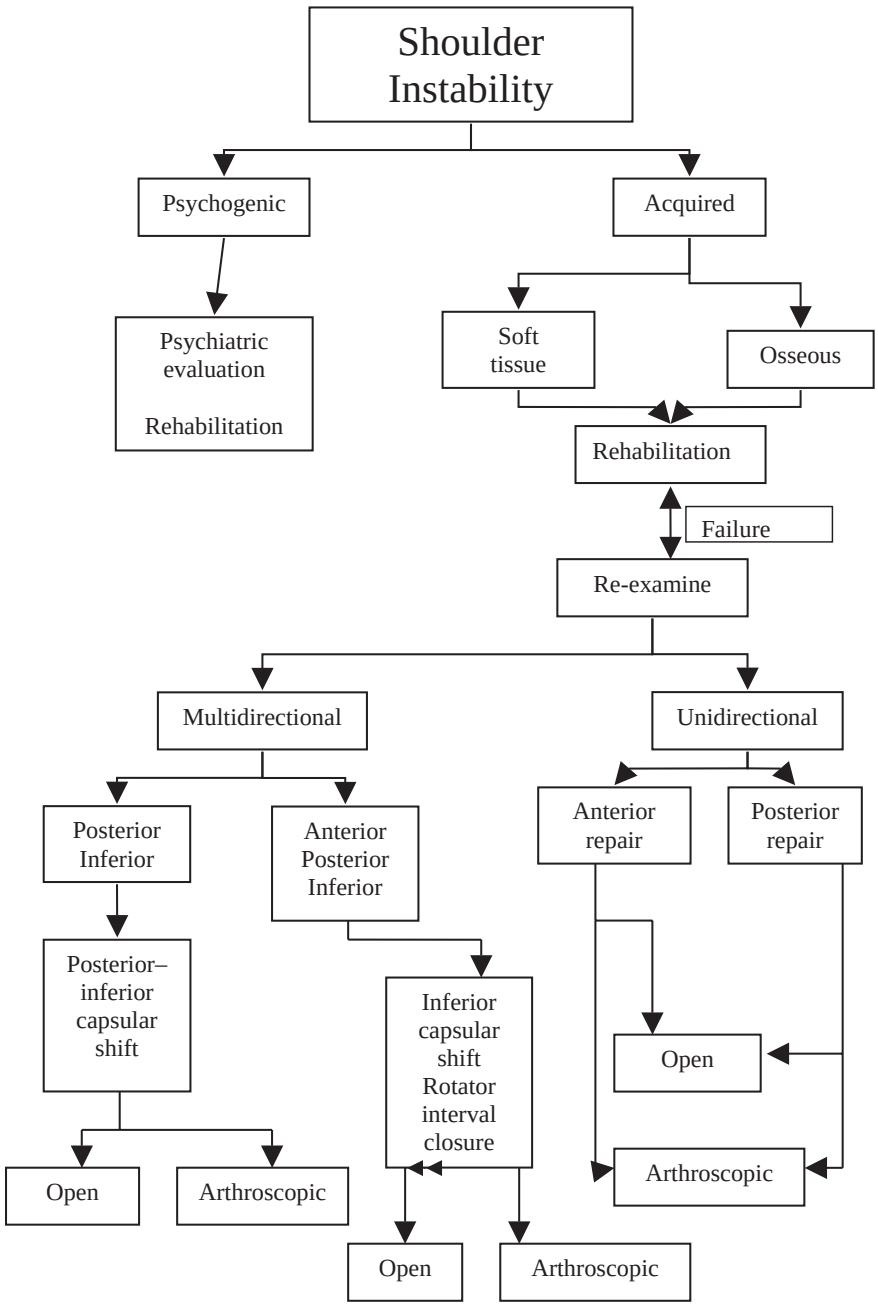


Figure 14-1 Treatment algorithm for multidirectional instability.

surgical, and arthroscopic surgical techniques. Figure 14-1 depicts a treatment algorithm for such management.

DEFINITION

Multidirectional instability has been defined simply as "global instability" or "instability in more than one direction," and in more complex terms, as part of a classification system with multiple subtypes.[57] A useful and comprehensive description of multidirectional instability defines it as *a global shoulder laxity (anterior, posterior, and inferior) that is associated with the concurrent reproduction of symptoms inferiorly and in at least one other direction. Additionally most symptoms are experienced in the midrange of glenohumeral motion, resulting in frequent limitation in activities of daily living.*[44]

It is important to discern the difference between instability and laxity. Laxity is the clinical condition associated with ligaments that have the ability to stretch beyond what is considered normal. Bear in mind that there is no absolute "norm" in measuring or assessing ligament compliance to tensile force. The more important concept to understand is that laxity, by definition, is not symptomatic in itself, nor does any "lax" joint lead to clinical awareness or complaint. By contrast, "instability" is a clinical syndrome manifest by patient complaint, pain, apprehension, or fear that application of a physiologic or nonphysiologic force may result in a mechanical dissociation of the joint.

CLASSIFICATION

Classification systems have been developed to assist the physician in organizing the variety of pathologic states of a given medical condition. They serve to assist in characterizing a condition by either its anatomic, histologic, or functional state. A classification system may also be utilized to direct a potential therapeutic algorithm. To be a functional classification system, it should be simple, applicable, predictable, and most of all, reproducible among physicians. In short, it should result in high levels of intraobserver and interobserver reliability when applied to the same pathologic condition.

Classification systems, when used to define and characterize shoulder instability, have been notoriously difficult to apply. Cofield and Irving noted that as proof of the complexity of classifying shoulder instability, there are between 24 and 54 subclassifications depending on the system used.[19] In an effort to ensure completeness, several systems have been employed based on different parameters. Some systems are based on the direction of the instability, others on cause (traumatic or atraumatic), and still others on volition.

Thomas and Matsen have provided perhaps the simplest classification of instabilities based on the mnemonics AMBRII and TUBS.[83] *A*traumatic *M*ultidirectional *B*ilateral *R*ehabilitation *I*nferior capsular shift with *I*nterval closure defines the typical clinical scenario and within, describes

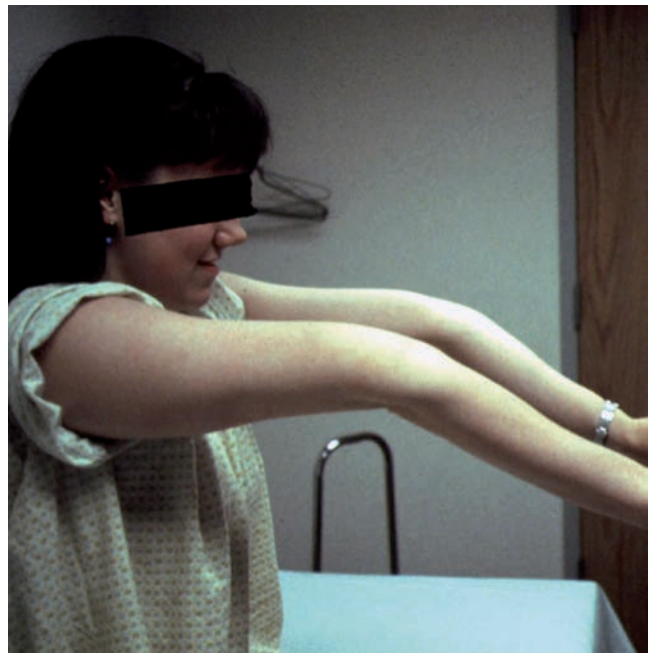


Figure 14-2 This young woman with multidirectional instability displays hyperextensibility of the elbow joints. This finding would suggest a congenital collagenopathy.

predictable treatment regimens. So, too, the *T*raumatic *U*nidirectional *B*ankart—*S*urgery defines the other common clinical scenario and treatment. But within such a simple classification, other important parameters of clinical or therapeutic significance are lost.

It may be important to classify multidirectional instability by cause. The condition may be acquired, congenital, or traumatic in origin. In patients with the congenital type, there are usually multiple manifestations of lax ligaments involving many joints. The most classic manifestations that occur in the upper extremity include hyperextensibility of the elbow, hyperflexion at the wrists, and hyperextensibility of the metacarpophalangeal joints (Fig. 14-2). Laxity at these joints is rarely, if ever, of clinical significance. Patients with multidirectional shoulder instability may also have painful instability of one or both sternoclavicular joints, which can become disabling. In the lower extremity, the patient with congenital ligamentous laxity may complain of chondromalacia patella due patellar instability. Furthermore, ankle instability and recurrent ankle sprains may occur.

Some patients are thought to have acquired their pattern of multidirectional instability through activity or trauma.[20,32,63] The trauma is usually not overt, macrotrauma where there is a single episode of high energy absorption such as occurs in a motor vehicle accident, a fall while skiing, or a contact sport such as football. Rather, the laxity develops through the cumulative effect of repetitive use involving extremes of glenohumeral motion. Acquired laxity has been known to occur in competitive athletes (specifically gymnasts, weight lifters, and butterfly and backstroke swimmers) and in manual laborers. Athletes

who participate in racquet or throwing sports may also develop acquired laxity.[81] A number of factors may lead to the conversion of a functionally stable, ligamentously lax shoulder to one with clinically significant multidirectional instability. Precipitating events tend to be relatively atraumatic. The history obtained from the patient is one of trivial or mild injury, a moderate injury (defined as sufficient energy to disrupt ligament tissue), a period of overuse or fatigue, or even disuse. Occasionally, the precipitating event cannot even be identified. Consequently, a relatively atraumatic onset of instability suggests multidirectional instability as the cause of the clinical problem. The issue is made somewhat more complex because there are situations where an episode of significant trauma can be a factor in a shoulder with excessive laxity. The literature documents a significant number of cases where athletes with lax shoulders sustain traumatic events and then develop a clinical scenario consistent with multidirectional instability.[1,12] In this cohort of patients, Bankart lesions are occasionally found.

Classification schemes may be based on the direction of the instability. Inferior capsular redundancy and rotator interval lesions are considered the hallmark lesions of multidirectional instability, making inferior instability a universal finding in patients with multidirectional instability.[44] There are combinations of patterns of instability, which include the inferior component such as anteroinferior and posteroinferior, but it is the pure inferior component of the instability that defines the multidirectionally unstable patient. In their original study, Neer and Foster separated his patients into three groups based on the direction of the instability and the degree of instability: those with anterior and inferior dislocation with posterior subluxation; those with posterior and inferior dislocation with anterior subluxation; and those with dislocation in all three directions.[63] Gerber noted that while patients may have symptomatic instability in only one direction, they may exhibit asymptomatic laxity in other directions within the same shoulder.[25,26]

Another useful method of classifying instability is based on duration of symptoms. Instability may be either acute or chronic. These terms are more often applied to the patient with unidirectional instability as it is distinctly uncommon for a physician to see a patient with an acute episode of instability on a physiologic background of MDI. Most often patients will present *without* a known episode of subluxation or dislocation and remain oblivious to the fact that instability of the joint is responsible for their clinical presentation of shoulder pain. Therefore, a patient with multidirectional instability may present with a history of chronic shoulder pain, but *not* a history of chronic shoulder instability.[63,81]

One last method of classifying shoulder instability is based on volition. Volitional instability includes both voluntary dislocators (with or without documented psychiatric history or secondary gain issues) and habitual or positional dislocators. These are critically important distinctions to make. It has been said that a scalpel to the shoulder will never satisfactorily treat an intracranial disease! Patients with psychiatric illness, both overt and subtle, must be treated in the appropriate forum. Obsessive compulsive disorders, hysteria, and frank malingering for secondary gain must be considered—and ruled out—when treating any patient with shoulder instability. Voluntary instability is present in some form in a substantial number of patients with posterior instability and, less commonly, in the patient with multidirectional instability. Patients with voluntary instability do not necessarily have a psychiatric condition despite the connotation. Indeed, the history of voluntary instability complicates the evaluation and treatment of such patients, but in the absence of psychiatric illness or secondary gain, many patients with symptomatic multidirectional instability can be helped with surgery. Habitual instability refers to a dislocation or subluxation that occurs as a result of a neuromuscular imbalance: Abnormal muscle contraction and relaxation patterns result in the simultaneous activation and suppression of two halves of a force couple causing subluxation.[44] Habitual dislocators, regardless of their mental status, are very poor surgical candidates and remain refractory to surgical treatment. Biofeedback and muscular retraining must remain the mainstay of treatment in these patients. A positional dislocator is one who is able to demonstrate his or her instability by being able to place the arm in a position of risk. Unlike the person with psychiatric issues, these patients know their risk positions and carefully avoid them. A patient with multidirectional instability who is able to demonstrate the provocative positions that cause symptoms but otherwise tries to avoid them in life may benefit from treatment.

PATHOLOGY

There is no single pathologic lesion in multidirectional instability; there is no "essential" lesion, though a patulous inferior pouch is always present. Norris has stated that the most consistent physical finding is the "sulcus sign"[67] (Fig. 14-3). However, Harryman et al. demonstrated that a healthy control group of people will demonstrate significant degrees of inferior translation on physical examination.[30] It is the combination of inferior translation, which is symptomatic, that most appropriately defines multidirectional instability. Current understanding, though perhaps incomplete, focuses on structural or anatomic abnormalities, biochemical abnormalities, and neuromuscular abnormalities.[54] Another way to categorize etiologic factors considers global shoulder laxity and precipitating events, which may be either traumatic or atraumatic.[64]

In the normal shoulder, glenohumeral stability is conferred by an intricate balance of static and dynamic

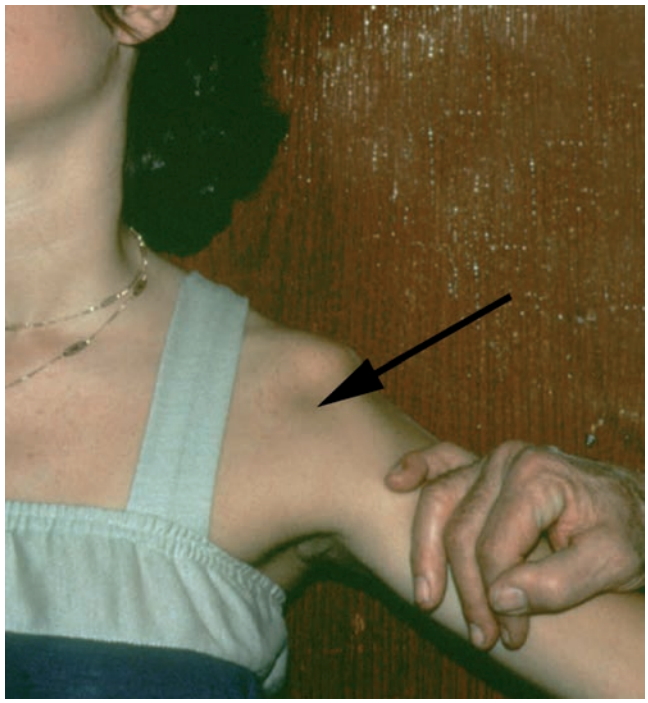


Figure 14-3 The arrow documents a "dimple" sign or a "sulcus" sign, which is considered pathognomonic of multidirectional instability. A gentle inferiorly directed force on the shoulder is used to elicit this clinical finding.

mechanisms that include muscle, nerves, ligaments, bone, and geometry.[55] The capsuloligamentous restraints in the shoulder should be considered as checkreins that provide stability at the extremes of motion. The function of these individual ligaments has been defined by several authors through basic science and cadaveric studies.[35,69] However, outside the endpoints of motion, stability of the glenohumeral joint is conferred by other mechanisms. The precise centering of the humeral head on the glenoid by the rotator cuff muscles is achieved by a mechanism defined as concavity–compression.[56,91] The presence of synovial fluid within the finite volume of the glenohumeral joint contributes to the formation of passive stabilizing articular adhesion–cohesion forces.[60] Also of importance is that an intact glenohumeral joint possesses negative intraarticular pressure.[49] These factors combine to create a stabilizing vacuum effect when inferior translation is placed on the glenohumeral joint. Howell et al. found that the humeral head resisted tangential forces up to 60% of the compressive force applied.[37,38] Studies have shown that when a cadaver shoulder is stripped of all muscle, the humeral head remains centered on the glenoid, but when the joint is vented with a needle, the head then demonstrates increased inferior translation.[49] In multidirectional instability, a defect in the rotator interval capsule may "vent" the joint and reduce the effectiveness of the dependent passive restraints. With recurrent instability, there is deconditioning of the dynamic stabilizers and, ultimately, loss of effective concavity–compression. Furthermore, with loss of labral integrity, which functionally deepens the glenoid fossa by 50%, concavity–compression is further compromised. The importance of concavity–compression and glenoid positioning may be reflected by the fact that many patients respond to a rehabilitative exercise program directed at improving strength and neuromotor coordination of the rotator cuff and scapular musculature.[16,20,52]

Long before Neer's contemporary description of multidirectional instability, Basmajian and Bazant studied the problem of inferior glenohumeral translation.[7] They postulated that the glenoid inclination in the resting state faced superiorly. According to their hypothesis, the humeral head could only translate inferiorly if it moved laterally simultaneously (Fig. 14-4). Subsequent investigators found that the resting glenoid actually faces inferiorly, not superiorly.[48,58] Despite the glenoid inclination, lateral motion of the humeral head is *obligatory* if the head is to translate inferiorly. This is thought to be related to the increased thickness of the anterior inferior labrum.[31,82] Itoi et al. also studied biomechanical relationships of the

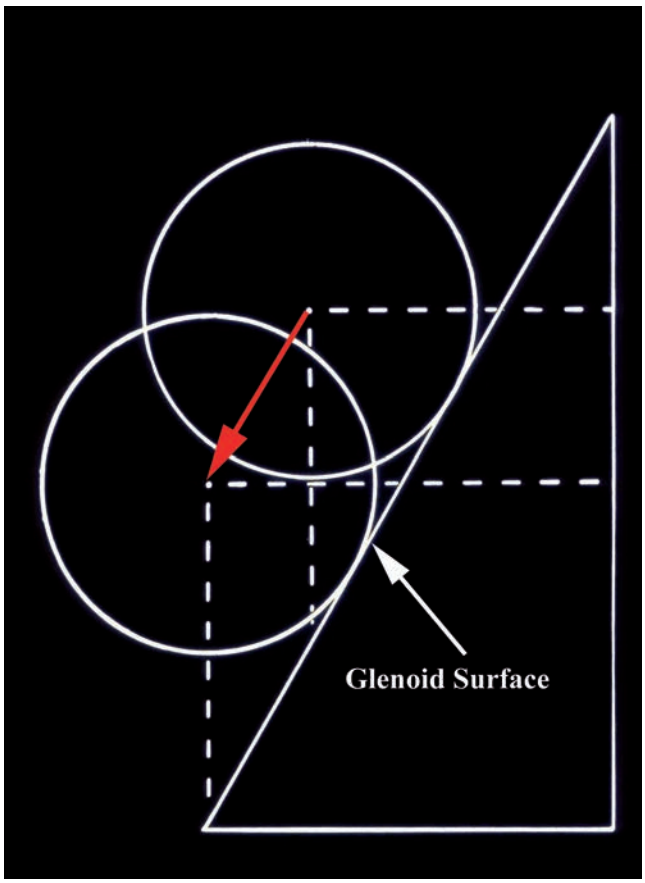


Figure 14-4 Biomechanical analysis of the glenohumeral joint suggests that regardless of glenoid inclination, the humerus is "obligated" to move laterally as it moves inferiorly. Although the natural glenoid may be inclined inferiorly, the thickened inferior labrum results in lateral motion of the head as it translates inferiorly.

glenohumeral joint and concluded, like Basmajian and Bazant, that there is a correlation between inferior scapular inclination and instability of the shoulder.[43] Several other authors have analyzed biomechanical effects that contribute to multidirectional instability.[42,72,77]

Two other anatomic lesions that result in multidirectional instability include a redundant inferior capsule and deficient rotator interval tissue. The triangular space separating the anterior edge of the supraspinatus from the superior edge of the subscapularis is defined as the rotator interval. It is normally bridged by a tissue considered capsule and is further defined as a "rotator interval capsule." Anatomically, the rotator interval includes the underlying superior glenohumeral ligament and is reinforced by the overlying coracohumeral ligament. Anatomic studies in cadaver specimens have led to the understanding that these structures together resist the inferior and posterior displacement of the humeral head. The superior glenohumeral ligament is thought to be the primary biomechanical restraint to inferior subluxation in the adducted arm. Patients with multidirectional instability consistently are found to have incompetence of this tissue. The rotator interval capsule is consistently characterized by the presence of either a discrete cleft or insubstantial and attenuated tissue. Defects in the rotator interval further disrupt the concavity–compression negative intraarticular pressure and may contribute to instability in this respect.[49,56] As the arm is progressively abducted, the inferior glenohumeral ligament complex functions as the primary restraint to inferior translation. In this abducted position, the anterior and posterior bands of this ligament reciprocally tighten with internal and external rotation. With a very large and redundant inferior capsule, it is easy to see how a patient may be clinically unstable in various positions of arm rotation while the arm is abducted.

Despite much investigation, a consistent biochemical explanation for capsular laxity remains elusive. Numerous studies have failed to identify a difference in the type or quantity of collagen between patients with multidirectional instability and controls.[79,84] Bell and Hawkins found that although there is no difference in the types of collagen between patients with multidirectional instability and controls, the patients with multidirectional instability had a significant increase in the rate of collagen formation.[10] Collagen fibril diameter and cross-linking are properties that are directly related to fiber tensile strength. Studies comparing capsular tissue from the shoulders of patients with multidirectional instability with those from patients with unidirectional anterior instability and from normal controls revealed some interesting findings. The capsular tissue from both instability groups demonstrated more stable and reducible collagen cross-links, greater mean collagen fibril diameter, higher cysteine concentration, and a higher density of elastin than did the normal samples.[79] However, one cannot conclude that these differences predispose clinical laxity. Skin samples from these same patients revealed significantly smaller mean collagen fibril diameter in patients with multidirectional instability than in those with unidirectional instability. This may suggest a possible underlying connective tissue abnormality.

More recent investigations of the cause of clinically significant multidirectional instability have sought a neurologic explanation.[6,51,86,88,96] Several observations support the idea of an underlying neuromuscular cause. Many patients with multidirectional instability in one shoulder have an equal or greater amount of laxity in their other, asymptomatic shoulder. In this condition, most symptoms occur in the midrange of motion where contribution of the ligaments to stability is minimal. High-speed photography of patients with symptomatic multidirectional instability reveals altered glenohumeral and scapulothoracic rhythms.[72] Most importantly, mechanoreceptors have been identified in shoulder joint capsule and proprioceptive deficits have been demonstrated both in patients with anterior instability and more recently in patients with multidirectional instability. It is possible that known proprioceptive receptors in the glenohumeral joint capsule, in addition to providing joint position sense, reflexly modulate rotator cuff forces during arm use to promote shoulder stability.[14,53] Patients with recurrent traumatic anterior instability appear to have deficits in joint-position sense compared with normal controls.[53] Although yet to be proven scientifically, a defect in proprioception may be a component of the cause of multidirectional instability. Interestingly, these deficits were shown to be reversible by surgical stabilization.[75,95]

One further plausible hypothesis is that the provocation of multidirectional instability occurs when the system of dynamic restraint is overwhelmed such as occurs when the arm is suddenly and unexpectedly moved or is fatigued due to repetitive use. This event, whether or not it results in identifiable trauma, results in pain and initiates a self-perpetuating cycle of increasing symptoms. When the now painful shoulder is protected, muscular weakness and subtle losses of refined neuromotor coordination are thought to ensue. Continued disuse further deconditions the dynamic constraints against glenohumeral instability, which are critical to maintaining stability in lax shoulders. With the further use of a deconditioned shoulder, the patient is more prone to experience painful episodes of instability, which promotes further disuse—and so on.[81]

Putting these various thoughts to practical use results in the concepts of the "shoulder at risk." These patients have multidirectionally lax shoulders that function well for years until a minor injury produces a loss of "balance" of the shoulder musculature. The resultant symptoms stem from an inability to control the entire shoulder girdle. The instigating incident may be small, repetitive, or large, but the result is an asynchronous firing pattern that eventually extends to the larger muscles of the shoulder

girdle, producing scapular winging, malpositioning of the shoulder girdle, secondary impingement, loss of proprioception, weakness, and pain. The scapula begins to protract, the humeral head begins to subluxate, and irritation develops in the rotator cuff and trapezius as these muscles try to compensate for the lack of normal dynamic serratus and rotator cuff function. It is the irritation of the tendons of the rotator cuff that produces pain in these lax shoulders.

The role of dynamic muscle contraction and coordination likely also play a role in shoulder stability. Dynamic, in vivo, electromyographic studies document rotator cuff and deltoid muscle activity throughout all shoulder motions.[39,73] Calculations reveal that the combined activity of the subscapularis, infraspinatus, and teres minor is balanced by the superiorly directed deltoid force vector.[73] The supraspinatus, which is more horizontally oriented, contributes more joint compression and, hence, stability. The net result of these muscle forces is that the force couple ensures that the humeral head remains precisely centered on the glenoid regardless of arm position.[27,39]

A relatively atraumatic onset of instability strongly suggests multidirectional instability. However, an episode of significant trauma can be a factor in a shoulder with excessive laxity. Reports in the literature suggest that athletes with symptomatic instability have such a history.[1,12] In this scenario, the athlete has a multidirectionally unstable shoulder in the presence of a Bankart lesion. Therefore, the presence of a Bankart lesion either on a radiograph or on a magnetic resonance arthrogram does not rule out multidirectional instability as the correct diagnosis. As Neer noted, this is a critical distinction to make.[64] He noted that when there is a history of significant traumatic event, multidirectional instability can be mistaken for a traumatic unidirectional instability. If a surgical repair designed for a unidirectional instability is performed, it is likely to result in a fixed subluxation in the opposite direction. Not only will the surgery fail in the short term, but the likelihood of developing arthritis of dislocation becomes a very concerning consequence.[33,81]

MANAGEMENT: NONOPERATIVE

Nonoperative management includes a physical therapy program *and* patient education. Perhaps in this group of patients education is the more important initial program because if patients do not understand or comprehend the nature of their problem, they are less likely to participate in the rigorous therapy program. Patients have ready access to many sources of information: magazine stories, the Internet, sportscasters, television, etc. Typically the media outlets are deemed to have more "authority" than the treating physician. Professional and collegiate athletes are often deified in the press and, in the eyes of the public, athletes with media visibility always seem to recover faster, have less pain, and return to sport sooner! Therefore, it is our obligation to educate our patients about the nature and natural history of their conition.

As physicians, we must take the time to compare and contrast, in understandable words and terms, the nature of multidirectional instability. We must take the time to explain anticipated time frames of improvement and realistic goals in return to sport. The patient must be made to understand that the nonoperative program has a good record of improving symptoms and returning their arm to their premorbid level of comfort and function.[2,4,20,60,63,64]

One helpful tactic to use in patients with multidirectional instability who also have laxity of their uninvolved shoulder is to emphasize that compensated laxity (such as their asymptomatic shoulder) can result in a normally functioning arm. Patients must learn that their unstable shoulder has become deconditioned from its usual state and that they need to regain both strength and neuromotor coordination of the stabilizing muscles of the rotator cuff, deltoid, and scapula. Burkhead and Rockwood reported satisfactory results in 88% of patients who had symptomatic multidirectional instability treated with a specific program of physical therapy.[16,57]

If patients present with pain, suggesting an inflammatory synovitis, a program of oral nonsteroidal antiinflammatories should be initiated before commencing the rehabilitation program. Only rarely are mild narcotic analgesics required. The need for such medications should suggest some other cause of the patient's symptoms. When using antiinflammatories, one should respect the fact that effort is being made to treat the *condition,* not just the *symptoms.* Accordingly, the antiinflammatory choice should be one that allows the patient to obtain, and then maintain, a therapeutic level of the medication 24 hours a day for about 6 weeks. Long-acting antiinflammatories that have a half-life of at least 12 hours (twice-a-day dosing) result in the best therapeutic response and ensure the greatest probability of patient compliance.

From a basic science standpoint, the goals of shoulder rehabilitation are to restore effective concavity–compression through strengthening, to identify and correct abnormal muscle firing patterns, and to improve deficient proprioceptive function.[17,52,62,87] The exercise program consists of two phases. Phase I concentrates on progressive resistance exercises utilizing elastic bands for strengthening of the rotator cuff and deltoid musculature. Initial emphasis is directed toward the infraspinatus and subscapularis muscles. They are performed with the elbow at the side and with the arm below 90 degrees of abduction to avoid the possibility of shoulder impingement. Within several weeks, as progress is made, strengthening routines for the scapular muscles, including the rhomboids, levator scapulae, and serratus anterior, are incorporated.[46] Phase II exercises are added at about 3 months, which are designed to develop and retrain humeroscapular coordination.

The specific technique of the strengthening is a critical component for successful rehabilitation.[3,92] Several important concepts are conveyed to the patient to ensure the most successful outcome of this nonoperative program. The elastic tubing is pulled only through a short arc in an isotonic contraction, not more than 45 degrees. The resistance is held for at least 5 seconds in an isometric contraction, and then released *very slowly* to maximize the eccentric component of the muscle function. It has been demonstrated that the eccentric phase of muscle physiology is the most efficient type of contraction that restores or improves its strength.[36] The exercises are repeated in sets of 10 and are performed twice daily.

Because the patients who develop symptomatic instability of any type are usually teenagers and young adults who are not known to be especially compliant with exercise routines, special emphasis must be given to review the anticipated time frames of anticipated benefit. Frequently, if the patients do not perceive benefit from the exercise routines within the first week, they become less compliant and the exercise program is doomed to failure. The patients must be made aware that it may take at least 3 months before any benefit from the muscle exercises is perceived. Thus, it is nearly always appropriate to initiate treatment with an exercise program if only to evaluate patient compliance. It has been well understood that a surgical procedure does not make an otherwise noncompliant patient suddenly compliant in the necessary postoperative rehabilitation program. The exercises are continued for a minimum of 6 months. If successful, a program of maintenance exercises is given to be performed indefinitely.

Rehabilitation Concerns

Nonoperative treatment centers initially on dynamic stabilization of the scapulothoracic articulation and pain-free strengthening of the rotator cuff. Active abduction and "empty can" exercises that increase impingement in these unbalanced shoulders should be avoided until symmetrical scapulothoracic and glenohumeral motion develops. Attentive physical therapy is essential for biofeedback during the early phases of rehabilitation. All exercises must be performed with the scapula retracted to avoid rotator cuff irritation. As the patient is usually unaware of the malpositioned scapulothoracic articulation, hands-on therapy, taping, or bracing is required to ensure correct shoulder position during rehab. Recently, biofeedback braces emphasizing correct scapula position have been utilized with excellent improvement in correct positioning of the scapula (Fig. 14-5A,B). Correct scapular positioning is essential, as any rotator cuff exercises done with a protracted shoulder will increase irritation of the rotator cuff rather than strengthen it. As these patients do not have an awareness of the position of the shoulder, these braces hold great potential in increasing the success and compliance of nonoperative treatment.

Patients must be informed that they need to regain both strength and neuromuscular coordination of the rotator cuff, deltoid, and scapula. It is essential that the treating physician emphasize the need for correct scapula positioning during all exercises.[56]

Wirth, Burkhead, and Rockwood have reported on conservative therapy in patients with multidirectional instability, and reported satisfactory results in 88% of

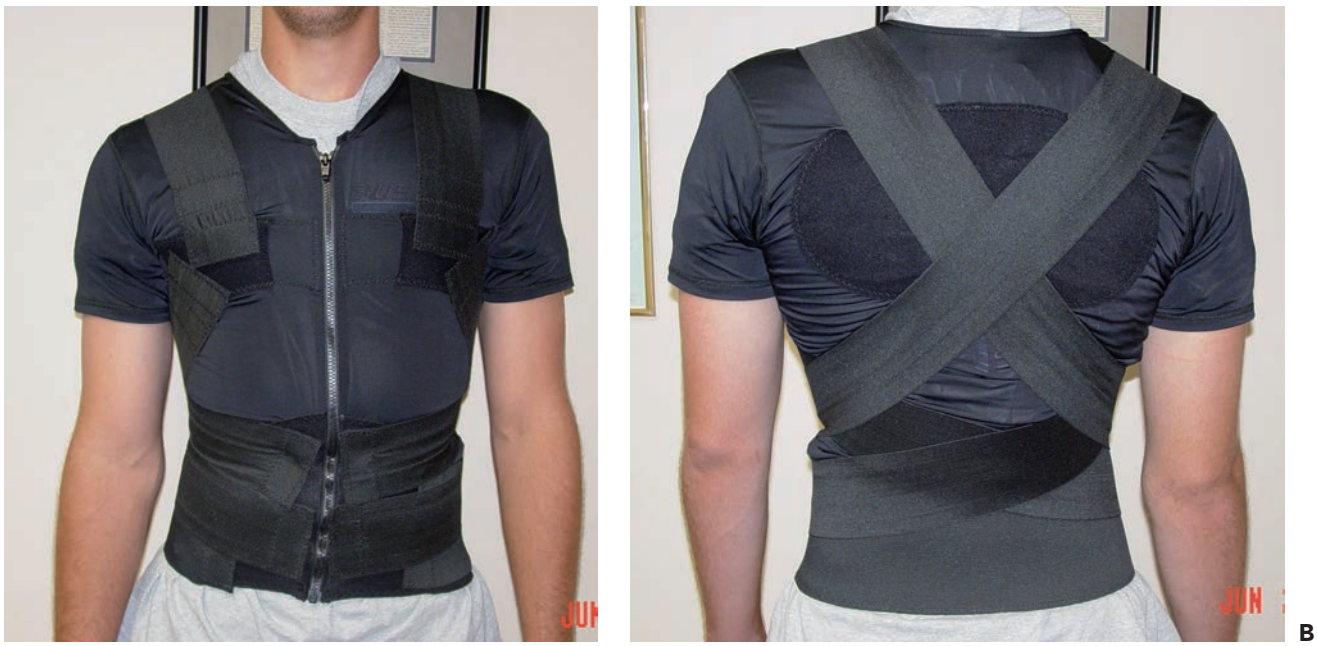


Figure 14-5 **(A,B)** The biofeedback scapular brace used to restore normal scapular kinematics.

patients who underwent a specific program of physical therapy.[16]

In the acute painful phase, antiinflammatory treatment is beneficial. Although many physicians recommend nonsteroidal antiinflammatory drugs, we have not found them to be particularly helpful. In addition to a course of physical therapy, an intraarticular shot of corticosteroids and a short course of low-dose oral prednisone may be initiated before beginning therapy in an attempt to calm the inflammation. We generally recommend this aggressive treatment plan if the patient is experiencing night pain and has palpable swelling. It is emphasized to our patients that although this will help with the pain, it is merely palliative to allow proper rehabilitation and not curative. These patients will begin utilizing the scapular brace immediately as tolerated, progressing to utilization of the dynamic supports during all waking hours.

A minimum of 6 months of adequate therapy is recommended before consideration of surgical intervention.

Results of Nonoperative Treatment

Burkhead and Rockwood reviewed 140 shoulders in 115 patients who had a diagnosis of traumatic, atraumatic, or multidirectional instability who were treated with a specific set of muscle strengthening exercises. They noted that in the subgroup of traumatic instability, only 16% of the shoulders had a successful outcome from exercises alone compared to 80% of the shoulders with a diagnosis of multidirectional instability.[16] They concluded that it is critically important to define the exact nature of the instability through history, physical examination, and radiographic studies so that appropriate treatment programs can be initiated.

Kiss and coinvestigators reviewed a series of 84 shoulders in 59 patients who had a diagnosis of multidirectional instability.[47] All patients were treated nonoperatively with a specific exercise routine. At an average 3.7-year follow-up, 38 shoulders were either "cured" or improved with an exercise program alone and only four required surgery. They also concluded that those patients who had a failed surgery for instability prior to the exercise program had a much poorer outcome with physical therapy alone.[47]

Brostrom and coworkers reviewed a series of 33 shoulders in 29 patients with recurrent instabilities of several types.[15] Twenty-eight shoulders improved and only four required surgery at 1 year. As has been stated with respect to surgical stabilization, failures may occur many years later. Nonoperative treatment should also be evaluated over similar lengths of time before ascribing success to the treatment plan.

There is little guidance in the literature regarding either long-term or short-term bracing in the treatment of multidirectional instability. Ide and investigators have recently reviewed their experience combining a custom-made brace with a shoulder strengthening program.[41] They studied a cohort of 46 patients (73 shoulders) with multidirectional instability and quantified changes in muscle strength over the time of the study. All patients performed a structured exercise routine for 8 weeks. At a mean follow-up of 7 years, only three patients required surgical stabilization of the persistent shoulder instability. Uhl and Kibler have recently reported on the use of a biofeedback scapular brace in the preoperative treatment of MDI with increased success.

MANAGEMENT: OPEN INFERIOR CAPSULAR SHIFT

Patients who fail to respond to 3 to 6 months of nonsurgical treatment are considered candidates for surgical intervention. The patient must have been compliant in the execution of the nonoperative rehabilitation program. Surgery is not offered to voluntary dislocators with emotional problems or to behaviorally immature teenagers. Neer and Foster described the open inferior capsular shift in 1980, and it remains the standard procedure for surgical repair.[63] Over the years, numerous variations of capsular dissection, plication, and repair have been added, but the original inferior capsular shift remains the standard to which other modifications are compared.[1]

In recent decades, advances in the field of surgical anesthesia have found direct benefit in this procedure. In the 1980s, regional interscalene block anesthesia had not been in widespread use for shoulder surgery. The use of general anesthesia made it difficult to apply a modified shoulder spica in the operating room at the conclusion of the surgical procedure. Neer and Foster felt it especially important to place the patient in a cast immediately upon completion of the procedure to minimize forces on the newly repaired capsule.[63] Recognizing the need to free the repaired capsule from multidirectional forces, the arm could not be placed in a sling in the internally rotated position because it would stress the posterior portion of the repair. The inferior forces created by the weight of the arm had to be eliminated by supporting the arm in the spica with the downward forces absorbed by the pelvic brim. Lastly, the spica cast had to be placed to keep the arm in neutral rotation, superiorly and anteriorly directed. Today this may be accomplished by a removable brace, but there will be further discussion of this issue later in the chapter. It is easy to understand that applying a cast to a patient who is under the effects of general anesthesia is a sizeable challenge.

With the more widespread use of interscalene block anesthesia, patients can be awake, alert, and, most importantly, cooperative during cast or brace application. Additionally, there have been numerous reports in the literature on the benefits of ultra–long-acting anesthetic agents in the management of postoperative pain. The authors' present choice is the use of Chirocaine supplemented with

Diprivan (propofol). This combination provides for excellent hypotensive surgical anesthesia and effective muscle relaxation while providing up to 18 hours of postoperative analgesia. A disadvantage of using interscalene block alone is that it prevents the ability to examine the uninvolved shoulder. Perhaps its most valuable asset is that this combination of agents allows the patient to be awake and cooperative at the completion of the procedure to allow placement of the cast or brace in the operative suite.

After the patient is fully anesthetized, a careful examination of *both* shoulders is performed. Assessment of instability patterns, including direction and degree, is documented for each shoulder. This examination should be used to reinforce the office clinical examination and to make a more detailed assessment of the instability and laxity patterns. It is particularly dangerous to use the examination under anesthesia as a substitute for an office clinical examination. Great harm can be done to a patient if asymptomatic laxity is misinterpreted during an examination under anesthesia and an inappropriate capsular tightening procedure is performed.[94] It should be restated for emphasis: The examination under anesthesia should be used to *reinforce* a clinical diagnosis made during repeated office examinations.

The direction of the surgical approach is dictated by the associated pathology. The presence of a labral defect will usually direct the surgical approach. Some authors feel that the surgical approach should be made from the direction of the maximal instability, while others feel that the anterior approach should be the default approach, unless there is a posterior labral tear.[13] An approach from the most unstable side allows for direct imbrication and reinforcement while simultaneously shifting the capsule to reduce global capsular laxity. If there is an anterior labral tear documented by magnetic resonance arthrography, an anterior approach is made even in the presence of a significant component of posterior instability. Similarly, if there is a documented posterior labral tear, a posterior approach is made for the capsular shift procedure even in the presence of significant anterior translation. When an open repair is appropriate for isolated posterior instability, this author performs an anterior approach in an effort to protect the critical function of the infraspinatus muscle and its role in dynamic stabilization. A potential drawback to the posterior approach is the thin and pliable nature of the posterior capsule. The more robust anterior capsular tissues provide for a more secure repair, and appropriate posterior capsular tensioning can be achieved through the capsular shift. An anterior approach also facilitates closure of the rotator interval, a necessary step in the surgical management of most multidirectionally unstable shoulders.[29] A posterior labral lesion, in the presence of a multidirectionally unstable shoulder, demands a posterior approach, either arthroscopic or open.

The patient is placed in a semi-recumbent, beach-chair position (Fig. 14-6). A special headpiece is used to permit access to the superior aspect of the shoulder. It is useful to have the longitudinal axis of the body at the far edge of the bed so the arm and shoulder extend beyond the operating table itself (Fig. 14-7). This allows the operative shoulder to be unencumbered by the bed and facilitates shoulder extension.

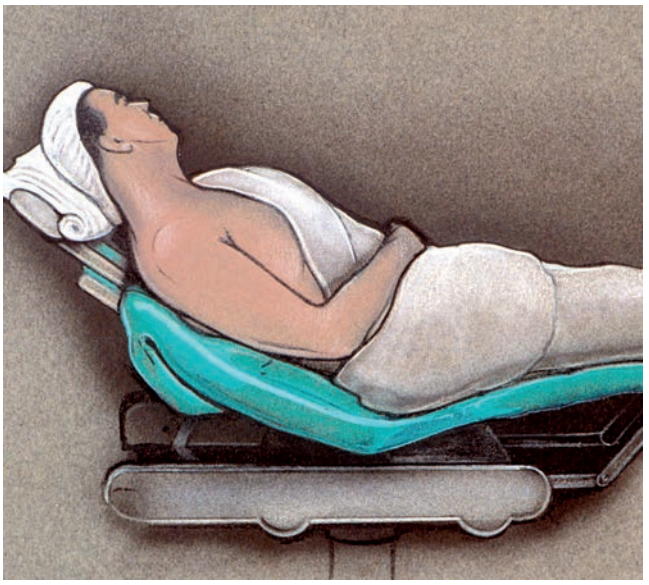


Figure 14-6 The patient is placed in a beach-chair position with the back at 45 degrees from the horizontal. This position allows excellent access to the medial and inferior aspects of the joint capsule.

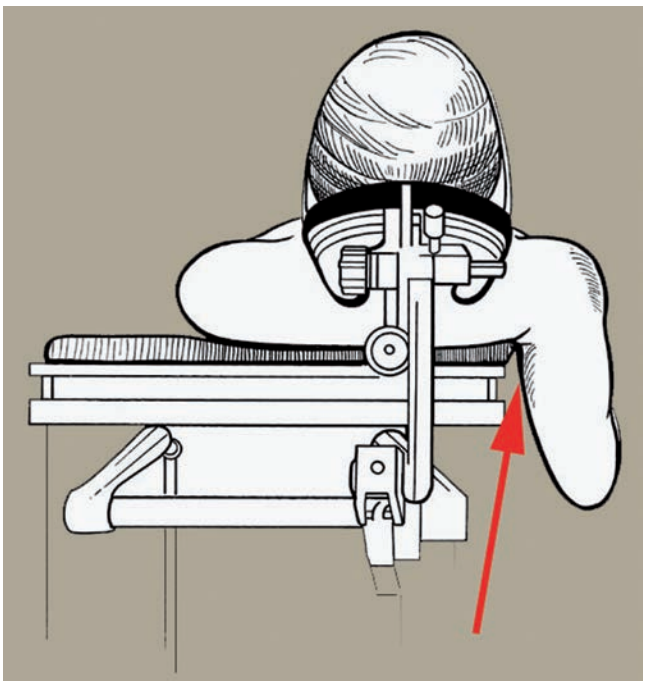


Figure 14-7 The head is placed on a head support, which allows the patient's body to be translated off the edge of the bed. As the arrow shows, this facilitates extension of the arm during the procedure.

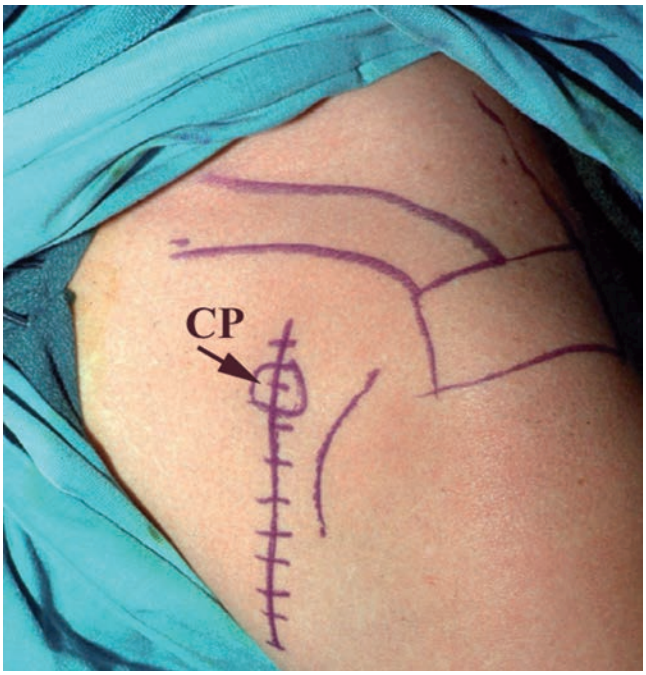


Figure 14-8 The incision is vertical in orientation and begins in a skin crease at the apex of the axilla. It extends superiorly to the coracoid process (*CP*).

The location and placement of the surgical skin incision is made according to the surgeon's choice but must permit access to both the deltopectoral interval while also allowing access medial to the glenohumeral joint. Therefore, the incision of choice is one that originates at the tip of the coracoid process and extends inferiorly to the superior aspect of the axilla (Fig. 14-8). Prior to making the incision, the arm is adducted across the chest and a skin fold at the superior margin of the axilla is marked. In making an incision along this skin fold, which joins the coracoid tip, a very cosmetically acceptable scar predictably results. Some surgeons choose to confine the incision to the axilla itself, thereby avoiding a scar on any part of the anterior aspect of the shoulder. While this is commendable from a cosmesis standpoint, deep surgical exposure becomes much more challenging.

Once the incision is carried down to the investing fascia of the pectoralis major and deltoid muscles, extensive skin flaps are developed. Care must be exercised during the initial incision down to the fascia because the incision will cross the cephalic vein in the midportion of the wound. Although the vein often lies deeper, embedded in fat, on occasion it has been found more superficial within the deltopectoral interval and can be lacerated inadvertently. The skin flaps must be extended laterally to the anterolateral margin of the deltoid, superiorly to the level of the clavicle, medially to the base of the coracoid process, and inferiorly to the apex of the axilla. This degree of tissue mobilization is necessary to ensure medial and inferior access to the capsule. This degree of exposure also provides the ability to visualize and protect the axillary nerve, which is at considerable risk during the capsular shift procedure. The vascular supply to the shoulder is abundant, and this author has never seen or read of a circumstance where skin necrosis resulted either from the location of the incision or from the extensive dissection of skin flaps.

Once the flaps have been created, the deltopectoral interval is developed from the clavicle at the superior extent of the wound, and distally to the deltoid insertion. It is predictably easier to take the vein laterally with the deltoid muscle because the majority of the feeding veins drain from the deltoid. On the other hand, it is not altogether uncommon to find some smaller veins from the pectoralis major also draining into the cephalic vein, but they are easily coagulated and divided. There is some risk in taking the vein laterally in that it must take a relatively longer course during retraction of the deltoid muscle. This is especially problematic if the superior aspect of the cephalic vein penetrates the clavipectoral fascia medial and inferior to the coracoid. In this circumstance, either the vein should be kept in continuity and dissected in such a way that it is retracted medially with the pectoralis major, or it is ligated proximally and the distal portion retracted laterally with the deltoid. Ligation of the cephalic vein in a young patient, free from other vascular or lymphatic problems, is not known to have any adverse effects.

As the deltopectoral interval is developed and opened, the deltoid branches of the thoracoacromial trunk are encountered in the upper third of the wound. They course from the medial to the coracoid to the medial aspect of the deltoid on its deep surface and consist of an artery and several veins. Once these vessels are ligated or coagulated, the deltopectoral interval can be widely opened from the clavicle at the proximal extent to the deltoid insertion at the distal extent.

The clavipectoral fascia is incised parallel to the deltopectoral interval. This incision should be made lateral to the muscle fibers of the short head of the biceps. In more muscular individuals, a moderate amount of muscle tissue extends lateral to the tendon of the short biceps, even up to the coracoid process where it originates. In keeping the clavipectoral fascial incision lateral to the muscle fibers, bleeding is minimized. At its superior extent, the clavipectoral fascia is initially incised up to the anterior border of the coracoacromial ligament. Inferiorly, the clavipectoral incision extends to the superior border of the tendons of the pectoralis major insertion.

Returning to the superior aspect of the wound, the acromial branch of the thoracoacromial trunk is coagulated as it courses on the superior aspect of the coracoacromial ligament. The anterior third of the coracoacromial ligament is incised over the rotator interval. This interval is easily palpated just inferior to the coracoacromial ligament and approximately 1 cm lateral to the coracoid process. The

posterior two-thirds of the coracoacromial ligament is kept intact to provide whatever component of superior stability it may contribute. There is no indication to "decompress" the shoulder in a patient with a clinical instability problem.

Inferiorly, the superior 2 cm of pectoral major insertion are divided from their insertion on the shaft of the humerus. Both the superficial and deep heads are divided simultaneously, which provides significantly better exposure to the inferior aspect of the glenohumeral joint. Furthermore, by mobilizing the now released portions of the pectoral major, the areolar tissue joining the long and short heads of the biceps can be divided. Doing this with the cautery minimizes bleeding of the numerous small vessels that inhabit this area. At the completion of this inferior dissection, the inferior aspect of the glenohumeral joint should easily be seen; the axillary nerve can be safely palpated and protected. For those surgeons who are more comfortable only after visualizing the nerve, this inferior exposure also facilitates that effort.

To permit the medial exposure, a plane is developed under the lateral border of the short head of the biceps and conjoined tendons. A retractor is placed to reflect the conjoined tendons medially, thus protecting the musculocutaneous nerve specifically and the entire brachial plexus. This maneuver brings the anterior humeral circumflex vessels into view at the inferior aspect of the subscapularis muscle. These vessels tend to run an extremely variable course with respect to the muscle and tendon of the subscapularis. While some surgeons have advocated their preservation, routine ligation or coagulation has not resulted in any documented cases of avascular necrosis in this patient group. Furthermore, when dissecting the capsule inferiorly and posteriorly for a complete inferior capsular shift, these vessels must be transected for capsular exposure and repair.

At this point, the subscapularis insertion should be exposed in its entirety. One will notice that the superior portion of the muscles has thick tendon that attaches to the lesser tuberosity. The tendinous portion of the tendon extends medially, usually for several centimeters. Conversely, the inferior portion of the muscle has no tendon and the muscle fibers themselves insert directly onto the humeral shaft inferior to lesser tuberosity. The rotator interval defines the superior aspect of the subscapularis. An incision is made in the *superficial* portion of the subscapularis only, beginning at the rotator interval superiorly and extending to the inferior aspect of the glenohumeral joint. This incision is made 1 cm medial to the tip of the lesser tuberosity (Fig. 14-9). By making the incision at this location with respect to the lesser tuberosity, there should be an adequate sleeve of tendon tissue remaining on the tuberosity to allow a tendon-to-tendon repair at the completion of the procedure.

Once through the anterior one-half of the tendon, the scalpel is placed in the coronal plane and the dissection proceeds medially. An important concept is to split the subscapularis tendon coronally with the desire to keep the posterior fibers of the tendon in their normal intimate contact with the underlying capsule (Fig. 14-10). This will serve to reinforce the often tenuous capsule. The anterior half of the tendon is dissected medially until the muscle fibers are encountered. At this point, the muscle fibers are teased from the capsule and the construct composed of the muscle belly of the subscapularis in continuity with the anterior half of its tendon is retracted medially. This then results in the anterior capsule with overlying posterior subscapularis tendon attached (Fig. 14-11). During the dissection of the subscapularis tendon, it is important to note the horizontal orientation of the tendon fibers. Dissection performed within this layer of horizontally oriented fibers ensures the proper dissection plane. Lastly, it is critical to understand the three-dimensional aspect of the dissection. Because the humeral head (and overlying soft tissue) is essentially spherical, as the subscapularis dissection proceeds inferiorly, the scalpel begins relatively posterior, advances relatively anterior at the midaxis, and finally becomes relatively posterior again at the inferior portion of the dissection. At the same time, as the dissection is developed medially, the scalpel begins relatively posteriorly, becomes more anterior as the dissection progresses medially, and finally becomes more posterior at the medial extent of the tendon release. The concept of peeling an apple with a scalpel *and keeping the skin intact* becomes a convenient visual aid.

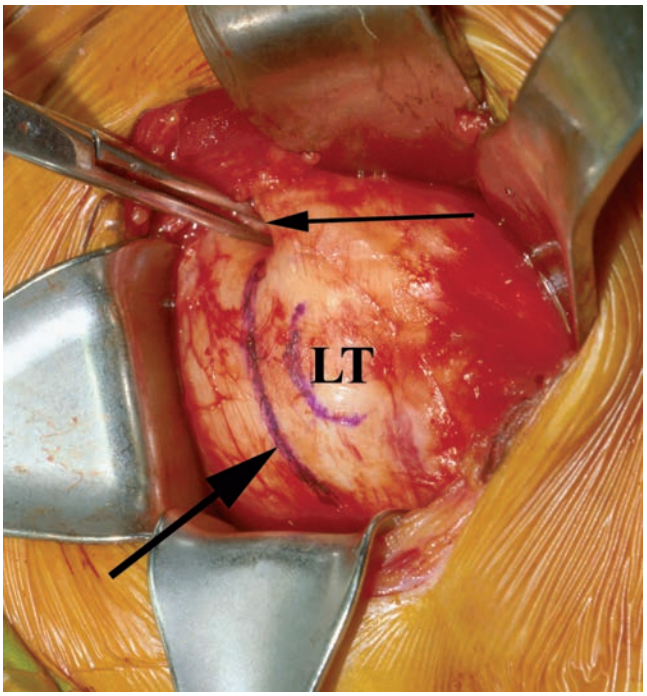


Figure 14-9 The thick arrow marks the line of subscapularis incision. It begins 1 cm medial to the lesser tuberosity (*LT*) and starts at the rotator interval (*narrow arrow*).

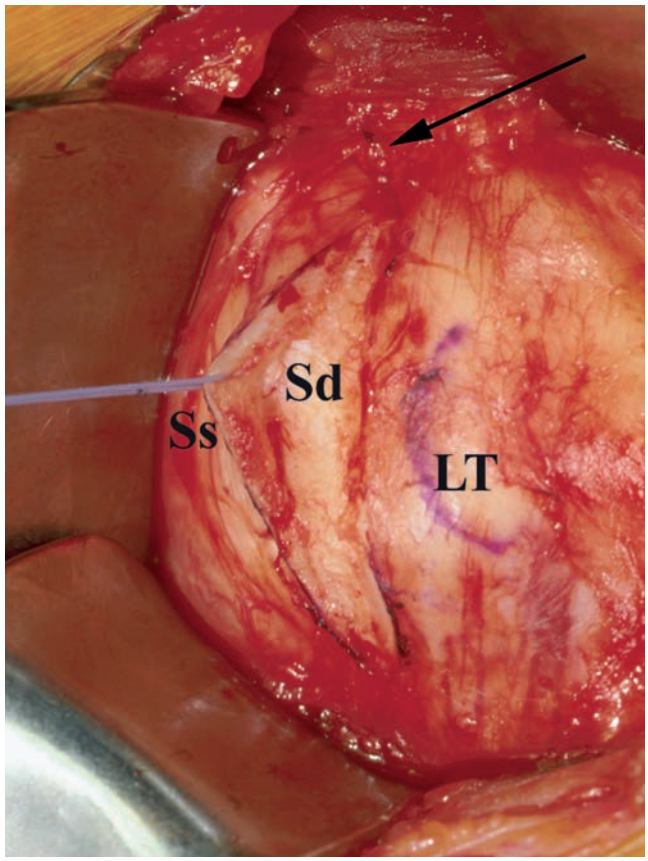


Figure 14-10 Once the subscapularis incision (*Ss*) is made to the appropriate depth, horizontal fibers will be seen in the deep portion of the tendon (*Sd*). The dissection continues medially separating the superficial portion of the tendon from the deeper portion. The arrow delineates the superior extent of the tendon at the rotator interval. LT = lesser tuberosity.

With the subscapularis retracted medially, the rotator interval is easily seen and, in the patient with multidirectional instability, is typically widely opened (Fig. 14-12A,B). This interval is reapproximated at this time with nonabsorbable suture. It is easiest to begin the closure medially and progress laterally. At the upper portion of the interval, just beneath the anterior border of the superior glenohumeral ligament, lays the long head of biceps tendon (Fig. 14-13). During the rotator interval closure, care must be taken to ensure that this tendon is not inadvertently tenodesed. During the interval closure, the anterior border of the superior glenohumeral ligament complex is approximated to the superior border of the middle glenohumeral complex. As will be seen, this maneuver will result in a superior capsular flap based off of this closed interval.

At this point, before the capsular incision is made, a surgical skin marker is used to mark a line along the margin of the resected subscapularis tendon at the lesser tuberosity. This will ensure the ability to repair the subscapularis at its anatomic length. Furthermore, as traction sutures are placed in the subscapularis during its dissection, notation is made on the lateral flap with the marking pen to ensure the anatomic superior and inferior relationship at the time of reconstruction.

The capsulotomy is made, again beginning at the rotator interval and extending down to the 6 o'clock position on the humeral neck (Fig. 14-14). This incision is made at the anatomic neck of the humerus, which can be palpated before the incision is made. The capsular incision should be made in a way that leaves approximately 1 cm of capsular tissue remaining in the humeral neck. It is this remaining tissue on the humerus to which the capsule will be repaired after the shift is completed. At the midportion of the capsular incision, a separate capsular incision is made toward the center of the joint. Traction sutures placed in the corners of the superior and inferior capsular flaps facilitate the exposure. As the capsular incision proceeds medially, it should be aimed toward the Bankart lesion if one is present. Variations on capsulotomies have been described by several authors.[1,40] Some recommend midcapsular incisions while others prefer a glenoid-based capsular release and repair.[40] While each author advocates a specific technique for specific reasons, the classic description that has yielded exceptional results is reviewed here. Releasing the capsule laterally provides the greatest options in capsular management with the least risk to vital neural structures while providing excellent surgical outcomes.

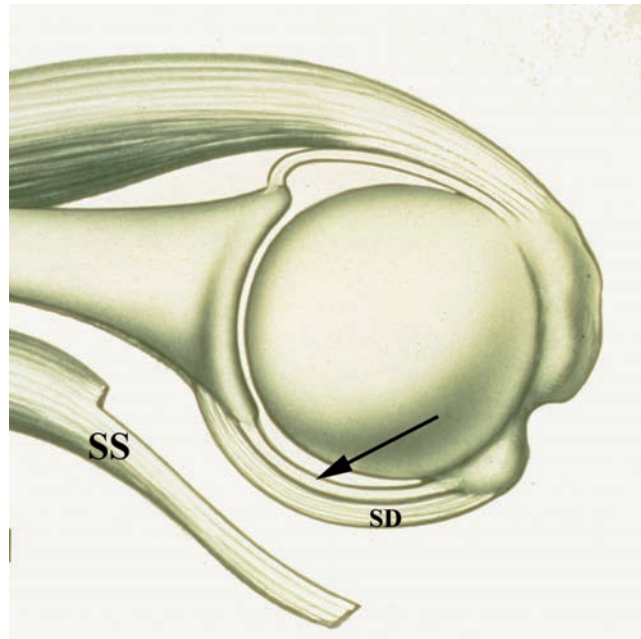


Figure 14-11 This cross-sectional view demonstrates how the anterior portion of the subscapularis tendon (*SS*) is continuous with the entire muscle belly. The deeper portion of the tendon (*SD*) is kept in continuity with the anterior shoulder capsule (*arrow*) to reinforce it.

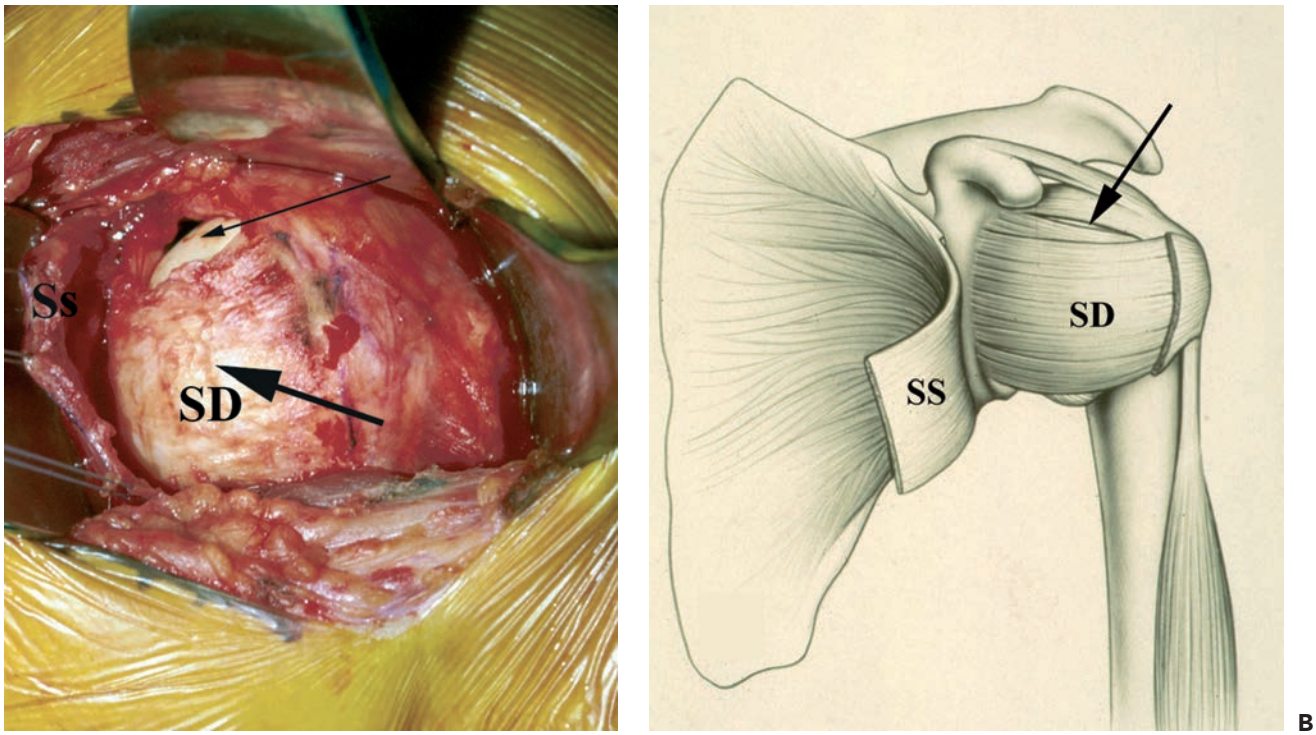


Figure 14-12 **(A)** The anterior half of the subscapularis tendon (*SS*) is retracted medially. The deeper portion of the tendon remains fixed to the deeper shoulder capsule (*SD* and *thick arrow*). The thinner arrow demonstrates the widely patent rotator interval. **(B)** Schematic drawing of the intraoperative photograph (A). Note that the entirety of the muscle belly of the subscapularis is contiguous with the anterior half of its tendon. The arrow points to the open rotator interval, which is a cleft between the superior border of the middle glenohumeral ligament and the anterior border of the superior glenohumeral ligament. The long head of the biceps tendon lies immediately beneath the upper edge of the superior ligament.

A ring retractor is placed in the joint to carefully displace the humeral head posteriorly. This permits a thorough examination of the joint and the labrum in particular. Often there is evidence of synovitis and hemosiderin deposition in the periarticular tissue as evidence of instability and hemorrhage. The articular surface of the glenoid may appear "scuffed" and discolored. In the classic case of multidirectional instability, the humeral head will fall so far posteriorly that the entire glenoid fossa is visible with little retractor force.

At the completion of the joint inspection, any labral pathology is addressed and repaired. With the ring retractor removed, an assessment is made of the inferior capsular volume. Although there is no truly objective means to measure the inferior capsular volume, it is generally understood that during intraoperative assessment, it should not be able to accept more than the distal phalanx of an average index finger! Perhaps a slightly more scientific way of judging the presence of symptomatic inferior capsular laxity is at the time of the clinical examinations with manifestation of a symptomatic sulcus (dimple) sign.

With global multidirectional instability, the capsular dissection proceeds from inferior to posterior (Fig. 14-15). It is this segment of the dissection that places the axillary at most risk. Knowledge of the location of the nerve in three dimensions becomes absolutely critical. Even in Neer and Foster's hands, in their original description, they documented temporary axillary nerve dysfunction in several patients.[63] The key to a safer dissection is to continually and progressively forward flex the arm with simultaneous increasing external rotation. These maneuvers alone enhance the ability to release the capsule from the humerus inferiorly and posteriorly while maximizing nerve safety. The axillary nerve is at most risk with the arm in the adducted or abducted position. The inferior and posterior capsule is released as one contiguous structure to include the posterior band of the inferior glenohumeral complex. This is generally at the midequatorial level of the posterior humeral neck (Fig. 14-16). As the capsular dissection progresses, traction sutures are placed in the tissue at 1-cm intervals. They serve to pull the capsule anteriorly and further buffer the axillary nerve from injury.

When the capsule is completely released, the shoulder is reduced, allowing the head to become centered and balanced on the glenoid. By placing gentle traction on the capsular sutures, the posterior capsule is advanced inferiorly and the inferior capsule is simultaneously advanced anteriorly, thus reducing the overall capsular volume (Fig. 14-17). Predictably, the anterior corner of the capsule where the initial capsulotomy was made will be pulled superior to the

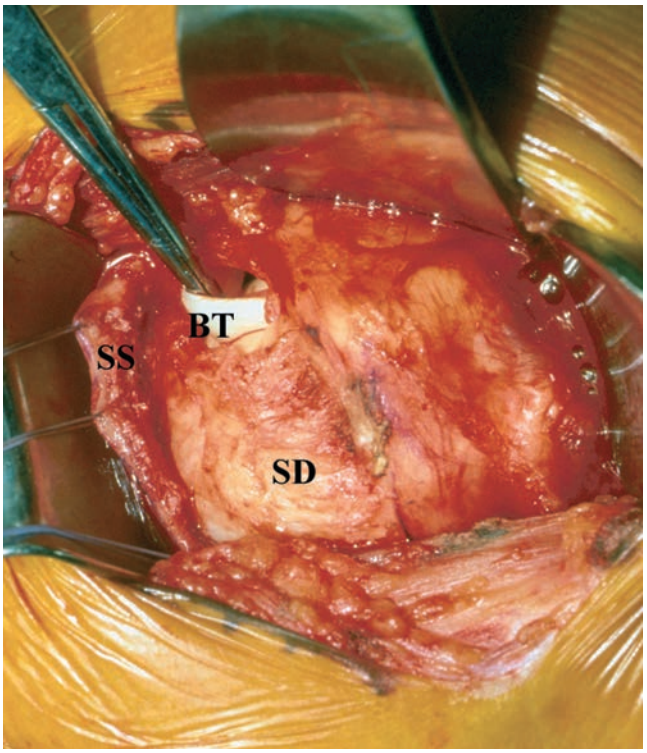


Figure 14-13 The long head of biceps tendon (*BT*) is immediately under the upper edge of the interval tissue. Care must be taken not to tenodese it inadvertently at the time of interval closure. The superficial (*SS*) and deep (*SD*) portions of the subscapularis are shown.

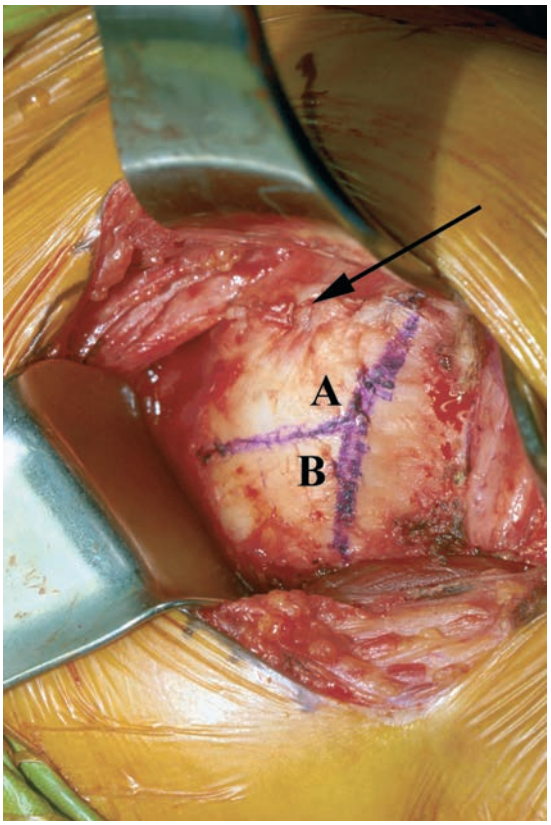


Figure 14-14 The blue marks show the line of the reinforced capsular incision. The incision begins at the rotator interval superiorly and extends to the inferior portion of the humeral neck. The horizontal incision extends to the glenoid and is aimed at the Bankart lesion if one is present. This results in capsular flaps based at the glenoid. The superior flap is tagged with a suture at (*A*); the inferior flap is similarly tagged at (*B*).

rotator interval and well above the superior capsular flap. This represents the amount of excess capsular tissue. While holding these traction sutures, the surgeon can now place his or her finger in the inferior pouch and verify that the capsular volume has been eliminated. Once satisfied that the capsular release is complete, the repair is initiated.

A high-speed burr is used to decorticate the anatomic neck of the humerus where the capsule is to be repaired. Again, holding the arm in forward flexion and external rotation while decorticating the inferior and posterior humeral neck will facilitate axillary nerve protection. At the time of capsular reconstruction, the arm is positioned in 10 degrees of forward flexion and in 45 degrees of external rotation. It is critical not to overtighten the capsule while at the same time making every effort to balance the capsular forces posteriorly, inferiorly, and anteriorly. The development of arthritis of dislocation has been well established when capsular forces are excessive in any direction or when an imbalance of capsular tension is iatrogenically produced.[33]

The capsular repair incorporates a "pants-over-vest" suture technique, which lays the mobilized capsule along the decorticated humeral neck and positions the lateral capsule that remained on the humerus over it. As the repair is performed, the capsule is pulled cephalad to hold the capsular volume reduced and balanced. Frequently, at the

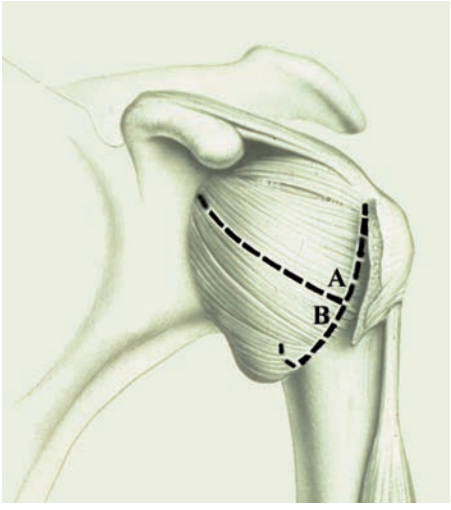


Figure 14-15 Schematic representation of Fig. 14-14. Note that the capsular incision is carried well around posteriorly while simultaneously forward flexing and externally rotating the arm. This position minimizes risk of injury to the axillary nerve.

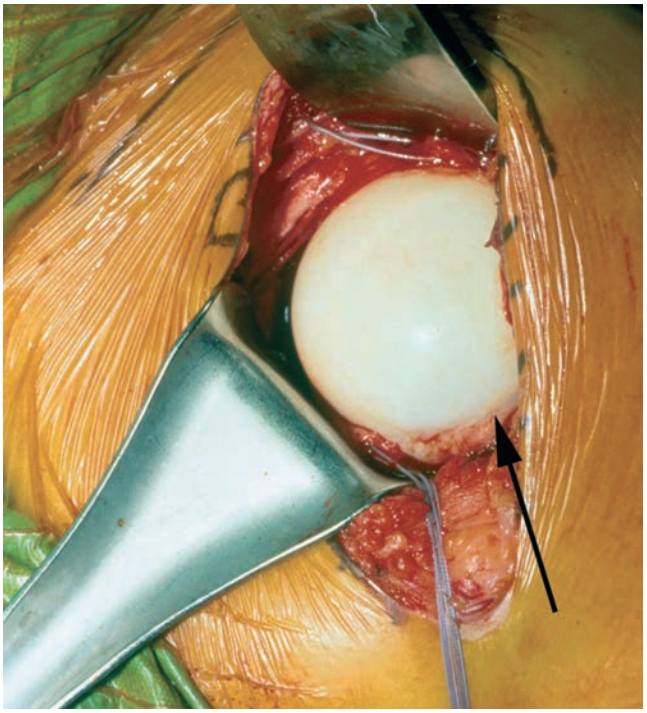


Figure 14-16 When a complete capsular release is accomplished, the entire humeral head can be seen. The arrow points to the inferior aspect of the humeral neck when the shoulder is reduced and the arm is in the anatomic position. That region between the arrow and the blue traction sutures represents the posterior aspect of the humeral neck where the capsule has been released.

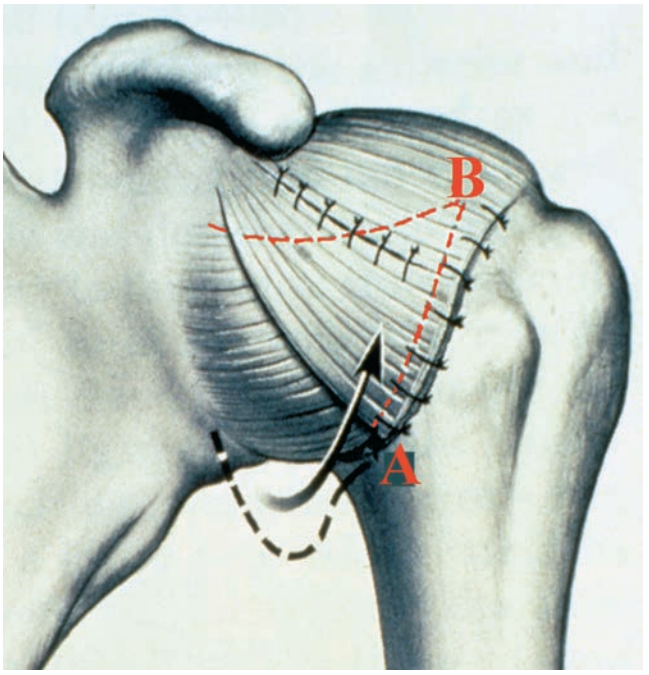


Figure 14-17 This schematic drawing demonstrates how the capsule is advanced during the capsular shift. Point (*B*) is the apex of the inferior flap, which has been advanced from posterior to inferior to anterior, reducing the capsular volume. The apex of the superior flap (*A*) is pulled inferiorly. This results in a "double breasting" of the anterior capsule.

completion of the inferior flap repair, there is excess capsular tissue, which is resected at the level of the rotator interval. Once the inferior flap is secured to the humeral neck, the superior flap is pulled inferiorly and laterally and similarly repaired to the humerus. This has the effect of doubly reinforcing the anterior capsule in a "cruciate" fashion. When both portions of the capsule have been secured, joint motion and stability are assessed. Perhaps the most critical assessment is that of external rotation with the arm at the side. With rare exception the arm should fall to a point of 45 degrees of external rotation with *no force applied*. Forward flexion should be to 160 degrees effortlessly. Any motion short of these goals should result in revision of the capsular repair.

At the completion of the capsular repair, the subscapularis is sutured back at its anatomic length as determined by the previously placed marked tissue. Not only is restoration of anatomic length important, but also appropriate position with reference to the superior and inferior placement is necessary for normal physiologic function.

Following irrigation of the wound, a drain is placed in the subacromial space and the deltopectoral interval is repaired. The skin is closed with a running absorbable subcuticular suture, which provides a nearly invisible incision line when healing is complete.

ARTHROSCOPIC TECHNIQUES

The indications for surgery via arthroscopy are the same as those previously written for open surgery. A further consideration involves the experience and skill of the operative surgeon with both arthroscopic and open surgery. The need for preoperative rehabilitation and counseling is similarly the same. Functional impairment and pain along with a failure of adequate nonoperative treatment, including integrated rehabilitation, is the only indication for surgical intervention of any kind.

Arthroscopy offers several advantages over open surgery in the management of MDI. Diagnostically one can get an overview of the joint under direct inspection, ascertaining the presence of labral tears, capsular tears, and capsular and rotator interval laxity. Although the surgeon will already know the primary directions of instability by the history and physical examination both awake and under anesthesia, repeating the examination while visualizing the movement with the arthroscope in the joint is quite useful (Fig. 14-18). The usual advantages of arthroscopy—preserving muscle attachment, better visualization of pathology, anatomic specific repairs based on this visualization, small incisions, and less pain—also apply.

Once the decision to operate is confirmed by the patient and the operating physician, surgery can be initiated. The surgery can be performed in either the beach-chair or lateral

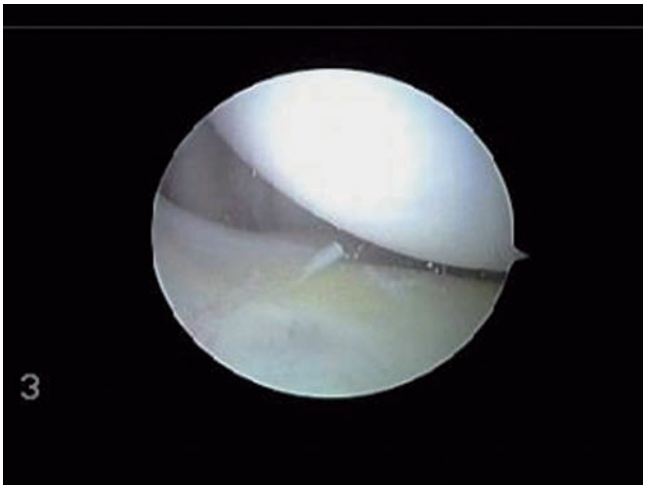


Figure 14-18 Arthroscopic view of a shoulder showing shifting of the humeral head on the glenoid during diagnostic arthroscopy.

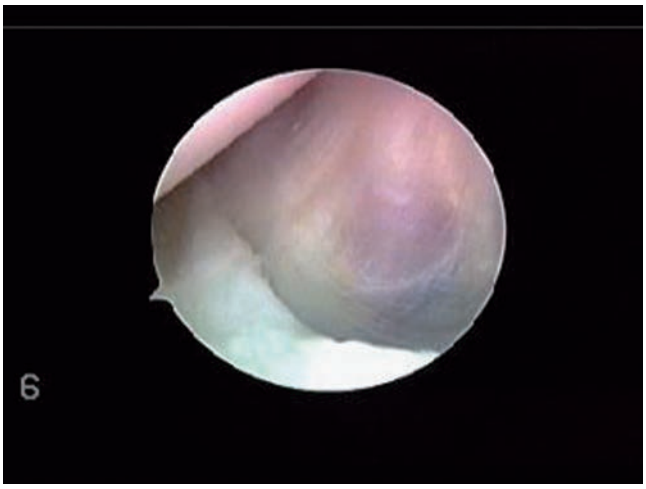


Figure 14-20 A lax anterior capsule.

decubitus position. We feel the lateral decubitus position is easier in the management of MDI, but both have been shown to be successful.

We routinely use an interscalene block for postoperative analgesia placed by an experienced regional anesthesiologist prior to general anesthesia. This helps significantly with postoperative pain and allows the use of less anesthetic agents and narcotics during the surgery. In the beach-chair position the head and neck must be carefully controlled to prevent traction injury to the brachial plexus. In the lateral decubitus position a minimal amount of weight (5 to 10 lb, just enough to suspend the arm) is used for the same reason.

Once adequate positioning, prepping, and draping have occurred, the diagnostic arthroscopy can begin. Specific areas of evaluation include the rotator interval (Fig. 14-19), anterior capsule (Fig. 14-20), inferior capsule (Fig. 14-21), and posterior capsule (Fig. 14-22A). In many patients with MDI the posterior capsule may be so thin that the muscle of the infraspinatus can be readily visualized through the capsule (Fig. 14-22B).

One should also visualize the attachment of the anterior and posterior capsule to the humerus, looking for capsular splits, perforations, or humeral avulsion of the glenohumeral ligament lesions (Fig. 14-23A,B).

TREATMENT OF THE LAX CAPSULE

The lax capsule of reasonable quality is best managed by plication sutures. It is up to the individual surgeon as to the use of absorbable or nonabsorbable suture. The initial step involves abrasion or cutting of the capsule from the preserved labrum. This may be performed with a

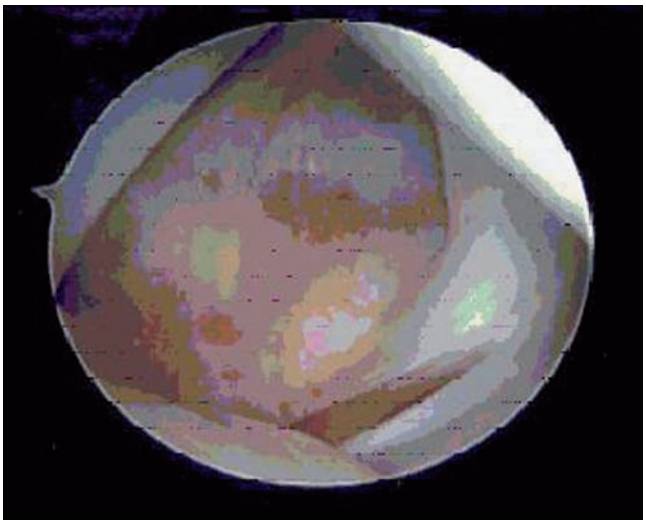


Figure 14-19 A lax rotator interval.

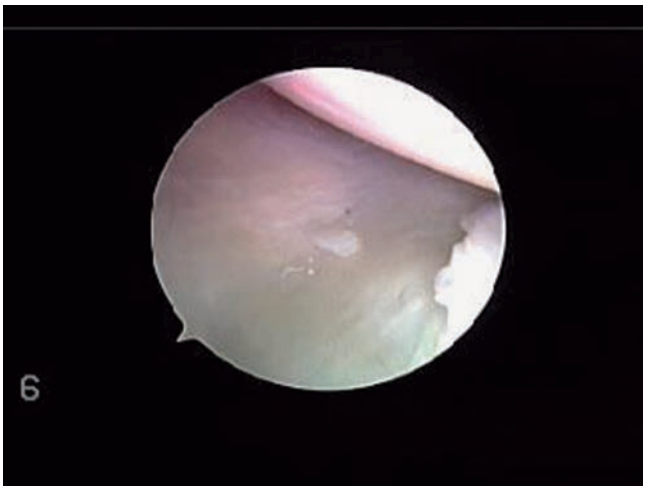


Figure 14-21 A lax inferior capsule.

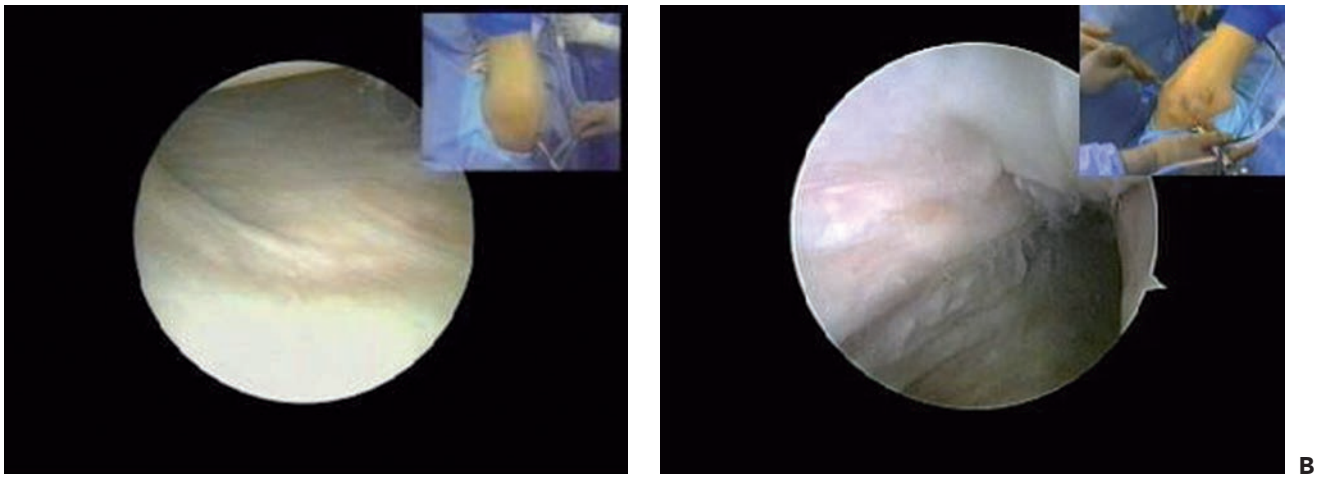


Figure 14-22 **(A)** Arthroscopic view from the anterior portal of a lax posterior capsule. **(B)** Arthroscopic view from anterior of a thin posterior capsule, showing muscle fibers of the infraspinatus through the capsule.

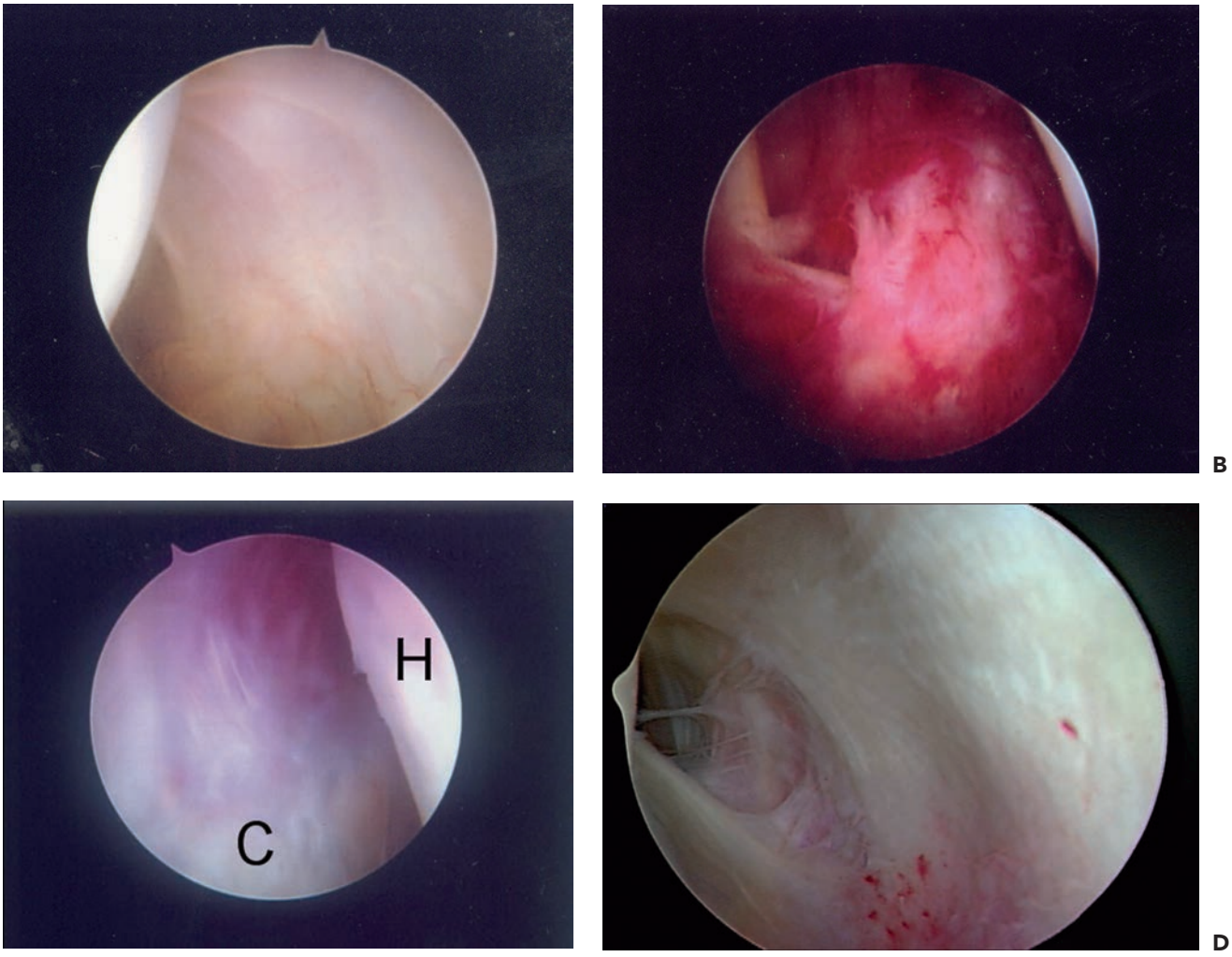


Figure 14-23 **(A)** The normal attachment of the anterior capsule to the humerus. **(B)** Arthroscopic view of a capsular shift. **(C)** Arthroscopic view of an anterior humeral avulsion of the glenohumeral ligament (HAGL) lesion. **(D)** Arthroscopic view of a posterior HAGL lesion.

shaver or with a rasp (Fig. 14-24) to create a healing bed of tissue. A suture hook is then used to perforate the capsule approximately 1 cm from the labrum. The exact site is determined by drawing an imaginary line parallel to the glenoid and to the capsule; the first suture is placed through the capsule at the 6 o'clock inferior position and rotated until the hook emerges from the capsule. The entire capsule is then advanced superiorly and anteriorly (Fig. 14-25A) until the capsule appears taught (Fig. 14-25B). This is the point of advancement of the first suture. The hook is then placed beneath the labrum and delivered into the joint between the labrum and the articular surface (Fig. 14-25C). The suture is delivered and then retrieved and tied by arthroscopic knot tying. We favor a self-locking knot, but half-hitches or knot-tying devices may certainly be used.

These steps are repeated along the anterior aspect of the capsule. The second stitch is usually placed through the capsule at the 5 o'clock position and advanced to the

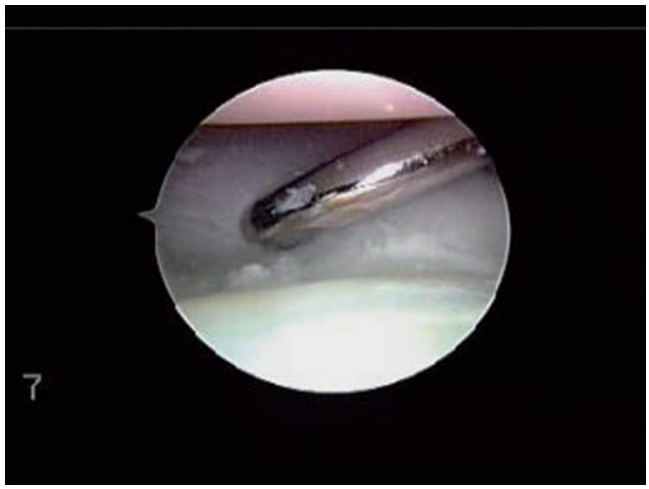


Figure 14-24 Rasping of the capsule to create a healing bed in preparation for capsular placation.

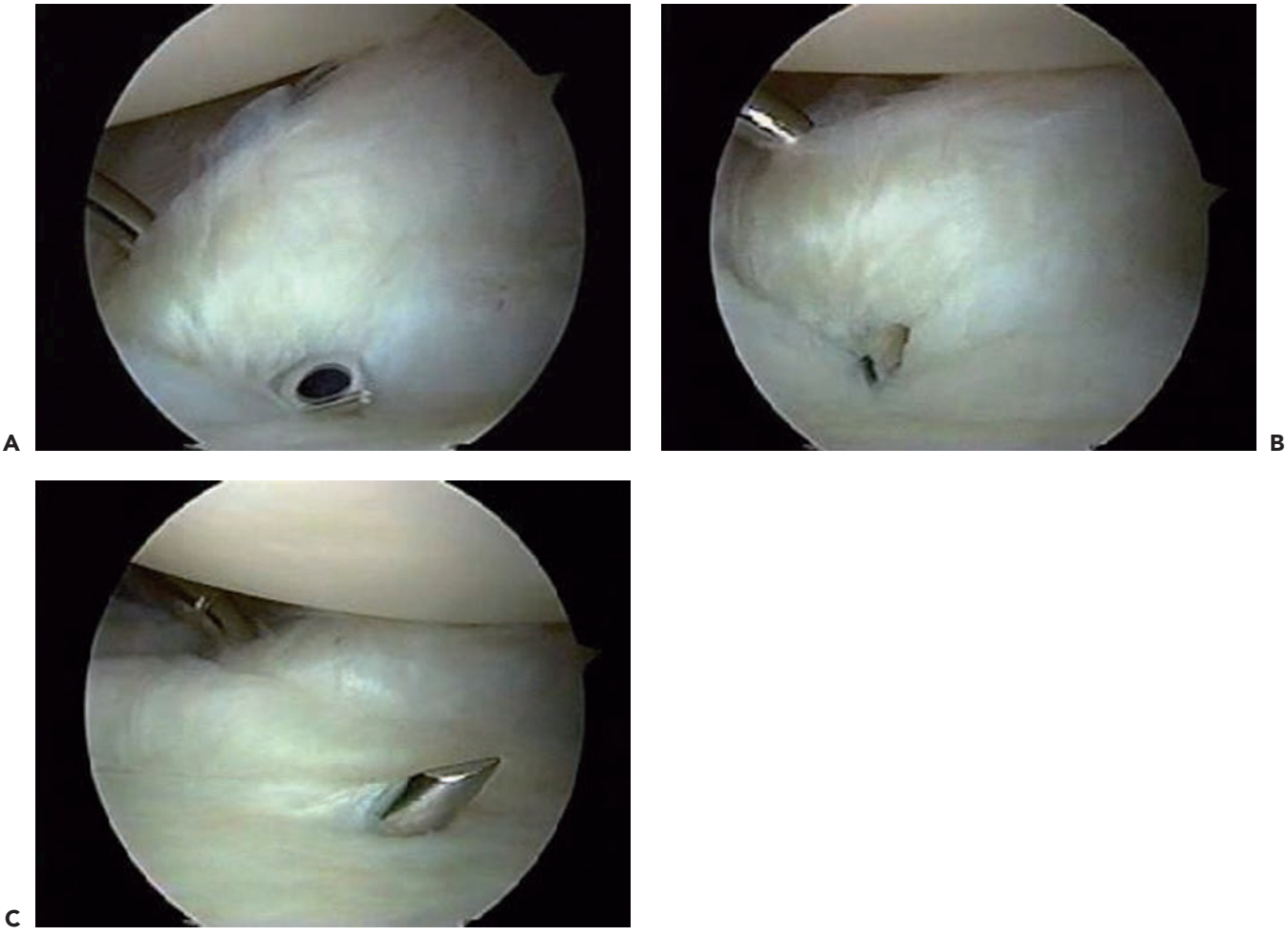


Figure 14.25 **(A)** View from anterior of the suture hook through the inferior capsule. **(B)** The suture hook is pulled superiorly to vertically shift the capsule as much as possible. **(C)** The suture hook is placed between the labrum and articular cartilage to anchor the shifted capsule to the labrum and create a buttress to anterior shifting.

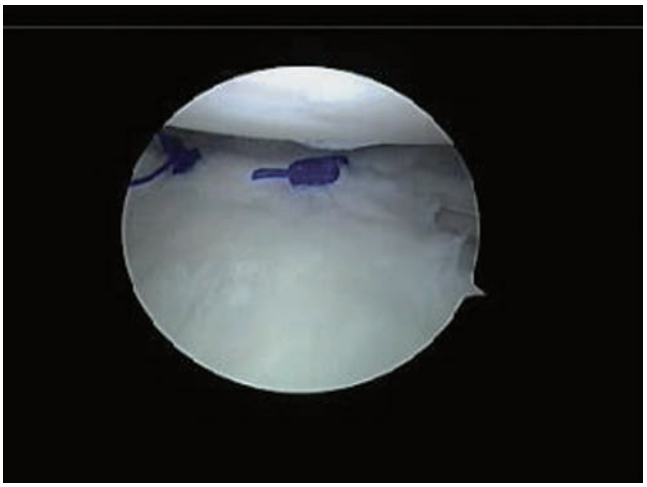
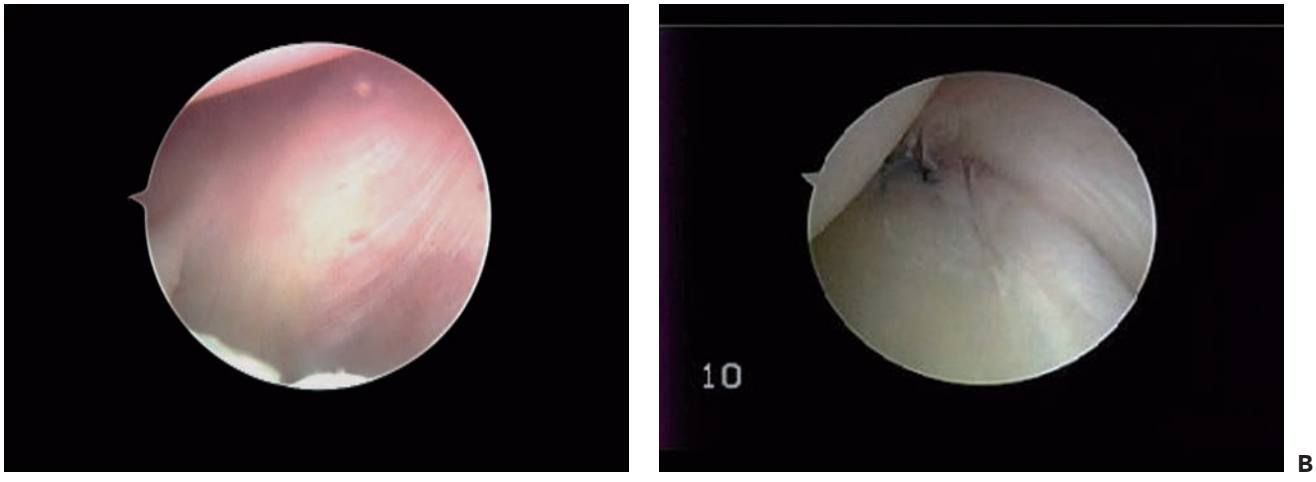


Figure 14-26 Arthroscopic view from posterior of a completed anterior capsular shift.

4 o'clock position on the glenoid. Additional sutures are placed until the entire anterior capsule is reconstructed (Fig. 14-26).

The posterior capsule is then addressed in a similar way, beginning with the 7 o'clock position and continuing superiorly until all capsular redundancy is eliminated (Fig. 14-27A,B). In many cases of MDI the posterior capsule is insufficient for plication sutures. In these cases one may use a suture plication technique that includes the infraspinatus tendon. In these cases the lateral capsule is pierced percutaneously with a large lumened spinal needle (Fig. 14-28A) and a suture placed though the needle into the joint. The initial stitch should enter the capsule around the 7 o'clock position. The suture is grasped and the needle removed. A suture retrieval device is then placed through the capsule adjacent to or under the labrum and the suture retrieved out of the posterior canula (Fig. 14-28B). The canula is then retracted until it is just outside the infraspinatus and, using a switching stick, it is placed into the subacromial bursa. A crochet hook is utilized to blindly grab the suture while watching from inside the joint. One should see the canula indenting the infraspinatus during this retrieval. This suture is then tied and the capsule tightening assessed (Fig. 14-28C). These steps are repeated until the posterior capsule is sufficiently tightened. In most cases two to four sutures are needed.

The arthroscope is then placed posterior above the reconstruction and the rotator interval assessed. In many cases the superior–anterior plication may have already closed a majority of the interval and the procedure may be finished by using the same suture hook to close the middle and superior glenohumeral ligaments as described by Wolf (ref here of Eugene Wolf treatment of mdi). In most cases, however, additional suturing of the interval is necessary. To tighten both the inner and outer layers of the interval, a spinal needle is inserted approximately 1 cm from the articular margin just at the anterior edge of the supraspinatus (Fig. 14-29A). A nonabsorbable suture is placed through the needle into the joint and placed for retrieval. The anterior canula is then pulled out of the joint until it is just anterior to the anterior layer of the rotator interval (which is anterior to the subscapularis). A suture retriever device is placed through the anterior layer of the rotator interval tissue, around the subscapularis, and through the capsule (Fig. 14-29B). The suture is then grasped and pulled out the anterior canula. A switching stick is then used to rotate the canula over the subscapularis tendon into the bursa. The suture is then grasped blindly with a crochet hook as described for the posterior technique and tied using a self-locking knot, tightening the rotator interval (Fig. 14-29C). Additional sutures may be placed using the same technique as needed.



A B

Figure 14-27 **(A)** Arthroscopic view of a lax posterior capsule. **(B)** Arthroscopic view from anterior of a plicated posterior capsule.

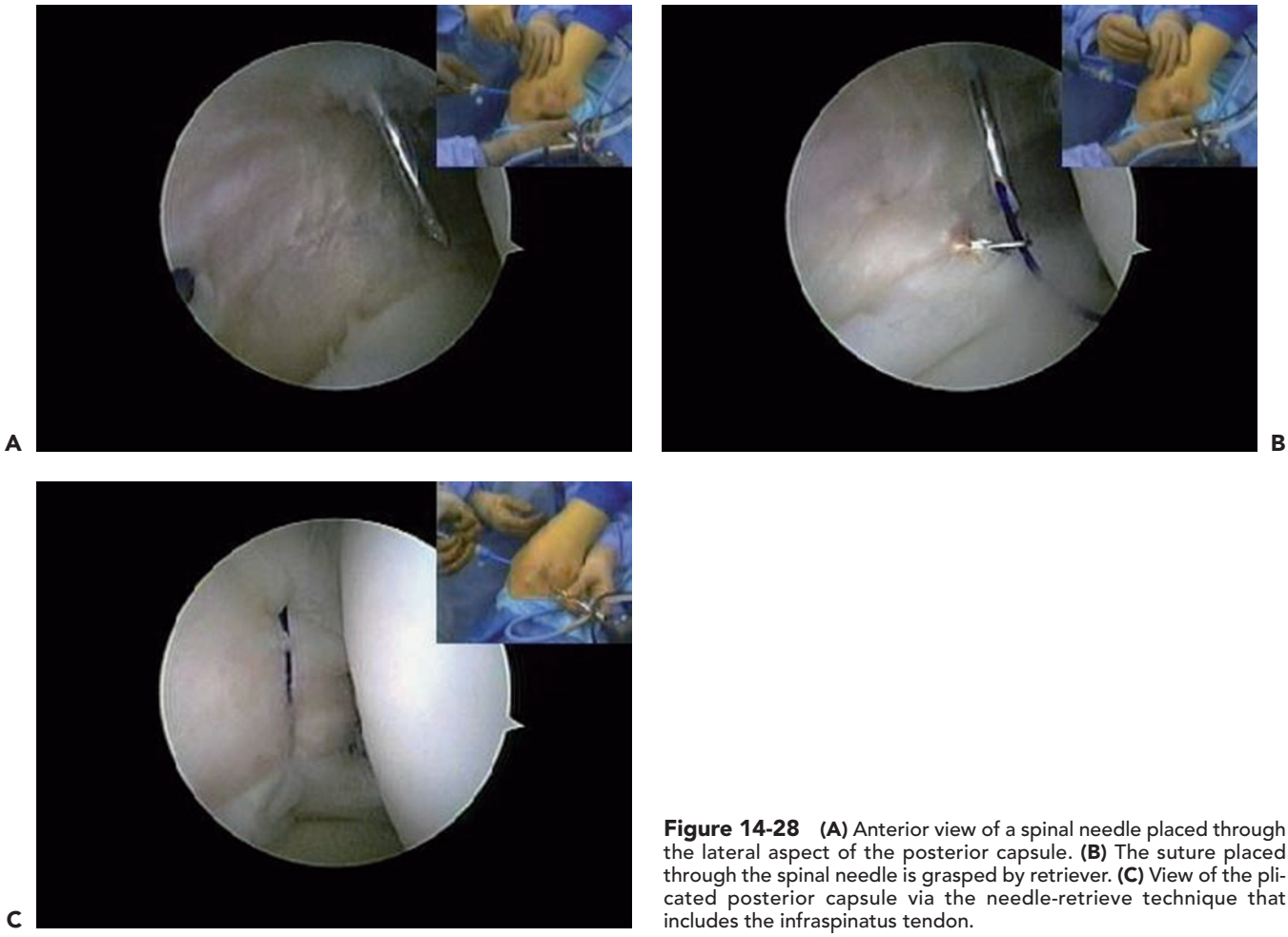


Figure 14-28 **(A)** Anterior view of a spinal needle placed through the lateral aspect of the posterior capsule. **(B)** The suture placed through the spinal needle is grasped by retriever. **(C)** View of the plicated posterior capsule via the needle-retrieve technique that includes the infraspinatus tendon.

TREATMENT OF THE TORN CAPSULE AND SPECIAL TECHNIQUES

In many cases MDI may be associated with labral or capsule tears. In these patients it is important to remember that additional treatment of the capsule is necessary. The labral tear and/or capsule split are repaired using techniques described elsewhere in this text (see Chapter 12). Once the repair has been accomplished, the inferior capsule and opposite side are plicated as described above. If this restores adequate stability, the rotator interval may be addressed. In all patents with MDI it is important to close both layers of the interval in addition to any other procedures that are performed.

Several modifications can be made in the previously described techniques. The capsule may be incised rather than abraded to allow a more complete shift. The initial suture may be used as a shuttle, pulling a larger, nonabsorbable suture through the same area (Fig. 14-30). A retrieval device may be used through the capsule in the same area to create a mattress suture while still using the labrum as an anchor.

In cases in which there is a labral tear or the labrum is deficient, an anchor may be placed into the glenoid and the sutures retrieved through the capsule. The recent availability of dual-sutured anchors makes this an attractive option in some cases with capsular splits or tears.

The use of thermal devices in the management of MDI has fallen into disfavor in recent years. Although still utilized by many surgeons with excellent results, the variability of the effect of such devices on the tissue and the long-term effects of intraarticular temperature extremes on articular cartilage may present a caution as to its ongoing use. If utilized for capsular laxity, we recommend suturing around the treated area and additional sutures in the rotator interval to supplement the thermal procedure.

COMPLICATIONS

Arthroscopic management of MDI has the same risk of complications as the open procedures. Stiffness and recurrence of instability both can occur, although the incidence of each has been quite low in published reports.

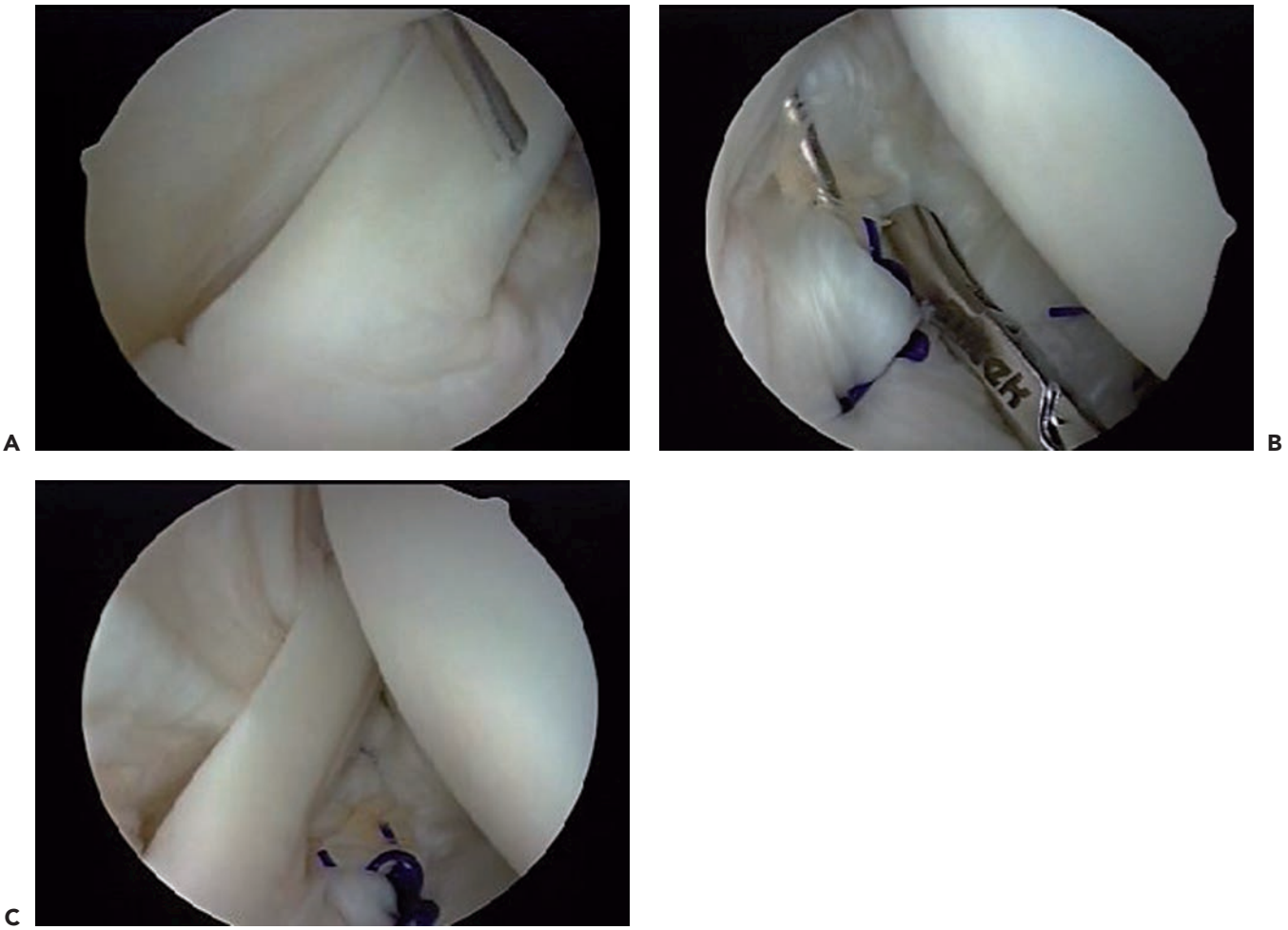


Figure 14-29 **(A)** View from posterior showing the spinal needle coming in from superiorly at the posterior edge of the rotator interval (anterior edge of the supraspinatus). **(B)** The retriever is used to pierce the anterior layer of the rotator interval, around the subscapularis, and through the posterior layer of the interval and perhaps the middle glenohumeral ligament. **(C)** View from the posterior of the closed rotator interval.

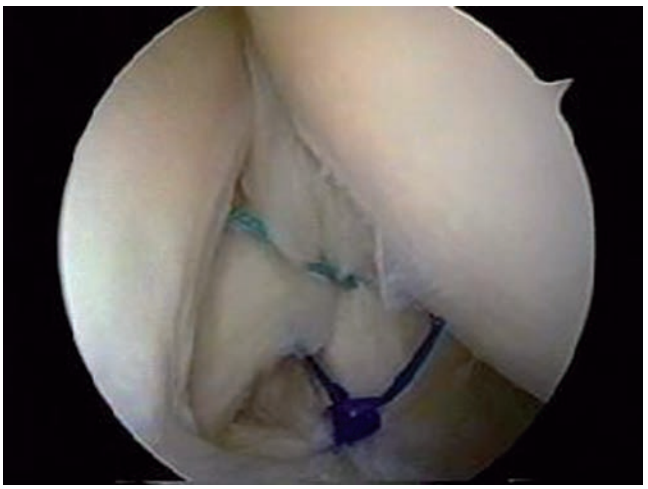


Figure 14-30 View from the posterior of the plicated anterior capsule using a nonabsorbable suture.

The most devastating complication reported as a result of thermal management has been chondrolysis. The exact mechanism by which this destructive disease occurs has yet to be elucidated, and chondrolysis has also been reported in association with other procedures. Management of the shoulder with chondrolysis includes intraarticular injections of steroids or hyaluronic acid, arthroscopic débridement, interposition grafting, and humeral head replacement.

POSTOPERATIVE MANAGEMENT

The patient is placed in either a gunslinger brace or an abduction sling before transport to the recovery room. The brace is maintained with the exception of personal hygiene for 6 weeks. Initial rehabilitation centers on trying to maintain correct shoulder posture while in the brace.

Each of these shoulders has a unique response to surgery. In those in whom the capsule repair is maturing rapidly, passive motion and scapular stabilization exercises may begin at 4 weeks, but in most cases these are delayed until 6 weeks postoperatively. Occasionally the abduction sling is maintained and exercise delayed until 8 weeks postreconstruction. Active exercise begins at 6 to 8 weeks with careful attention to maintain correct scapular position during all exercises. In patients in whom the scapula remains protracted, early dynamic bracing is initiated to retrain the scapula to remain in its correct retracted position. This may include static bracing or taping for short periods of time. It is vital that the patient and therapist recognize the need to reestablish proper shoulder positioning in space at all times, but especially during all exercises. The more quickly normal shoulder posture is reestablished, the more likely there will be a good recovery.

Once the capsule reconstruction has matured, based on clinical endpoint examination and lack of palpable capsular edema (usually 4 to 6 months) and the patient is able to maintain correct scapular position, the therapy is progressed to include rotator cuff strengthening exercises, proprioceptive neuromuscular facilitation exercises, and plyometrics. No passive stretching by the therapist is allowed during the first 6 months. Sometime in the 4 to 8 months postoperatively, integrated rehabilitation as described by Kibler is initiated along with sport-specific conditioning in the athletic population. Sports are allowed between 6 and 12 months postoperatively, depending primarily on shoulder position and tracking patterns.

POSTOPERATIVE CONSIDERATIONS

The immediate aftercare following an inferior capsular shift has become "debatable" with the introduction of a variety of orthotic brace devices. Nevertheless, the *principles* of postoperative management should not change. If the patient had a true inferior capsular shift procedure as detailed above, then it follows that the capsular repair must be protected in *all of its components and directions.* Surgeons generally understand that following an anterior repair such as a Bankart procedure, the arm is kept in internal rotation by means of a sling device. Similarly, following a posterior repair, the patient's arm is held in external rotation with some type of brace device or cast to protect the posterior capsular repair. Since the inferior capsular shift also addresses the inferior capsule, it too must be protected during the healing phase for a minimum of 6 weeks. Conventional braces that can be removed by the patient are a poor choice of immobilization in the initial postoperative management following an inferior capsular shift. My considerable experience suggests that if a brace can be removed, *it will be removed,* especially in the age group that undergoes this type of procedure. Gravity is ever present and removing a brace for dressing, showering, etc., subjects the repair to inferiorly directed forces that place the capsular repair in a precarious situation. Several authors have reported on the importance of cast immobilization following inferior capsular shift.[4,20,59]

This concept of immobilization is neither new nor revolutionary. We have learned this lesson in the initial early return of patients following arthroscopic repairs and thermal procedures. The capsular ligaments and tissues can certainly stretch out in the early weeks following surgical intervention, either open of arthroscopic. Therefore, the capsular repair that involved the management of at least three directions of instability must be immobilized in those same three directions. Removable braces and other forms of removable immobilizers cannot possibly provide the degree and certainty of joint immobility that a well-placed spica cast is capable of providing. Altcheck et al. reported on their series of 40 patients, and although they were not placed in a conventional cast, they were placed in a specially fabricated orthosis for 6 weeks.[1]

The cast is placed in the operating suite immediately following the procedure. By using regional anesthesia, the patient is fully awake and cognitive in the moments following the procedure. Cooperative patients are critical because they must stand erect as the cast is being applied. At all times during the cast application, an assistant is holding the arm in neutral rotation to balance the anterior and posterior capsule while simultaneously holding the arm in a cephalad position. The waist belt portion of the cast is applied and molded around the iliac crests bilaterally. The iliac crests will ultimately bear the downward force of the arm and prevent any forces on the inferior capsular repair (Fig. 14-31). The arm cylinder, which extends from just above the wrist to the upper arm, is then applied. Lastly, the waist piece is fixed to the arm piece with a strut of casting material. While the cast epoxy is curing, the arm is continually held in the position of neutral rotation with superior force. Despite the apparent weight of the cast, which appears enormous to the patient, measurement of the weight following cast removal has determined that it rarely exceeds 2 lb.

Following surgery, regardless of the surgeon's choice of immobilization, significant alterations in activities of daily living become readily apparent. Patients cannot (should not) drive, shower, or dress in pullover shirts, sweaters, and other similar garments. If braces are provided because of "convenience," the patients must be instructed to wear the brace at all times to protect every element of their repair. Again, if the immobilization device is removable, it will be removed! Over the years, I have had more than one occasion when less than discriminating patients took a saw to their cast to remove it.

The cast is worn for 6 weeks and adjusted or trimmed as necessary throughout the 6-week course. Most patients have their cast trimmed or padded the morning following surgery just prior to discharge. Only rarely are other adjustments ever necessary during the subsequent 6 weeks (Fig. 14-32).

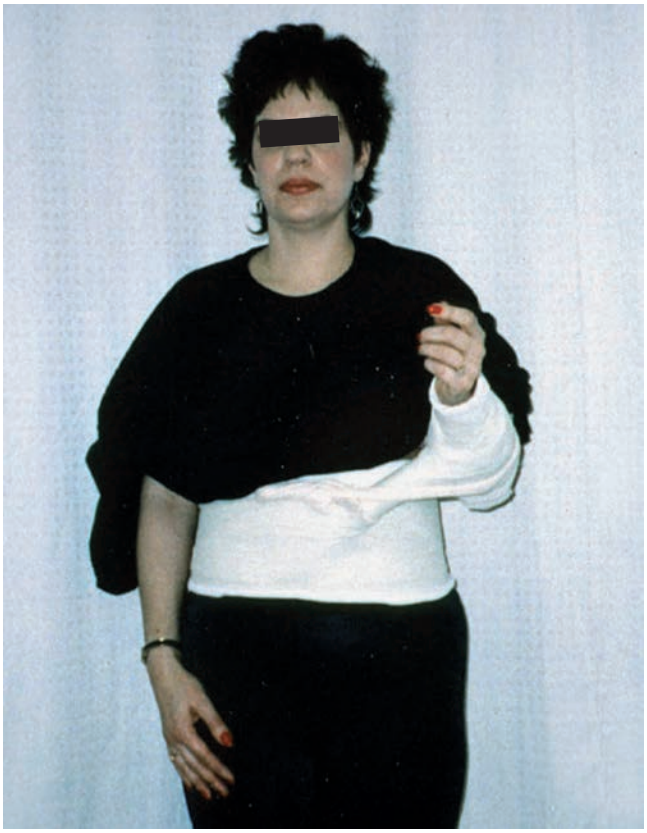


Figure 14-31 Immobilization of the arm is a necessary component of the procedure to allow capsular healing without tension in any direction, especially inferiorly (see text).

When the cast is removed 6 weeks following the surgery, a formal period of rehabilitation begins. The initial several weeks are devoted to recovery of motion through passive and active assisted techniques. Specific limits are given so elevation does not exceed 160 degrees and external rotation does not exceed 60 degrees. These limits are usually attained within 6 weeks of initiation of the program. Once the passive motion has recovered, a structured strengthening program follows. Particular attention is devoted to eccentric strengthening of the internal and external rotator cuff muscles. Shortly after the cuff program is started, further emphasis is given to strengthening of the scapular stabilizing muscles. Patients should be encouraged to remain compliant with their exercise routines for a minimum of 1 year following their surgery.

Return to activities and sport are dependent on recovery of motion, strength, and endurance of shoulder-related muscles. Generally, caution is recommended for those activities that have a higher incidence of injury to the shoulder such as downhill (alpine) skiing, water skiing, gymnastics, and certain types of swimming. Also, weight lifting routines that place excessive mechanical forces on the glenohumeral joint such as bench press and pushups should be avoided.

Figure 14-32 A well-motivated patient can learn to adapt to the "challenges" of a cast for 6 weeks. The outcomes are likely worth the misery.

RESULTS OF OPEN SURGICAL MANAGEMENT

The first series of results reported for surgical management of multidirectional instability were those of Neer and Foster following the inferior capsular shift procedure.[63] The original series included 32 patients, and there was only one patient who was considered to have an unsatisfactory result related to an episode of subluxation 7 months following the procedure. However, only 17 patients were followed for longer than 2 years, and eight patients in the series were evaluated less than 12 months following their reconstruction. In his book published a decade later, Neer reported that more than 100 additional patients underwent the inferior capsular shift with similar results.[65]

In a much longer follow-up series, Pollock et al. reviewed 52 shoulders in 49 patients at an average of 5 years following inferior capsular shift, but some patients were seen 11 years following surgery.[74] In their series, there were 94% excellent or good results and only two patients had a poor result over this relatively longer follow-up time frame. In terms of stability, 94% of patients were stable at an average of 5 years following their surgical reconstruction.[74]

In another relatively longer follow-up analysis, van Tankeren and coworkers studied 17 patients following an inferior capsular shift for multidirectional instability at an average of 3 years following their surgery. Using the Constant and Rowe scores, 14 of the 17 patients had an excellent outcome, and there was only one poor result.[85]

Other authors confined their postoperative evaluation to the athletic age group. Bak et al. reviewed 26 shoulders in 25 athletes who underwent an inferior capsular shift for multidirectional instability.[5] At an average of just short of

5 years, 84% of the athletes returned to their preinjury level of activity within 5 months of surgery. More importantly, with respect to athletes who required the use of their involved arm for overhead throwing activities, 76% returned to their preoperative level of sport.[5] Choi and Ogilvie-Harris noted similar results in their series of athletes who underwent an inferior capsular shift.[18]

Altcheck et al. reviewed a series of 40 patients who had both an inferior capsular shift and a Bankart repair for a labral injury.[1] At average of 3 years follow-up, 95% rated their result as excellent, although 10% had a recurrent episode of instability within the follow-up period. The authors emphasized that a patient with a Bankart lesion and multidirectional instability may have a better prognosis than one without a Bankart lesion. They suggested that in the presence of a Bankart lesion, the multidirectional component of the instability had a traumatic origin and therefore may have a better prognosis.

Cooper and Brems[20] reported on the results of 38 patients with an average follow-up of 39 months with a minimum of 24 months following an open inferior capsular shift procedure. Eighty-six percent of patients were improved; however, 10% continued to have clinical instability. In their series, despite early satisfactory results, six patients (15%) felt their shoulders had deteriorated over time.

Lebar and Alexander reviewed a series of 10 patients at an average of 28 months following an inferior capsular shift.[50] In this small series from the Oakland Naval hospital, all 10 patients felt they were improved, yet one had recurrent symptoms enough to require a revision procedure.

Care must be taken when trying to interpret results of these few series because follow-up intervals are short. Several authors have noted that meaningful results demand much longer follow-up time frames.[45,60,68,76] Shoulder instability may recur many years following the repair. Furthermore, patients in the age group who typically undergo this type of surgery are very active and subject to recurrent trauma through their activity. Hawkins et al. reported a failure rate of 39% (12 of 31 patients) at a longer follow-up of 2 to 5 years.[34] Because of the concern of failure at longer follow-up, some surgeons advocated additional or secondary procedures.[9,29,66,70,89-91]

Bigliani and coworkers reviewed a series of 52 patients following an inferior capsular shift with a minimum follow-up of 5 years. They reported a satisfaction rate of 94% in nonathletic patients.[11] In a more recent study, Bigliani et al. reviewed the outcome of 75 athletically active patients who underwent an inferior capsular shift.[12] Eighty-nine percent were able to return to their major sport, while 73% remained at their anticipated level of competitiveness.

In the series with the longest follow-up, Hamada and colleagues reviewed the outcome of 26 patients (34 shoulders) who underwent an inferior capsular shift for multidirectional instability with a mean follow-up of 8.3 years.[28] They reported that 85% had satisfactory outcomes and 59% were rated as good and excellent on the Rowe rating scale. In this study, the authors were able to re-review 19 of their patients 3.5 years and 8.3 years following their surgery. Remarkably, they reported no change in the Rowe rating scale in any of these patients over the 8-year period.

Several reports have documented high success rates for arthroscopic capsular shift in the treatment of MDI. Duncan and Savoie reported preliminary results of arthroscopic capsular shift in 10 patients for patients with MDI with follow-up from 1 to 3 years.[58] No patients in the study developed recurrent instability. Four athletes returned to sports participation, although no information regarding their preoperative level of activity was reported. Two patients developed pain postoperatively over the posterior suture knot that required removal. All 10 patients had a satisfactory rating score according to the Neer criteria. Treacy and Savoie reported on 25 patients who underwent arthroscopic capsular shift for MDI with an average follow-up of 5 years.[31] Three patients had episodes of instability after the procedure but none had repeat episodes of dislocation. Eighty-eight percent of patients had satisfactory results according to the Neer system.

Tauro and Carter reported preliminary results of a modified arthroscopic capsular shift for anterior and anteroinferior instability in four patients with a minimum follow-up of 6 months.[82] No patients developed recurrent instability, although follow-up is short term.

Gartsman reported on 47 patients who underwent arthroscopic capsular plication for MDI.[43] The average follow-up was 35 months and 94% had good to excellent results. Eighty-five percent of athletes retuned to their desired level of sports participation.

McIntyre reported results of arthroscopic capsular in patients with MDI using a multiple suture technique in the anterior and posterior capsule with follow-up of 32 months.[42] Recurrent instability occurred in one patient (5%) who was treated successfully with a repeat arthroscopic stabilization. Thirteen athletes (93%) returned to their previous level of performance.

Wichman and Snyder reported results of arthroscopic capsular shift for MDI in 24 patients with an average age of 26 and a minimum follow-up of 2 years.[72] Five patients (21%) had an unsatisfactory rating using the Neer criteria. Three of these patients were involved in worker's compensation cases that were not yet resolved, and an additional patient was involved in litigation over a motor vehicle accident.

Lyons et al. reported on laser-assisted capsulorrhaphy for MDI in 26 patients (27 shoulders) with a minimum follow-up of 2 years.[77] Twenty-six of the 27 shoulders remained stable and 86% returned to their previous level of sports participation.

McIntyre et al. reviewed the results of 20 consecutive shoulders in 19 patients treated with arthroscopic capsular shift for posterior instability at an average of 31 months postoperatively.[79] They had 15 excellent, two good, one

fair, and three poor results. They reported two recurrent dislocations and three subluxations for an overall recurrence rate of 25%.

Williams et al. reported on a series of 26 patients (27 shoulders) treated with arthroscopic repair for traumatic posterior instability (posterior Bankart lesion) with an average follow-up of 5 years.[84] Symptoms of pain and instability were eliminated in 92% of patients. Two patients required additional surgery.

Wolf reported on a series of 14 patients with recurrent posterior instability treated with arthroscopic capsular plication.[10] Follow-up averaged 33 months and 12 patients reported excellent results. There was one recurrence of instability that was remedied by a second operation. All 14 patients were satisfied with their procedure. Hovis et al. reported on a series of six elite golfers with posterior instability who underwent posterior thermal capsulorrhaphy.[88] At an average of 4.5 years follow-up all six had returned to their previous level of play. Antoniou presented results on 41 patients undergoing arthroscopic capsulolabral augmentation for posteroinferior instability.[86] Patients were followed for an average of 28 months and 35 (85%) of patients had improved stability of the shoulder.

Jorgensen et al. followed 41 patients with posterior instability that were treated with either open or arthroscopic technique.[6] Patients were evaluated for an average of 36 months and no significant difference was seen in outcome between the two groups. The group treated with the open procedure had a longer hospitalization, slight decrease in external rotation, and more frequent cosmetic complaints.

These multiple reports document the efficacy of arthroscopic management of MDI. The ability to see within the joint allows an extremely accurate assessment of the pathology and thereby improves the results of operative treatment. Current techniques utilizing multiple capsular plication techniques with various sutures hold great promise in increasing the success in the management of symptomatic MDI. Progress in rehabilitation holds much promise to help both operative and nonoperative patients.

SUMMARY

Although it has been more than two decades since multidirectional instability became recognized as a distinct clinical entity, our understanding of the condition remains incomplete. Despite research efforts on multiple fronts, the instability remains incompletely defined and characterized. Perhaps there is not one unified pathologic process or concept that fully explains the multiple characteristics of the condition. Research efforts continue to explore the many seemingly disparate causes including biologic, biomechanical, biochemical, neurologic, and genetic in an effort to define this unique instability pattern. Because it is likely that each of these issues contributes in some way to the clinical manifestations of multidirectional instability, continued efforts to expand our knowledge and understanding remains an obligation to both our profession and to our patients.

Treatment options likewise remain confusing and long-term outcomes disappointing in this population of patients who are predictably young, active, and challenging. What does seem clear is that a prolonged trial of nonoperative management is appropriate. Relatively high success rates have been achieved when a well-executed rehabilitation program has been dutifully executed by a well-motivated patient. What remains less clear is the management of the patient who has been refractory to a well-performed trial of nonoperative management. Both open and arthroscopic procedures have been reviewed and outcome studies have been published by talented surgeons in both camps. Nevertheless, the orthopaedic community still lacks the necessary long-term outcomes of a procedure performed on a cohort of patients who predictably live 50 years following their treatment.

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Chronic Dislocations

15

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INTRODUCTION

Dislocations of the shoulder account for approximately 45% of the dislocations seen in the major joints.[20] In 1974, Cave et al.[5] presented the demographics of a large series of shoulder girdle dislocations. In their series, anterior glenohumeral dislocations accounted for 84% of shoulder girdle dislocations, acromioclavicular dislocations for 12%, sternoclavicular dislocations for 2.5%, and posterior glenohumeral dislocations for 1.5%. Although these injuries are common and are usually diagnosed by careful physical examination and appropriate radiographic examinations, they are occasionally missed.

In 1968, Schulz and colleagues reported on 61 chronic shoulder dislocations.[36] In their review of both anterior and posterior shoulder dislocations, they found the following: (a) 50% had associated fractures about the joint, (b) 33% had neurologic injury (5 of the 17 posterior dislocations and 18 of the 44 anterior dislocations), and (c) 28% of the dislocations were posterior. They also found that seizures were the cause of 29% of these dislocations and that 11 of the 18 dislocations caused by seizures had associated fractures. In addition, 50% of seizure-related injuries were posterior dislocations.

In 1982, Rowe and Zarins performed a survey of 208 New England orthopedists to determine how often chronic dislocations were seen by the average orthopedist.[35] Their survey found that 50% of orthopedic surgeons in practice for 5 to 10 years had seen a chronic dislocation, 70% of those in practice for 10 to 20 years had seen this injury, and 90% of orthopedic surgeons in practice for over 20 years had seen at least one chronic dislocation. Based on their survey, 65% of chronic dislocations were anterior and 35% were posterior. Rowe and Zarins also presented 23 patients with 24 dislocations: 14 were posterior, eight were anterior, one was inferior, and one was superior. Remarkably, 79% of the posterior dislocations that they evaluated were missed by the original treating physician.

A more recent study presented by Checchia et al.[6] reviewed 66 patients with a total of 73 posterior fracture dislocations of the shoulder treated at Santa Casa Hospital in Sao Paulo, Brazil. The causative facture was a convulsive episode in 37 patients. Sixteen shoulders had an associated fracture of the proximal humerus. Thirty-six patients with 42 locked dislocations were considered to have chronic

injuries. Over 50% of the chronic cases had been misdiagnosed as contusions at the time of the original injury.

The true incidence of chronic dislocations of the shoulder is unknown. One reason for this is the lack of consistent criteria for defining what time frame should be used to declare a dislocation to be chronic. In the literature, the definition of a chronic dislocation has ranged from as early as 24 hours to as late as 6 months.[6,8,14,15,35,36] Schulz and colleagues[36] used 24 hours as their definition of chronic. Since the treatment of these dislocations is likely to be the same both before and after 24 hours, a longer time point seems more logical. Checchia et al.[6] used 4 weeks and Rowe and Zarins[35] used 3 weeks as the cutoff between acute and chronic dislocations. In this chapter, 3 weeks will be used as the definition of chronic dislocation. The term "locked dislocation" has also been used to describe these injuries, presumably because of the difficulty in reducing them.[15] The term "locked dislocation" may apply to both acute and chronic injuries. The time from injury is an important factor in determining treatment of locked dislocations.

The purpose of this chapter is to review the diagnosis, treatment, and the results of treatment of locked dislocations of the glenohumeral joint. An algorithmic approach is presented based on the time from injury as well as the pathologic findings. This treatment algorithm can be used effectively to treat acute as well as chronic injuries. As mentioned above, for the purposes of this chapter, the term *chronic* is used to describe any dislocation that has been present for at least 3 weeks. This time frame is chosen not only because there is historical precedent for it, but also because of its implication in treatment. If the injury is less than 3 weeks old, a closed reduction with or without open treatment of associated fractures may be effective, and if the injury is more than 3 weeks old, this option is unlikely to be successful. The time from injury also affects the structural quality of the articular cartilage and its underlying bone. If the injury is greater than 6 months old, it is unlikely that the articular cartilage surfaces of the glenohumeral joint will be viable and the bone quality of the humeral head will be of sufficient quality to allow for salvage of the native joint. Therefore, the treatment of chronic locked dislocations will be variable, depending on, among other things, the time from dislocation.

The outcomes of specific types of locked dislocations are discussed later in this chapter. However, in general, the number of series reported in the literature is small and the number of patients in each report ranges from a single case report to 61 patients.[6,8,11–15,19,26,32,35–42] Because of the small numbers in each series or groups of patients within a single series, evaluation of outcomes in specific groups is difficult. In addition, there is a lack of consistency with regard to outcome measurement tools among series, making comparison of results among series difficult. Despite these limitations, the treatment algorithm outlined in this chapter is based on analysis of the literature as well as personal experience of the senior authors.

POSTERIOR LOCKED DISLOCATION

Although posterior dislocations account for only 1.5% of shoulder dislocations, they account for a much higher relative percentage of locked dislocations of the glenohumeral joint. This is due to the fact that these injuries are more easily missed than anterior dislocations. In fact, Rowe and Zarins[35] reported that 11 of the 14 locked posterior dislocations that they evaluated, in a referral practice, were unrecognized by the initial treating physician. In Checchia et al.[6] series of locked posterior dislocations, 22 of 36 chronic lesions were missed at initial presentation. It is estimated that more than 60% of posterior dislocations of the glenohumeral humeral joint are missed at the time of initial evaluation.[6,14,15,35]

There are several potential reasons that these injuries may be missed on initial evaluation. Most posterior dislocations of the shoulder occur from either seizures or violent trauma. These patients may present to the emergency department with more urgent medical problems. Additionally, the outward appearance of a posteriorly dislocated shoulder may be subtle. In posterior dislocations the contour of the shoulder may appear normal, particularly if the shoulder is large, and the injured arm is locked in a position of internal rotation with the elbow at the side. To suspect a posterior dislocation, one must attempt to passively rotate the humerus to identify the characteristic loss of external rotation. Finally, the radiographic findings of a posterior dislocation on standard anteroposterior radiographs are extremely subtle and often misinterpreted as being normal (Fig. 15-1).

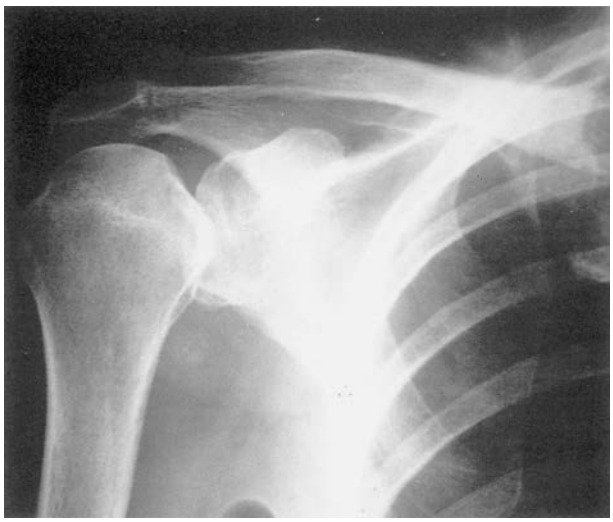


Figure 15-1 An almost normal-appearing anteroposterior radiograph of the shoulder in a patient with a chronic posterior dislocation.

History

Patients presenting with chronic posterior dislocations of the shoulder usually give a history of major trauma or a history of a convulsive episode from either an intrinsic seizure disorder or an electrical shock. They initially had substantial pain in the shoulder with a loss of use of the arm. The initial shoulder discomfort often resolves to a tolerable level within a few weeks of the injury and the patient may begin to use the shoulder for waist-level activities. Many times, this slow resolution of pain and the return of limited function are mistaken for the process of recovery by the patient or the treating physician. In fact, there are multiple reports of patients with locked posterior dislocations being sent to therapy with the diagnosis of a frozen shoulder, only to later be given the correct diagnosis once treatment fails.[6,15,35,36]

One group of patients to be especially wary of are those with alcohol or drug dependence. Often these patients present days to weeks after a traumatic episode with pain and inability to use their arm. They will be unable to give a clear history of the event and may not have sought initial treatment. The examining physician should have an extremely high index of suspicion for posterior dislocation, especially if external rotation is lacking. The presence of alcohol and drug dependence confounds not only the diagnosis, but also the treatment of the locked posterior dislocation, as patient compliance may not be adequate for some treatment options.

It is important to obtain a thorough past medical history to determine if there are any important medical problems that would influence the choice of treatment. These patients may surprisingly adapt sufficiently to the limitations of motion and to the discomfort produced by this injury. This allows the surgeon to forgo surgical management in elderly, medically infirm patients or in patients that are at risk due to compliance issues. It is important to obtain a thorough past medical history to determine if there are any important medical problems that would influence the choice of treatment.

Physical Examination

The patient should be examined with the entire shoulder exposed to allow adequate visual inspection. The contour of the shoulders is best visualized by standing behind and slightly superior to the patient. The dislocated shoulder will appear to have less fullness anteriorly. While standing behind the patient, the examiner should palpate both shoulders, noting any asymmetry between the anterior deltoid and the coracoid processes. If the shoulder is dislocated posteriorly, the coracoid of the involved shoulder may be more prominent, and there will be a loss of the normal anterior contour of the shoulder. These findings can be subtle in patients with bulky shoulders.

Loss of external rotation beyond neutral is almost pathognomonic of a locked posterior dislocation of the shoulder. In fact, in any patient with less than 0 degrees of passive external rotation following a shoulder injury, a posterior dislocation is assumed present until proven otherwise. Limitation of glenohumeral abduction to less than 60 degrees is also usually present (Fig. 15-2). With long-standing dislocations (6 months to a year) in which the humeral head is locked onto the glenoid, the windshield wiper effect of the glenoid against the humeral head during motion can create a large enough defect to permit surprisingly functional motion. The physician should also note the level of discomfort experienced by the patient while moving the extremity in his or her adapted range of motion. This information will be important when determining the treatment plan.

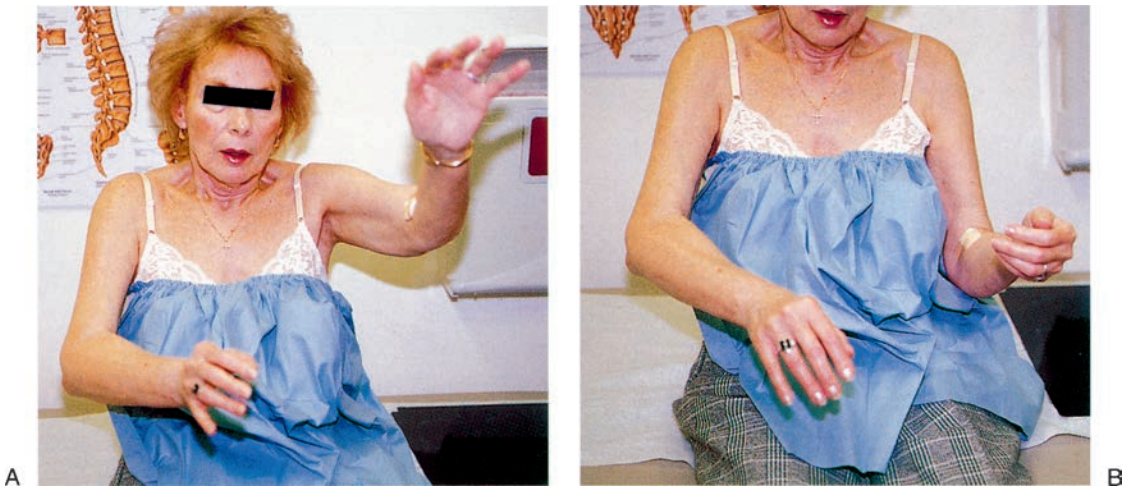


Figure 15-2 Clinical photographs of a patient with a locked posterior dislocation of the shoulder: **(A)** loss of forward elevation; **(B)** loss of external rotation.

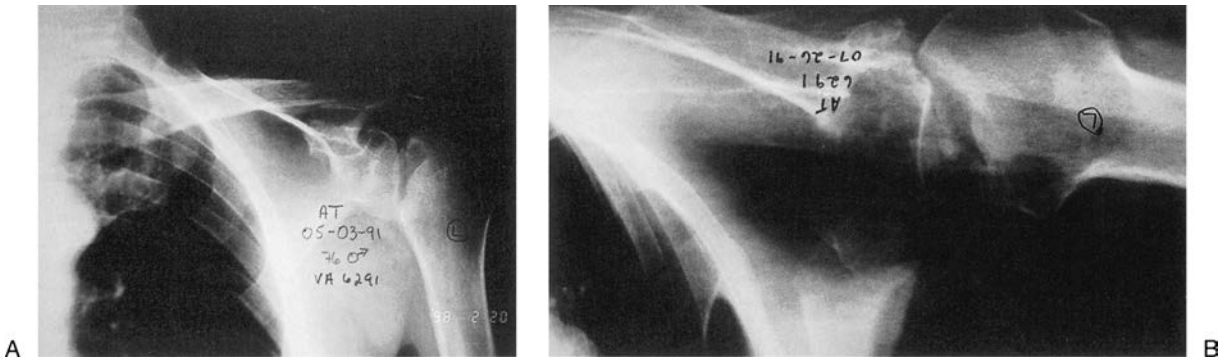


Figure 15-3 **(A)** An anteroposterior radiograph showing a posterior dislocation of the humeral head. **(B)** The axillary lateral radiograph of the same patient confirming the direction of the dislocation.

In cadaver studies, posterior dislocations of the shoulder have been associated with disruptions of the rotator cuff.[30] Gerber stated that he has seen three such lesions in the patients on whom he has operated.[12] Functional testing of the rotator cuff muscles with the shoulder posteriorly dislocated is not reliable. Therefore, the muscles of the rotator cuff should be examined for any evidence of increased atrophy compared to the deltoid. If there is any suspicion of a rotator cuff tear, it should be confirmed with appropriate imaging studies such as a magnetic resonance imaging (MRI) scan. A thorough examination of the remainder of the extremity must be done to rule out associated neurovascular injuries.[10,29] Schulz et al. reported that 5 of the 17 posterior dislocations that they evaluated had an associated nerve injury.[36]

Radiographic Evaluation

Plain radiographs will confirm the clinical diagnosis of a posterior dislocation of the shoulder only if the appropriate views are obtained.[31] Hawkins and colleagues showed that in 50% of the cases that they evaluated, the diagnosis could not be made with certainty based solely on an anteroposterior radiograph taken with the beam directed at 90 degrees to the plane of the scapula.[15] However, the diagnoses could be made in all patients when an axillary lateral radiograph was obtained. It is therefore imperative that any injury of the shoulder have, at a minimum, an anteroposterior and axillary lateral radiograph taken of the shoulder (Fig. 15-3A, B). If for any reason a patient with suspected posterior dislocation cannot obtain an axillary radiograph, a computed tomographic (CT) scan or MRI scan is necessary.

These views will permit evaluation of the glenohumeral joint and are essential for preoperative planning. The axillary view will often show the extent of glenoid wear and the amount of humeral head impaction that invariably is present with the locked posterior dislocation (see Fig. 15-3B). These radiographs should also be evaluated for evidence of associated fractures of the humerus and glenoid. In the series reported by Schulz et al.,[36] Hawkins et al.,[15] and Wilson and McKeever,[40] 50% of posterior dislocations had an associated fracture of the humeral neck or tuberosities. The presence of these injuries will influence the surgeon's choice of treatment. In the patient with a more chronic dislocation, these fractures may have developed into a malunion or a nonunion, and this will add further challenges to successful treatment (Fig. 15-4).

The axillary lateral radiograph is important for preoperative evaluation of the impaction fracture of the humeral head. By dividing the arc of the impacted surface of the humeral head and comparing it with the length of the arc created by the articular surface of the humeral head in its preinjury state, the surgeon can estimate the percentage of the humeral head involved in the impacted segment (Fig. 15-5). If there is difficulty in estimating the size of the humeral head defect, glenoid deficiency or fracture is suspected, or if the area of impaction is measured to be greater

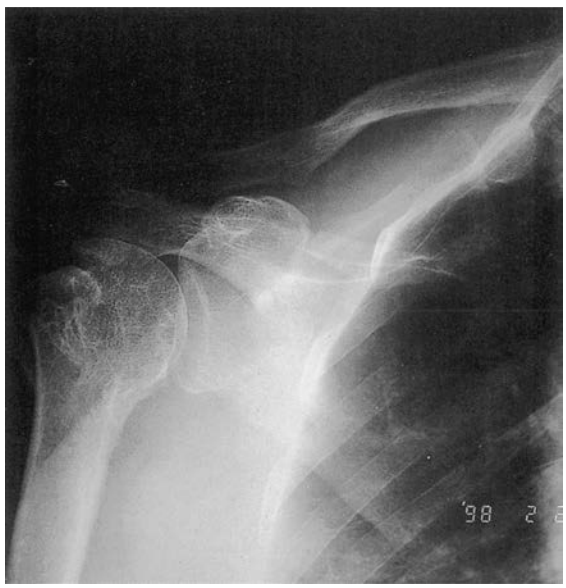


Figure 15-4 An anteroposterior radiograph of a shoulder that is posteriorly dislocated. A malunion of the tuberosity can be seen.

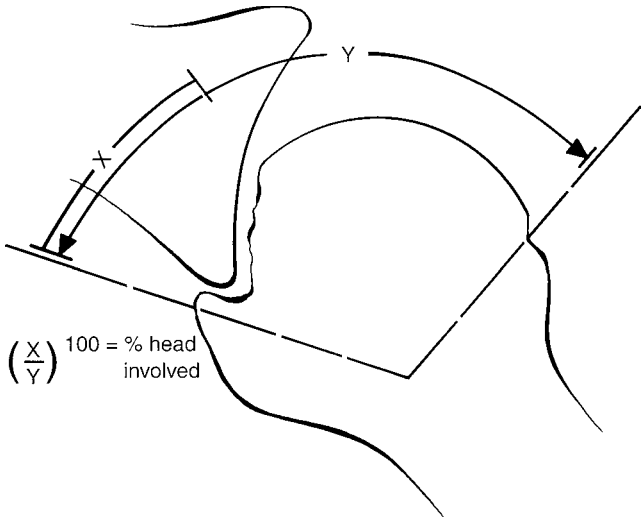


Figure 15-5 The percentage of the head involved can be determined by measuring the arc of the area of impaction and dividing by the arc of the intact humeral head. This number is then multiplied by 100 to give the percentage of the head involved.

than 20% of the humeral head surface, then a CT scan should be obtained. The CT scan is used to evaluate the amount of humeral head and glenoid erosion[22,27] (Fig. 15-6), the status of any fractures, and the quality of the remaining bone stock of the humeral head.[12] Changes in the appearance of the humeral head, such as thinning of the subchondral bone and loss of the normal trabecular architecture, indicate the presence of osteopenia. This information will prepare the surgeon for the possibility of the shoulder requiring replacement.

Many patients are referred to our office having already undergone an MRI study. If the study is adequate to determine the size of the humeral head defect, the presence of a glenoid fracture, and the amount of any glenoid deficiency, a CT scan is not ordered (Fig. 15-7). An electromyogram (EMG) should be obtained if there is any evidence of nerve injury. An arteriogram is needed if there is any evidence of vascular compromise.

Treatment Choice and Preoperative Planning

The first step to choosing the proper treatment of these patients is to have an understanding of the patients' perceived disability, their general health, their ability to undergo a surgical procedure, and their ability to understand and comply with the postoperative rehabilitation. In general, if the patient has low demands on the shoulder, has adapted his or her activities to cope with the limitations of the shoulder, or has pain that is easily controlled, then he or she should be considered for conservative treatment. Patients with the inability to follow the directions of rehabilitation and patients who are considered a high risk for surgery should also be considered for nonoperative treatment. Under these circumstances, it is imperative that the patient understand the rationale and expected limited outcome goals of nonoperative management.

Once surgical treatment has been established, the specific method of operative management must be chosen. This is determined by the patient's age, the time from the

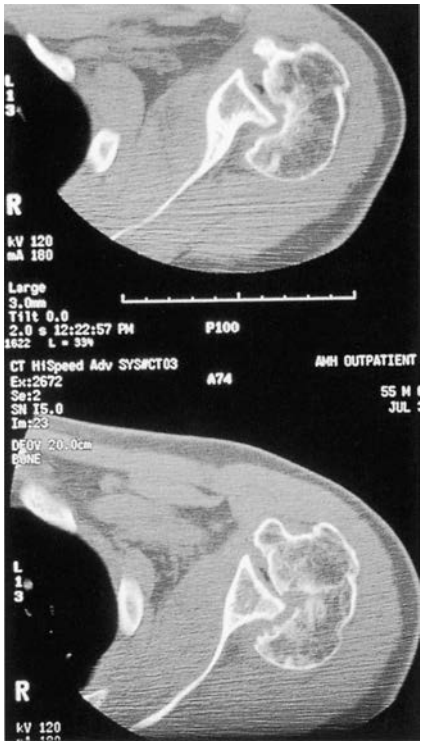


Figure 15-6 Computed tomography can provide valuable information. Here, it shows a posterior fracture–dislocation, with evidence of glenoid wear and osteopenia.

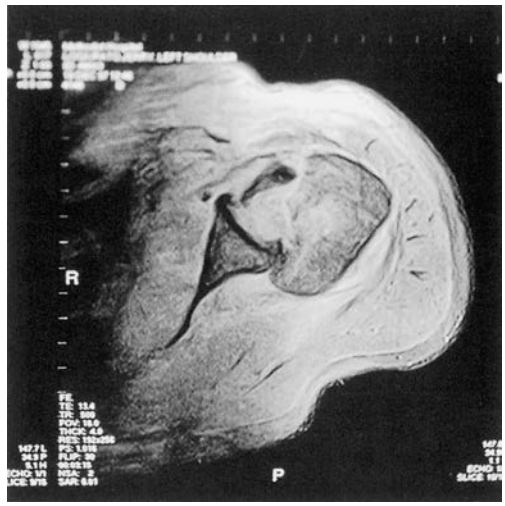


Figure 15-7 A magnetic resonance imaging scan obtained before referral can also be used to evaluate the humeral head defect and glenoid. An axial cut is shown. We prefer computed tomography scan, owing to the better visualization of the bony architecture.

initial injury, the demands on the shoulder, and the available bone stock present in the shoulder. Here, bone stock refers not only to the amount of bone loss because of the injury, but also to the intrinsic quality of the bone. The duration of the dislocation greatly influences the choice of treatment. Generally, the more chronic the injury is, the more likely there will be disuse osteopenia, loss of the articular surface's structural integrity, or both. Hawkins et al.,[15] Rowe and Zarins,[35] and Checchia et al.[6] suggest that any injury of longer than 6 months' duration may have irreversible damage to the articular surfaces. If significant osteopenia or loss of articular surface viability are suspected, the surgeon must be prepared to perform an arthroplasty. Patients with long-standing dislocations are also more likely to have erosion of the glenoid rim, requiring reconstruction and prosthetic arthroplasty. A preoperative CT scan will help determine whether this will be needed.

If the injury is relatively fresh (defined as less than 3 weeks), there is a possibility that the defect in the humeral head can be disimpacted and bone grafted. In this circumstance, the patient must give consent for the possibility of a bone graft and should be positioned and draped appropriately at the time of the procedure.

With the foregoing preoperative evaluation, the patient can be placed into a "likely" treatment group. The surgeon, however, must be prepared to move along the treatment algorithm (Fig. 15-8). The goals are to have a general treatment plan before surgery, an understanding of which choices are available, and the knowledge required to make the correct intraoperative surgical decision.

For the purposes of preoperative evaluation, we will use 3 weeks or greater to define a chronic injury. Three weeks is chosen because after this time it becomes difficult to perform a successful closed reduction.

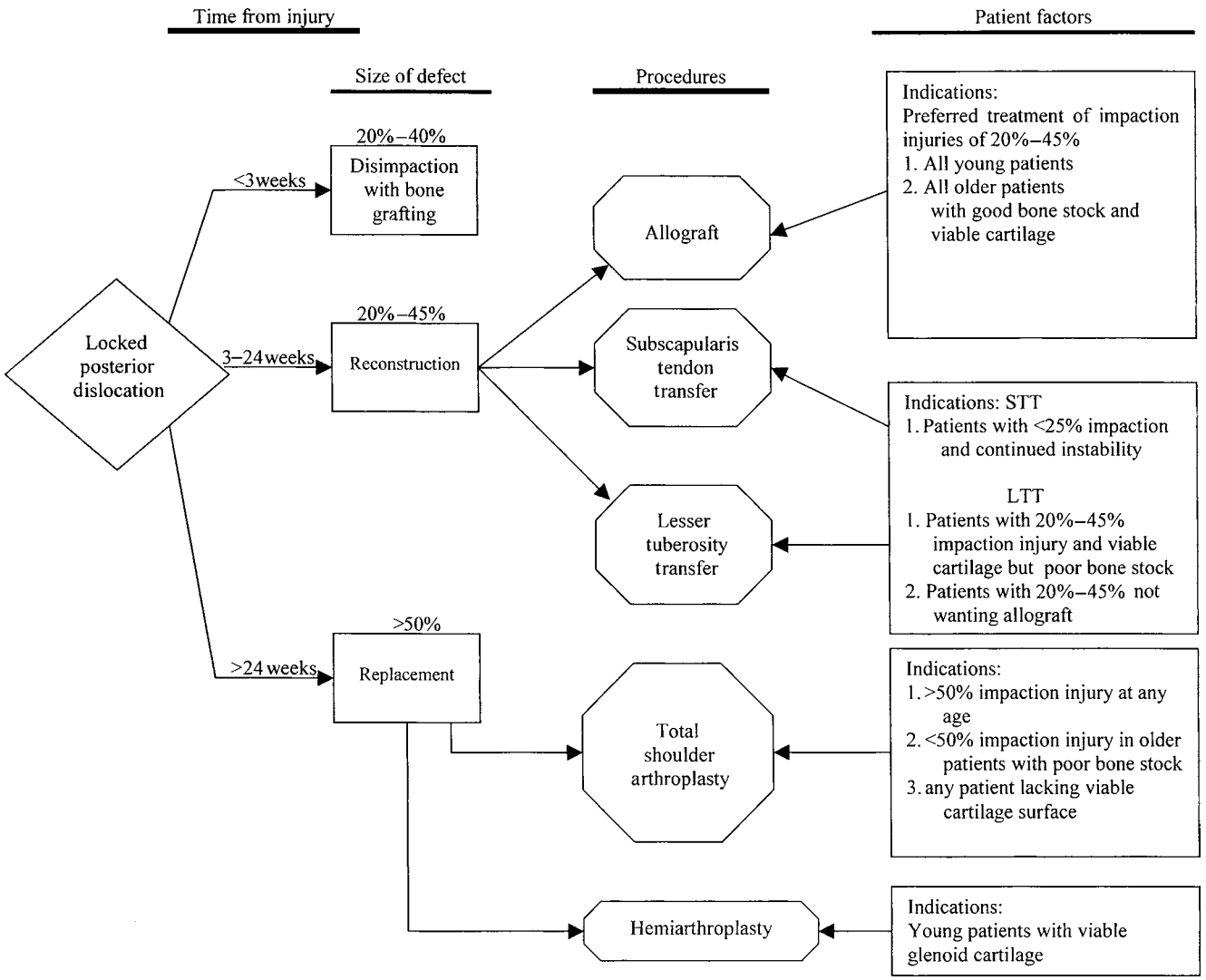


Figure 15-8 The treatment algorithm giving our approach to the surgical treatment of locked posterior dislocations. LTT = lesser tuberosity transfer; STT = subscapularis tendon transfer.

Treatment Options

Nonoperative Treatment

Nonoperative treatment includes the skillful neglect of chronic injuries of longer than 3 weeks' duration. Patients with acute injuries, with rare exceptions, are treated operatively. For patients with chronic dislocations who have minimal pain and do not perceive their limited motion as disabling, or for patients who are at high risk for surgery or have mental illnesses, skillful neglect is an acceptable treatment option. These patients are best treated with supportive measures to control pain and are encouraged to use their arm for daily activities as the pain subsides.

Closed Reduction

The criteria for choosing a patient who will be suitable for an attempt at closed reduction are the following: The patient can be of any age, the injury should be less than 3 weeks old, the impaction injury to the humeral head should be less than 25%, and the humerus must have no other fractures present. If these criteria are met, the patient may undergo an attempt at closed reduction.

The closed reduction is best performed under scalene block or general anesthesia.[7] The patient is placed supine and gentle traction is applied to the humerus in line with the long axis of the body. Gentle adduction and flexion are then applied. The humerus is gently externally rotated, and pressure is applied to the humeral head in a posterior to anterior direction. With this technique, the shoulder should reduce.

Once the joint is reduced, the humerus should be internally rotated and the point of instability noted. This position is the "danger zone" for dislocation. Patients who have no instability with the humerus internally rotated enough to place the hand on the abdomen are considered to have a stable injury. This is unlikely to occur with a chronic posterior dislocation, but may occur for acute injuries. If the shoulder is stable with the arm in internal rotation, the patient is immobilized in an orthosis. The humerus is held at the side in neutral rotation with the shoulder extended a few degrees.

Rehabilitation of the shoulder is begun on day 2 after the reduction. The patient is allowed unlimited external rotation and is allowed to perform isometric strengthening of the shoulder girdle. The brace is removed after 4 weeks, and the patient is encouraged to use the shoulder as tolerated. The patient is not allowed to bring the arm behind the trunk (maximum internal rotation) for 6 weeks. Lifting more than 5 lb above the plane of the chest is discouraged for 6 weeks. Heavy labor is discouraged for 3 months.

If after closed reduction the shoulder remains unstable and dislocates with internal rotation at a point before the hand reaches the abdomen, the patient should be placed in a prefabricated splint. The shoulder should be slightly extended and the humerus should be externally rotated 20 degrees from neutral. The patient is then further evaluated to determine why the shoulder continues to be unstable. A CT scan should be obtained to evaluate the glenoid for fracture and to reassess the size of the humeral head defect.[23] If a CT scan was obtained before closed reduction and the cause for instability after reduction can be determined, open surgical intervention is performed at that time.

If a fracture of the glenoid is present but well reduced with the arm in the splint, the patient continues to be treated conservatively. Weekly follow-up radiographic evaluation is required until early fracture union occurs. This usually takes 6 weeks. During this period of immobilization, the patient is allowed to externally rotate the humerus passively, and after 3 weeks is allowed to internally rotate to within 20 degrees of the danger zone, as determined at the time of reduction. At 6 weeks, the patient is allowed full passive range of motion and is allowed to progress with active motion as pain permits. Strengthening is not started until 10 to 12 weeks.

If the glenoid fracture is displaced or unstable, the patient will require an open reduction with internal fixation of the glenoid as described in Chapter 26. If the humeral head impaction fracture is the cause of continued instability and involves less than 45% of the articular surface, then the patient will need to undergo open repair of the humeral head defect.

If there are fractures present of the tuberosities or humeral neck, the reduction should be performed under general anesthesia. Because there is a risk of displacing these fractures during closed reduction, the humeral neck or tuberosity fractures should undergo percutaneous pin fixation before any attempt at reduction[16] or an open reduction and internal fixation should be performed (Chapter 28).

If closed reduction is successful, a good outcome is to be expected. Hawkins et al.[15] reported excellent results in the three patients whose shoulders could be reduced and were stable after reduction. Schulz et al.[36] reported satisfactory results in the three posterior dislocations that they were able to reduce. All six of these shoulders were reduced within 4 weeks of injury. Checchia et al.[6] reported that the nine patients (out of 10) available for follow-up that were treated with closed reduction and splinting had excellent results with a mean University of California, Los Angeles (UCLA) score of 34.9.

Open Reduction

Open reduction of locked posterior dislocations will be required if the injury is more than 3 weeks old, if the shoulder is unstable after closed reduction, if the impacted area of the humeral head involves greater than 25% of the

articular surface, or if there is a displaced fracture of the surgical neck or tuberosities. The best treatment of these injuries will be determined by the following factors: (a) the size of the humeral head defect, (b) the amount of time from injury, (c) the patient's age, (d) the amount of posttraumatic arthritis present, and (e) the quality of the humeral head bone stock. Most articles addressing the treatment of locked posterior dislocations have used the size of the humeral head impaction injury to determine the approach to treatment.[6,8,12–15,26,28,35,36] The other factors listed above are also important in determining the final choice of treatment.

Note that these injuries are uncommon, and the number of reported patients treated with each method is small; therefore, it is impossible to draw any statistical conclusions from the data presented in the literature regarding the treatment of these injuries. The surgeon should choose the method with which he or she is most comfortable.

Disimpaction and Bone Grafting

This method of treatment is best reserved for an acute injury (less than 3 weeks old) with a humeral head defect that involves less than 45% of the articular surface. The patient must have adequate bone stock to provide for secure fixation and must have structurally intact articular cartilage to be considered for this procedure. It is ideal for use in the younger patient, but may also be effectively used in older patients with good bone stock. The patient is positioned and the ipsilateral iliac crest is prepared and draped, so that bone graft can be obtained if needed.

The standard deltopectoral approach is used. The axillary nerve is always identified and protected. The ascending branch of the anterior humeral circumflex artery is identified and protected in procedures where the humeral head will be preserved. The subscapularis tendon is incised 1 cm from its insertion and dissected from the underlying capsule. This may be difficult, owing to the scarring caused by the injury. A vertical capsulotomy is then performed. It is important to maintain and repair the coracohumeral and superior glenohumeral ligament at their attachment sites as these structures provide posterior stability. The joint is débrided of any scar tissue. Once the articular surfaces are identified, an evaluation of the injury is performed. The glenoid is inspected for fracture or articular injury. The humeral head is reduced by placing a Cobb elevator into the site of impaction and using it to lever the head over the glenoid rim. In cases of long-standing dislocation (6 months), reduction of the head may not be possible. Alternatively, a laminar spreader (with a padded edge) placed on the glenoid rim and the other side placed on the proximal humerus can disengage the locked posterior dislocation and allow easy and gentle reduction. The posterior labrum and capsule are inspected. In the majority of shoulders with posterior dislocations, there is an avulsion of the capsule as a sleeve of tissue. Unlike the Bankart lesion seen with anterior dislocations, the injury to the posterior labrum and capsule does not always need to be reattached to the glenoid.[12,13,15] Usually, it is only necessary to roughen the posterior aspect of the glenoid with a curved curette, with the expectation that the capsule will heal to its origin. The impacted region of the humeral head and the remaining intact humeral head are inspected. A determination is made as to whether the cartilage surface at the site of impaction can be salvaged. If the articular surface can be salvaged, then disimpaction with bone grafting is performed as follows (Fig. 15-9).

Disimpaction of the articular cartilage with its underlying subchondral bone is performed by creating a bone window in the greater tuberosity directly opposite the impacted area (Fig. 15-9A). This will require internal rotation of the humerus to create the window and to disimpact the fracture through it. The surgeon will have to externally rotate the humerus to visualize the progression of the disimpaction of the articular defect. Once the window is created, a bone tamp or the blunt end of a 10-mm drill is placed into the window and impacted toward the defect opposite it (Fig. 15-9B). This will disimpact the humeral head defect in its central portion. Once the articular surface's contour is properly restored centrally, a small curved osteotome placed into the bone tunnel can be used to lever the edges of the defect. After the area is disimpacted, a void will have been created below the area of the fracture. This void is filled by packing the area with cancellous bone. The bone tamp is used to pack the defect (Fig. 15-9C). Once the void is filled, the cortical window is closed. Collapse of the disimpacted area is prevented by placing parallel screws along the articular margin and aiming them just inferior to and across the area of disimpaction (Fig.15-9D,E). Placed in this fashion, these screws act as a lattice to support the area of the grafting. The bone graft must be of good quality and adequate quantity. Cancellous bone can be obtained from the iliac crest. However, cancellous allograft or synthetic bone graft substitutes may also be used if the patient and surgeon so choose.

After the fragments are secured, the shoulder is taken through a range of motion in internal rotation. The position in which the grafted area comes into contact with the glenoid is noted. This position should be avoided postoperatively until the graft has consolidated, which usually requires at least 6 weeks. The anterior capsule is closed side to side and overtightening of the capsule is avoided. The subscapularis is reattached anatomically, and the wound is closed. Humeral internal rotation is avoided during closure. The patient is placed into a postoperative brace in neutral to 20 degrees of external rotation. If the defect is not repairable by this method, the patient is treated by either transfer of the subscapularis tendon into the defect, by insertion of the lesser tuberosity into the defect, or by allograft reconstruction. Hemiarthroplasty is generally not

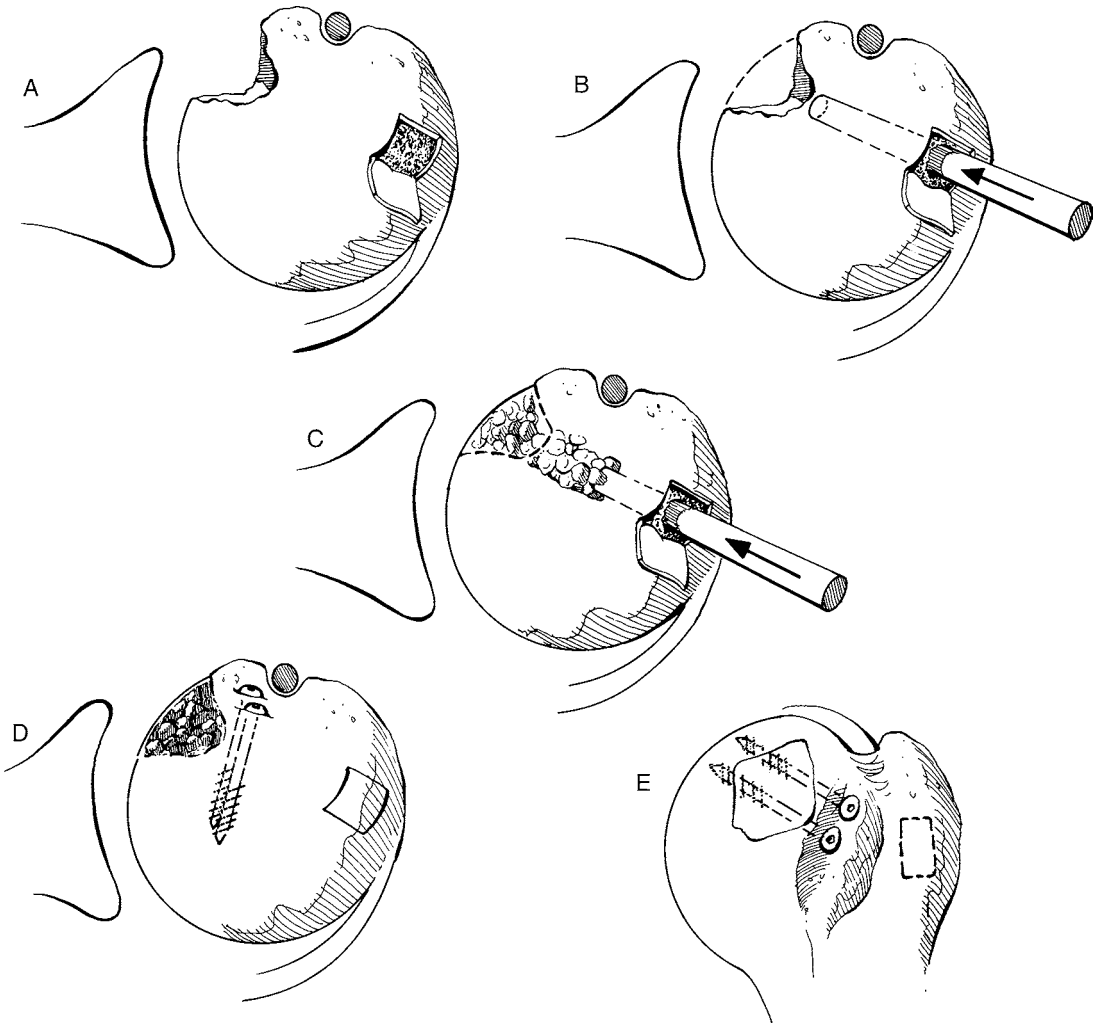


Figure 15-9 This schematic represents the technique of disimpaction and bone grafting of injuries less than 3 weeks old: **(A)** A bone window is made in the greater tuberosity opposite the fracture; **(B)** a bone tamp is used to disimpact the area; **(C)** cancellous autograft is packed into the defect to support the area; and **(D,E)** fixation is provided to shore up the area of grafting.

required for defects of this size, unless the remaining bone and cartilage viability is inadequate.

Rehabilitation is the same as that for closed reduction, with a few exceptions. The patient is not allowed internal rotation into the danger zone as determined intraoperatively for 6 weeks and, therefore, must wear the brace for 6 weeks. Gentle passive external rotation and isometric exercises are started on postoperative day 2.

Gerber has reported success with this technique.[12,13] No clinical series has been reported in the English literature.

Open Reduction with Transfer of the Subscapularis Tendon or Transfer of the Lesser Tuberosity

To successfully perform either subscapularis transfer or transfer of the lesser tuberosity, the patient must have adequate bone stock to securely hold either sutures or screws. The articular surface of the remaining intact humeral head must be structurally viable and the defect of the head should be less than 25%.

If the shoulder has been dislocated for more than 6 months, the possibility of the shoulder requiring a shoulder arthroplasty must be discussed with the patient, and the prosthesis must be available at the time of surgery.

Transfer of the Subscapularis

This procedure should be considered for patients with small humeral head defects (less than 25%) and continued symptoms of instability following reduction. The approach used to perform the subscapularis transfer is the same as that used for performing an arthroplasty, with the exception that the tendon of the subscapularis is released from

its insertion. The joint is exposed and débrided of scar tissue. The humeral head is reduced, as described earlier. The articular cartilage is evaluated and the size of the defect is confirmed visually. If the articular cartilage of the intact humeral head is viable and the defect is less than 25%, then transfer of the subscapularis is undertaken. If the remaining articular cartilage is not significantly damaged and the defect size is between 25% and 45%, a transfer of the lesser tuberosity or osteochondral allograft is considered. If the articular surface of the remaining humeral head is not viable or the defect is larger than the anticipated 45%, the patient is moved into the arthroplasty group. Arthroplasty is also performed if the bone in the intact portion of the humeral head is markedly osteoporotic.

The deltopectoral approach is used. The entire subscapularis tendon is raised from its insertion. Any adhesions to the subscapularis are released from its anterior and posterior surfaces. The humeral head is reduced and the posterior rim of the glenoid is inspected for the presence of any significant erosion that would preclude stability without reconstruction. If needed, the glenoid is reconstructed with bone graft. If minimal erosion is present the posterior rim of the glenoid is roughened to create a healing surface for the capsular structures. The defect in the humeral head is then cleared of all scar tissue until a bed of cancellous bone is prepared. Transosseous sutures are used to secure the subscapularis into the defect. The shoulder is taken through a full range of motion and should be stable. If the shoulder dislocates despite the repair, then the defect should be considered too large for this technique, and either an allograft or arthroplasty should be performed. If the shoulder is stable, then the sutures are tied and the wound is closed. The patient is then placed in a prefabricated splint with the arm held at the side in 20 degrees of external rotation.

Transfer of the Lesser Tuberosity

The standard deltopectoral approach is used, and the biceps tendon is identified. The rotator cuff interval is identified and released sharply. This allows access to the superior aspect of the lesser tuberosity and helps to visualize the articular side of the osteotomy. Once the lesser tuberosity is identified, it is osteotomized. The biceps is released from the supraglenoid tubercle and is routinely tenodesed. Once the lesser tuberosity osteotomy is complete, the freed tuberosity is secured with a traction stitch. With traction applied, the subscapularis and the underlying capsule are dissected as one layer away from the anterior surface of the glenoid. The subscapularis must be freed of adhesions. The joint is débrided of scar tissue. The articular surfaces are evaluated for viability. The glenoid is addressed as before and the area of the humeral defect prepared. The lesser tuberosity is secured into the defect with one to two cancellous lag screws at 90 degrees to the plane of the defect. The humerus is internally rotated to assess the stability of the shoulder. The shoulder should be stable after this procedure. If dislocation continues, then the defect was too large for this procedure, and the patient should be considered for allograft reconstruction or prosthetic arthroplasty.

Rehabilitation

Patients with subscapularis or lesser tuberosity transfer are immobilized in the brace in neutral to 20 degrees of external rotation for 4 weeks. The patient is allowed to begin passive external rotation and abduction of the shoulder on postoperative day 2. At this time, the patient also begins isometric strengthening of the external rotators of the shoulder.

At 4 weeks, the brace is removed and the patient is allowed full motion of the shoulder, except for active internal rotation. Active internal rotation is discouraged until week 6. At this point, gentle active internal rotation and light strengthening of the shoulder girdle muscles are commenced. More strenuous activity is not allowed for an additional 6 weeks.

McLaughlin[26] was the first to report the use of the subscapularis tendon transfer technique in the treatment of locked posterior dislocations of the humeral head. He reported using this technique in four patients and found the results to be satisfactory in the two patients with sufficient follow-up. Rowe and Zarins[35] reported satisfactory results in their two patients treated by subscapularis transfer.

In 1982, Hawkins et al.[15] reported on nine patients who had been treated with subscapularis transfer and reported excellent results in four of the patients they had treated with this procedure. The average range of motion obtained in these patients was 165 degrees of active forward elevation, 40 degrees of external rotation, and internal rotation to the level of the 12th thoracic vertebra. The remaining five patients were referred to them after already having undergone subscapularis transfer by the referring physician. These patients were therefore considered failures and were treated with a different procedure.

Hawkins et al.[15] and Neer[28] advocated transfer of the lesser tuberosity for patients with humeral head defects involving 20% to 45% of the articular surface. Neer developed the lesser tuberosity transfer procedure and treated four patients using this technique.[28] All four patients were reported to have excellent results. The average range of motion in these four patients was 160 degrees of active forward elevation, 45 degrees of external rotation, and internal rotation to the 12th thoracic vertebra.

Walch et al. reported the results of subscapularis transfer in 10 patients: three patients had excellent results, one good, five fair, and one poor.[39] Checchia et al.[6] used either a subscapularis transfer or lesser tuberosity transfer in 13 of their patients. Nine out of 13 patients were available for follow-up. Three of the outcomes were considered excellent,

four good, one fair, and one poor. The average UCLA score for this group was 28.8 points. They grouped both the tendon and tuberosity transfers together so that it is impossible to determine more than the general results or to determine if there was any difference in the outcomes between the two techniques.

Gerber[12,13] has argued against this method because it distorts the anatomy of the proximal humerus and limits the arc of humeral articular surface available for internal rotation. He also argued that this would cause revision to an arthroplasty to be difficult if the tuberosity transfer were to fail. Therefore, he advocates the use of an allograft to reconstruct defects of this size. Of note, no patient treated by Neer required a revision operation after transfer of the lesser tuberosity. However, his follow-up of these patients was limited at a range of 2 to 9 years.[28]

Allograft Reconstruction

In 1996, Gerber[13] reported on the use of allografts to treat the humeral head defects associated with chronic locked posterior dislocations of the shoulder. He recommended using this technique in patients whose shoulder had been dislocated at least 4 weeks and had defects involving up to 50% of the humeral head.[12,13] He advised against the use of this technique if there was significant osteoporosis present in the remaining portion of the humeral head. A suitable fresh frozen allograft must be available for this type of reconstruction. A size-matched, fresh frozen humeral head allograft is best suited for obtaining the graft. If this is not available, a fresh frozen femoral head will suffice. This procedure should be reserved for the younger patient and for the older patient with good bone stock. A CT scan is helpful in evaluating for significant osteopenia.

The exposure is the same as previously described for elevation and bone grafting. Again, a vertical capsulotomy should be used, so that the insertion of the coracohumeral ligament and the superior glenohumeral ligament are preserved or repaired. If the cartilage and bone quality are sufficient, then the reconstruction is performed. If the quality of either tissue is poor, then an arthroplasty is performed. The glenoid is addressed as previously described.

Instead of curetting the base of the humeral head defect, an osteotome or oscillating saw is used to cut a wedge out of the humeral head, which includes the edges of the defect (Fig. 15-10A). Once this is done, the breadth of the excised wedge, as well as its depth, should be ascertained (Fig. 15-10B). The allograft head should then be inspected for a surface that will match the contour of the humeral head at its defect. An oscillating saw is then used to cut a wedge from the allograft that is approximately 2 mm wider than the width of the true defect (Fig. 15-10C). This will

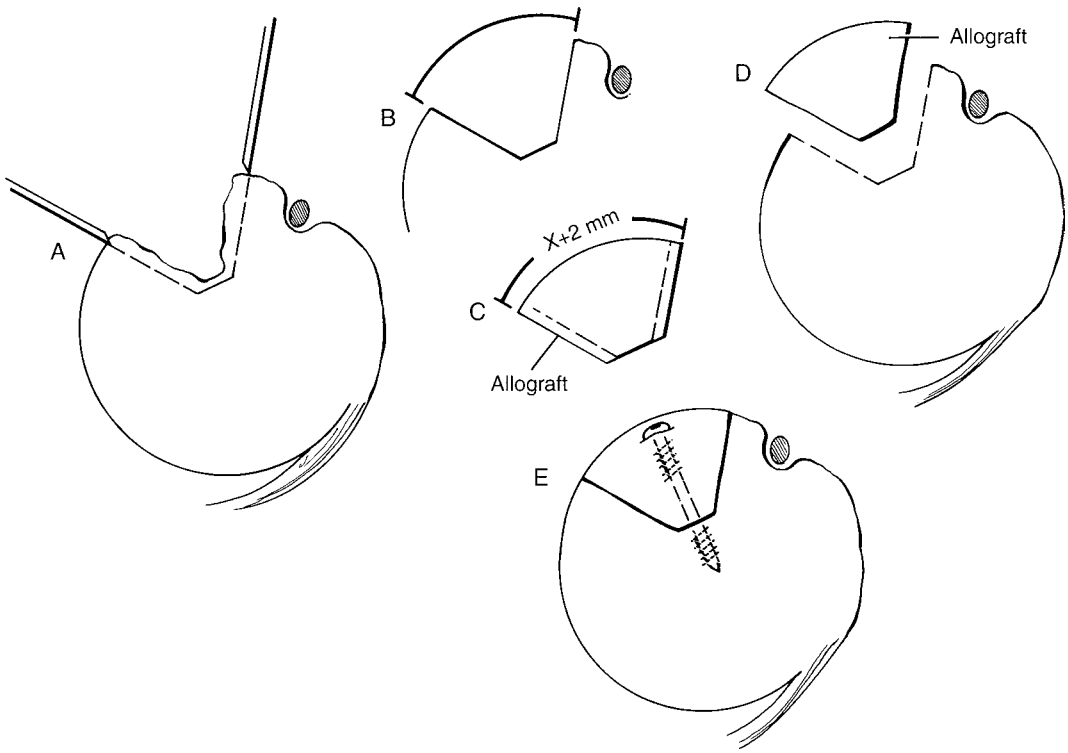


Figure 15-10 This schematic represents the technique of allograft reconstruction: **(A)** The edges of the defect are prepared sharply; **(B)** the defect is measured; **(C)** a suitable surface of the allograft is used to cut a wedge 2 mm larger than the defect; **(D)** the allograft is impacted into place; **(E)** the allograft is fixed into position with a cancellous screw.

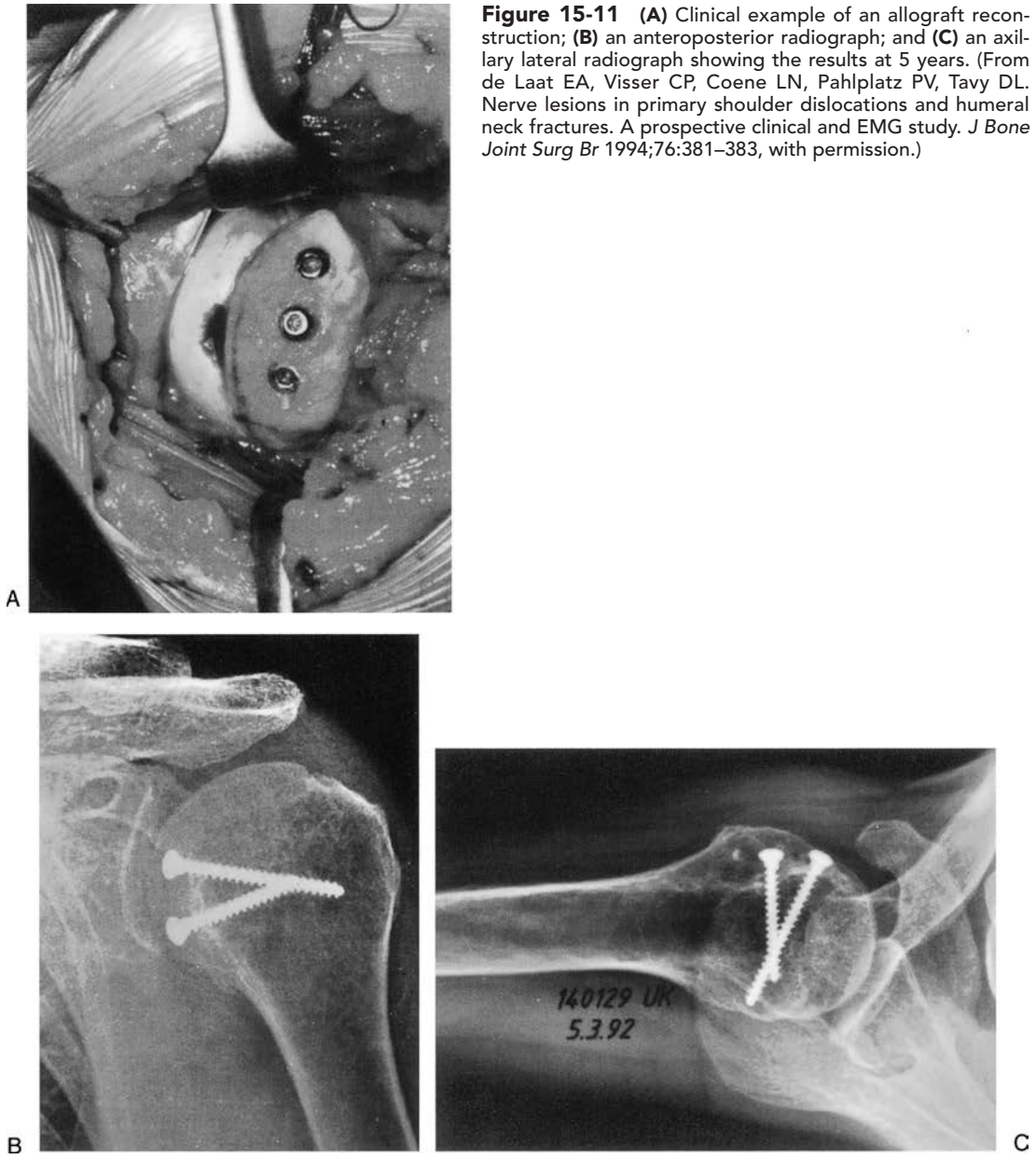


Figure 15-11 **(A)** Clinical example of an allograft reconstruction; **(B)** an anteroposterior radiograph; and **(C)** an axillary lateral radiograph showing the results at 5 years. (From de Laat EA, Visser CP, Coene LN, Pahlplatz PV, Tavy DL. Nerve lesions in primary shoulder dislocations and humeral neck fractures. A prospective clinical and EMG study. *J Bone Joint Surg Br* 1994;76:381–383, with permission.)

permit impaction of the allograft into the defect, creating a press fit (Fig. 15-10D). The graft is then fixed with one or two countersunk cancellous lag screws or Herbert screws (Figs. 15-10E and 15-11). The shoulder is then taken through a full range of motion and should be stable, with no further tendency for dislocation. If the shoulder is unstable, this can be remedied by either imbrication of the posterior capsule or by postoperative immobilization of the shoulder in a stable position. The posterior capsule avulsion or redundancy rarely, if ever, needs to be addressed by formal posterior capsulorrhaphy because experience has shown that the muscular and capsular tension about the shoulder will return with proper rehabilitation.[12] The subscapularis is repaired anatomically and the wound is closed. The patient is immobilized in a prefabricated brace in neutral rotation for 6 weeks. Rehabilitation is similar to that following subscapularis tendon transfer, with the exception of the additional 2 weeks of immobilization.

Allograft reconstruction of articular surface defects has been an efficacious and safe treatment for articular surface defects in lower-extremity, weight-bearing joints.[24,25] These data support the use of allografts in the reconstruction of defects in the humeral head. Moreover, the shoulder is typically less of a weight-bearing joint than lower-extremity joints. Therefore, fewer demands are placed on the allograft. In 1996, Gerber[12] presented the preliminary results in four patients treated for locked posterior dislocations of the humeral head using allograft reconstruction. Three of

the patients had satisfactory results, with an average score of 95% of that of an age- and sex-matched normal population. This scoring was based on the system of Constant and Murley.[9] The follow-up period for these three patients was more than 5 years from the time of the procedure. The one poor result was in a patient who developed avascular necrosis and collapse of the head after 6 years from the time of surgery. It was felt that this development was related to the patient's history of alcohol abuse and not to the reconstruction. Gerber later stated that he has had similar results in an additional six patients. He did, however, stress that this procedure should not be used if the patient's bone was osteoporotic by CT scan or by examination at the time of surgery.

The results of allograft reconstruction given by Gerber's series are similar to those shown with the other humeral head–preserving techniques. This technique potentially allows preservation of normal kinematics in the shoulder joint and preserves the normal anatomy of the proximal humerus. We agree with Gerber and believe that, if at all possible, allograft reconstruction should be performed as the treatment of choice in young patients and in older patients with adequate bone stock and intact articular cartilage on the remaining portion of the humeral head.

Hemiarthroplasty and Total Shoulder Arthroplasty

Indications for either a hemiarthroplasty or a total shoulder arthroplasty are a defect measuring more than 50% of the articular surface of the humeral head, severe articular cartilage damage, marked osteopenia, or erosion of the glenoid rim, leading to instability (see Figs. 15-3 and 15-6).

The choice of whether to use a hemiarthroplasty versus a total shoulder arthroplasty depends on the condition of the glenoid surface. If the glenoid has significant wear and erosion, then the glenoid may require resurfacing. If the dislocation has not been present long enough to irreversibly damage the cartilage of the glenoid, then a hemiarthroplasty is preferred (Fig. 15-12). In patients too young for glenoid resurfacing, consideration is given to arthrodesis or hemiarthroplasty with soft tissue interposition.

The standard deltopectoral approach is used. The tuberosities should not be osteotomized if at all possible. If a malunion of the neck is present, an osteotomy of the neck may be required. An osteotomy of the neck is preferred because it allows placement of the humeral component into correct rotational alignment, which restores more normal anatomy. In some cases, the humeral prosthetic may be placed in significantly less retroversion (0 degrees) to compensate for the change in the soft tissue balance caused by the dislocation. In general, the more chronic the injury is, the less retroversion is used. Hawkins et al.[15] reported that, with experience, they felt that they could dial in the precise amount of version required to restore immediate joint stability. Oversizing of the humeral head to make up for a redundant capsule, in general, should be avoided. The size of the prosthetic head should be chosen to match the anatomic size of the patient's native head. Once the trials are in place, the

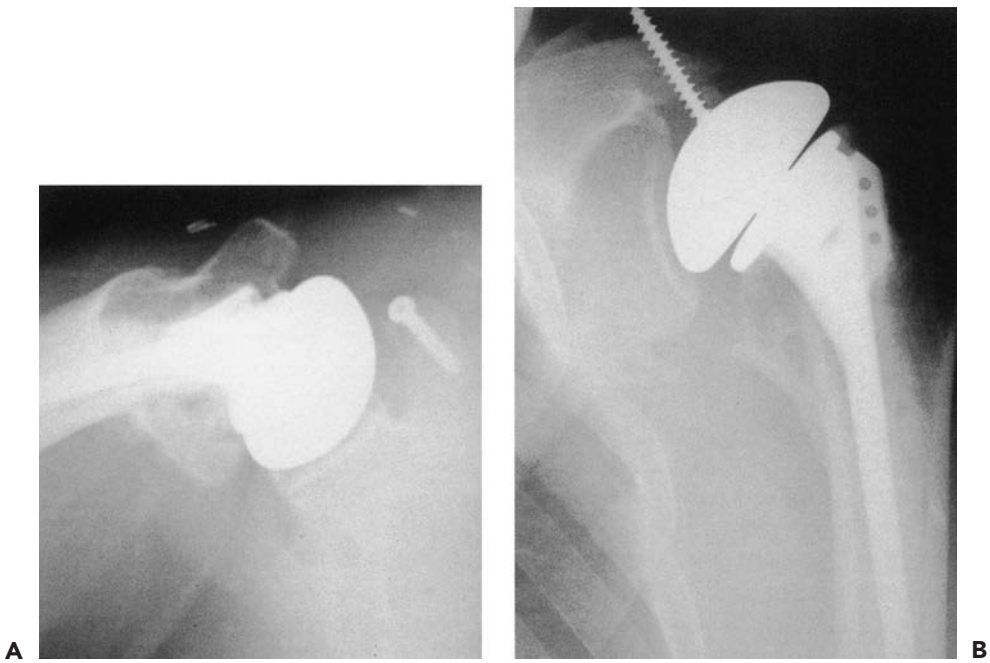


Figure 15-12 Radiographs taken 2 years after a hemiarthroplasty was performed on the patient whose computed tomography scan is shown in Fig. 15-6.

shoulder is taken through a full range of motion to determine joint stability. Because the posterior capsule may be patulous as a result of chronic distension, a posterior capsular plication may be necessary. In our experience, this is rarely needed. Stability of the joint must be evaluated intraoperatively. Active contraction of the rotator cuff in the awake patient will improve shoulder stability. This may correct subtle intraoperative instability, but will not correct for gross instability. This must be corrected intraoperatively through a combination of soft tissue balancing, bone grafting, glenoid reaming, and component version.

Rehabilitation

If the shoulder is stable after shoulder arthroplasty, the patient does not require bracing. The patient is allowed to perform range-of-motion exercises by postoperative day 3, and by 2 weeks active and light resistive exercises are added. No heavy use of the extremity is allowed for 3 months. If at the time of surgery the shoulder was found to be unstable despite plication of the posterior capsule, then the arm is immobilized in neutral rotation for 6 weeks. On postoperative day 1, the patient is allowed gentle active external rotation and is allowed isometric strengthening of the external rotators and deltoid. These exercises are performed in the brace. The brace is removed at 6 weeks, and the patient is allowed full motion of the joint. He or she is also started on muscle conditioning exercises at that time.

Hawkins and colleagues[15] remedied the tendency for posterior dislocation in patients undergoing prosthetic arthroplasty by changing the version of the humeral component. During the early period of their study, they suggested that the humeral component be placed in neutral rotation if the dislocation had been present for longer than 6 months, and in 20 degrees of retroversion if the dislocation had been present for less than 6 months. With more experience, the authors stated that they felt that they could adjust the precise degree of version to restore immediate stability. By using this technique, they were able to obtain satisfactory results in 11 of 16 patients. Their best results were seen in patients who had been treated with primary total shoulder arthroplasty (TSA), which was available only during the later part of their patient series. Of the six patients who underwent primary TSA, five showed excellent results and had an average active forward elevation of 152 degrees, external rotation of 40 degrees, and internal rotation to the level of the 12th thoracic vertebra. The one failure had a postoperative dislocation and refused further treatment. These results led them to recommend TSA as the procedure of choice in the treatment of chronic locked posterior dislocations in patients not felt to be candidates for humeral head preservation. Others have also advocated changing the version of the humeral component to control the tendency toward redislocation.[8,28,32]

Cheng et al.[8] reported on seven cases treated with TSA. Utilizing the American Shoulder and Elbow Society shoulder score, they noted an improvement from a preoperative mean of 20.1 to a mean of 55.6 postoperatively. Checchia et al.[6] reported their results of hemiarthroplasty in eight patients. The mean UCLA score was 25.6 points; three cases were classified as excellent, two as good, one as fair, and two as poor. Of the two poor results, one required revision to a total shoulder arthroplasty. They performed a TSA in five patients but did not distinguish the chronicity of the dislocation prior to treatment. Four of these patients were considered to have unsatisfactory results. Their poor results with TSA are in contrast to most other reported series of chronic dislocations treated with this method.[8,12,14]

It is Gerber's recommendation that the normal axis of rotation be recreated whenever treating a chronic posterior dislocation with an arthroplasty.[12] He felt that this would lead to more normal joint kinematics than would be seen if the humeral component was placed in excessive anteversion. To prevent postoperative instability, he recommended balancing of the soft tissues of the posterior capsule, the subscapularis, and the anteroinferior capsule. It was his belief that with postoperative immobilization and properly modified rehabilitation, the soft tissues would rebalance toward their normal tensions. He stated that the balancing of these tissues may require the use of a brace for 6 weeks after the surgery. The authors of this chapter agree with these recommendations and place the prosthetic in natural version and balance the soft tissues to achieve stability. It is the editors' experience that change in humeral version does not afford a significant improvement in joint stability. No clinical outcome data are available for the patients that he has treated with this technique. Gerber does concur with Hawkins and Neer that TSA is the treatment of choice in patients with long-standing dislocations.

CHRONIC LOCKED ANTERIOR DISLOCATION

Although many of the same principles used in the diagnosis and treatment of posterior dislocations are employed in the diagnosis and treatment of anterior dislocations, anterior dislocations have unique characteristics that require a distinct approach to diagnosis, treatment, and rehabilitation. For instance, an anterior dislocation leaves the arm in relative abduction and external rotation. If the arm remains locked in this position, the patient will be unable to reach the mouth and will be unable to perform daily care activities. This nonfunctional position of the arm is the likely reason that the functional outcome in patients with neglected anterior dislocations yields a poorer result than the results seen in locked posterior dislocations. In 1982, Rowe and Zarins reported on a series of seven patients with chronic, untreated shoulder dislocations.[35] In this study, they found that the three patients with

untreated posterior dislocations scored better in objective and subjective testing of shoulder function than the four untreated anterior dislocations. A recent case report by Jerosch et al.[18] reported a single patient with a locked anterior dislocation of 4 years' duration. The surprising function seen in this patient may represent expansion of the Hill-Sachs defect over the long period of follow-up. This case does point to the importance of an objective functional assessment of each patient prior to making a recommendation for surgery.

Classic teaching would lead one to believe that locked posterior dislocations are more common than locked anterior dislocations; however, this is not the case. The larger number of missed anterior dislocations likely occurs as a result of the proportionally larger rate of occurrence of acute anterior dislocations, compared with that of posterior dislocations. However, as a percentage of the type of injury, posterior dislocations are more frequently missed.

History

As with posterior dislocations of the shoulder, anterior dislocations of the shoulder may result from trauma or a convulsive episode. Classically, seizures have been related to the occurrence of posterior dislocations of the shoulder; however, anterior dislocations have been reported to occur in one-half of all dislocations caused by a seizure.[3,36] Clinical suspicion should, therefore, be raised in any seizure disorder patient who has shoulder pain or a diagnosis of a frozen shoulder that is not responding to therapy. Patients suffering from a multiple trauma can often have a shoulder injury that is overlooked because of the severity of other injuries (Fig. 15-13). Thorough repeat examinations of the trauma patient are necessary to avoid these unrecognized injuries.

Physical Examination

The physical examination of a patient with a chronic locked anterior dislocation of the shoulder typically reveals severe limitation of rotation. The patient usually is seen to hold the arm away from the body in external rotation. Pain with chronic dislocations is variable and may be relatively mild after a long-standing dislocation. Fig. 15-14 shows the clinical photographs of a patient who was misdiagnosed as having a shoulder contusion and was later treated as a frozen shoulder. After 3 months of failed therapy, the diagnosis of a locked anterior shoulder dislocation was made. The diagnosis was rendered by simply obtaining the appropriate radiographs (Fig. 15-15).

A thorough neurovascular examination should also be performed. Schulz et al. showed in their series that 40% of anterior dislocations had an associated neurologic injury.[36] In a prospective evaluation using EMG, de Laat and colleagues found evidence of a nerve injury in 45% of patients after an acute dislocation of the shoulder or fracture of the humeral neck.[10] Therefore, electromyography should be performed to document any suspected brachial plexus or axillary nerve injury before surgical correction of any chronic deformity. Damage or rupture of the axillary artery with anterior dislocations of the shoulder have also been reported.[1,21,29]

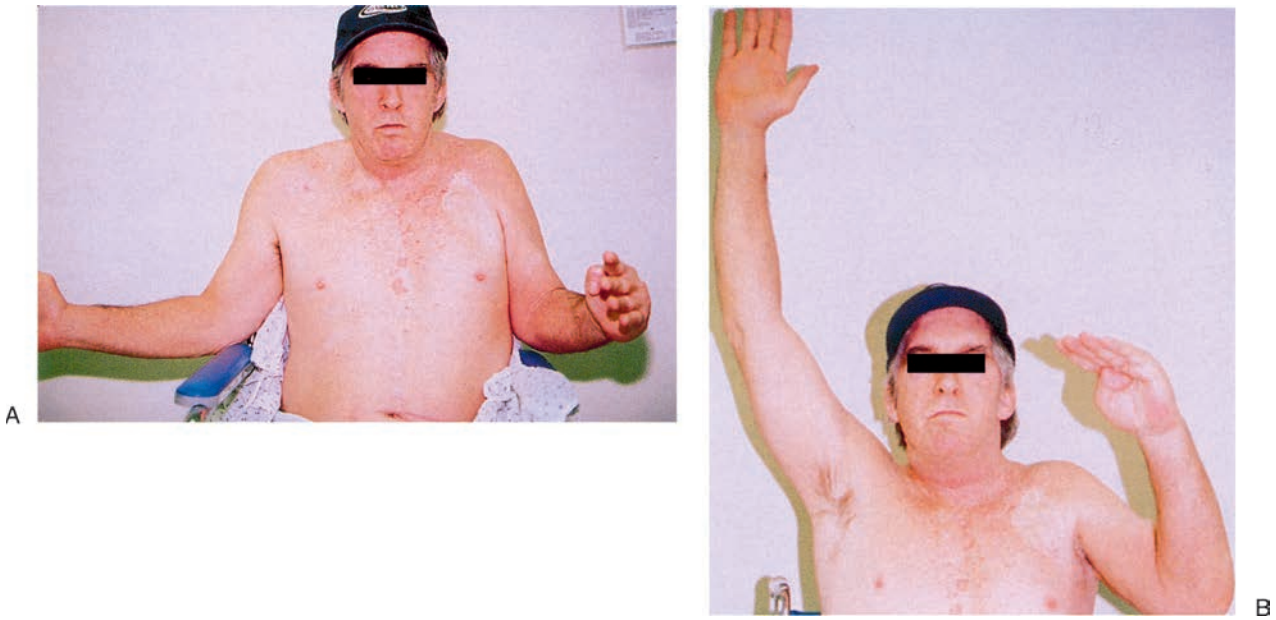


Figure 15-13 Clinical photographs of a multiply injured trauma patient who presented with a chronic locked anterior dislocation: **(A)** He has severe limitation of rotation, with a marked amount of pain. **(B)** The severe loss of forward elevation made reaching his face very difficult.

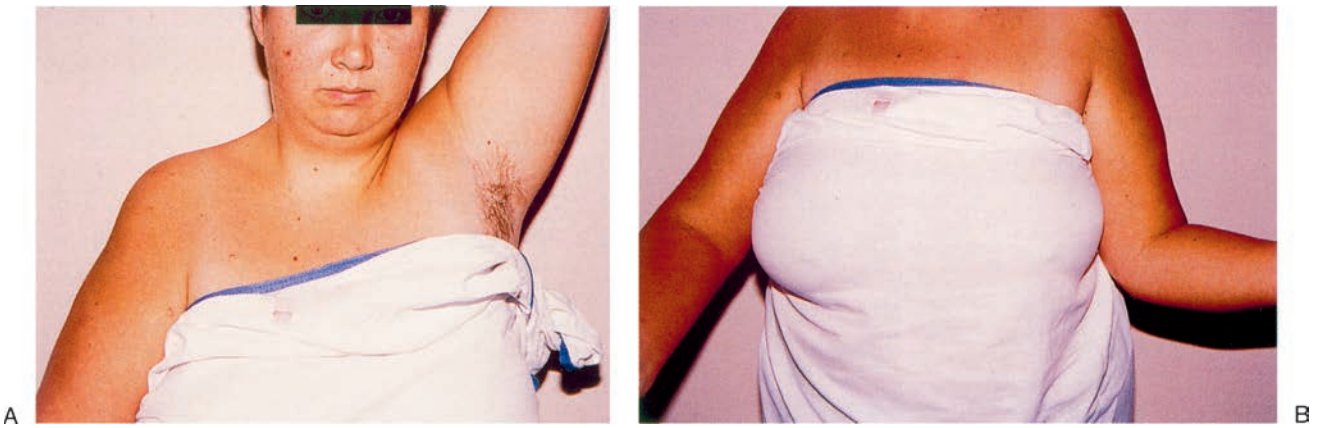


Figure 15-14 Clinical photographs of a patient misdiagnosed with a shoulder contusion after a fall: **(A)** Marked limitation of forward elevation of the right arm was both active and passive, secondary to a locked anterior dislocation with an associated axillary nerve palsy. **(B)** The loss of rotation of the right arm is depicted in this photograph. (Courtesy of Jeffrey S. Noble, MD, Akron, Ohio.)

Radiographic Evaluation

Radiographic evaluation of the locked anterior dislocation should include an anteroposterior and lateral view of the shoulder in the plane of the scapula, as well as an axillary lateral view (see Fig. 15-15). As with locked posterior dislocations, the axillary lateral is essential for diagnosis as well as to determine the extent of humeral head impaction. It will also provide information regarding the integrity of the glenoid. Associated fractures of the proximal humerus or glenoid can be seen in up to 50% of these injuries and should be ruled out.[36] Preoperative determination of bone quality is important for surgical planning where an allograft may be needed for reconstruction of a large Hill-Sachs lesion. A CT scan will give valuable information relative to bone loss in the humeral head and glenoid (Fig. 15-16).

Intraoperative Findings

Locked anterior dislocations of the shoulder are characterized by intraoperative findings different from those seen in posterior dislocations. In anterior locked dislocations, the humeral head compression fracture is found in the posterolateral aspect of the head (the classic Hill-Sachs lesion[17]). In contrast to posterior dislocations, anterior dislocations commonly have glenoid loss as well as humeral head deficiency. The anteroinferior aspect of the glenoid cavity may become deficient from chronic wear against the posterolateral aspect of the humeral head (Fig. 15-17). In locked posterior dislocations, the posterior capsule and labrum are usually avulsed from the glenoid as a contiguous sleeve and are not felt to require reattachment to the glenoid during repair. In contrast, reattachment of the anteroinferior capsular structures and glenoid labrum to the glenoid is very important in the recreation of a stable shoulder after reduction of a locked anterior dislocation.

The neurovascular structures are also at a much greater risk during surgical reconstruction of locked anterior dislocations than with posterior dislocations. Scarring of the brachial plexus and axillary vessels may occur to the anterior aspect of the subscapularis muscle, making soft tissue dissection difficult. The axillary nerve is frequently stretched and scarred to the inferior aspect of the subscapularis muscle, placing the nerve at risk during open reduction. The axillary nerve must be identified and protected throughout the procedure. Neurapraxias are not uncommon after open reduction of a chronic anterior dislocation. Recovery is common, as long as the nerve is gently protected and not transected.

Treatment Choice and Preoperative Planning

As with the treatment of locked posterior dislocations, the treatment options for locked anterior dislocations range from benign neglect to total shoulder arthroplasty. The treatment of choice will depend on the size of the defect, the time from injury, the condition of the humeral head and glenoid, and the patient's medical status. The surgeon must develop an algorithmic approach to the treatment of these injuries, so that the proper preoperative plan can be made and the surgeon can move along the algorithm if intraoperative findings require a change of treatment (Fig. 15-18).

Nonoperative

Nonoperative treatment is an option in the treatment of locked anterior dislocations of the shoulder. Benign neglect may be the treatment of choice in patients with little discomfort and minimal functional limitation.[18] It is also indicated in patients not felt to be medically fit for surgical intervention.[11,12,35,36] Although the functional

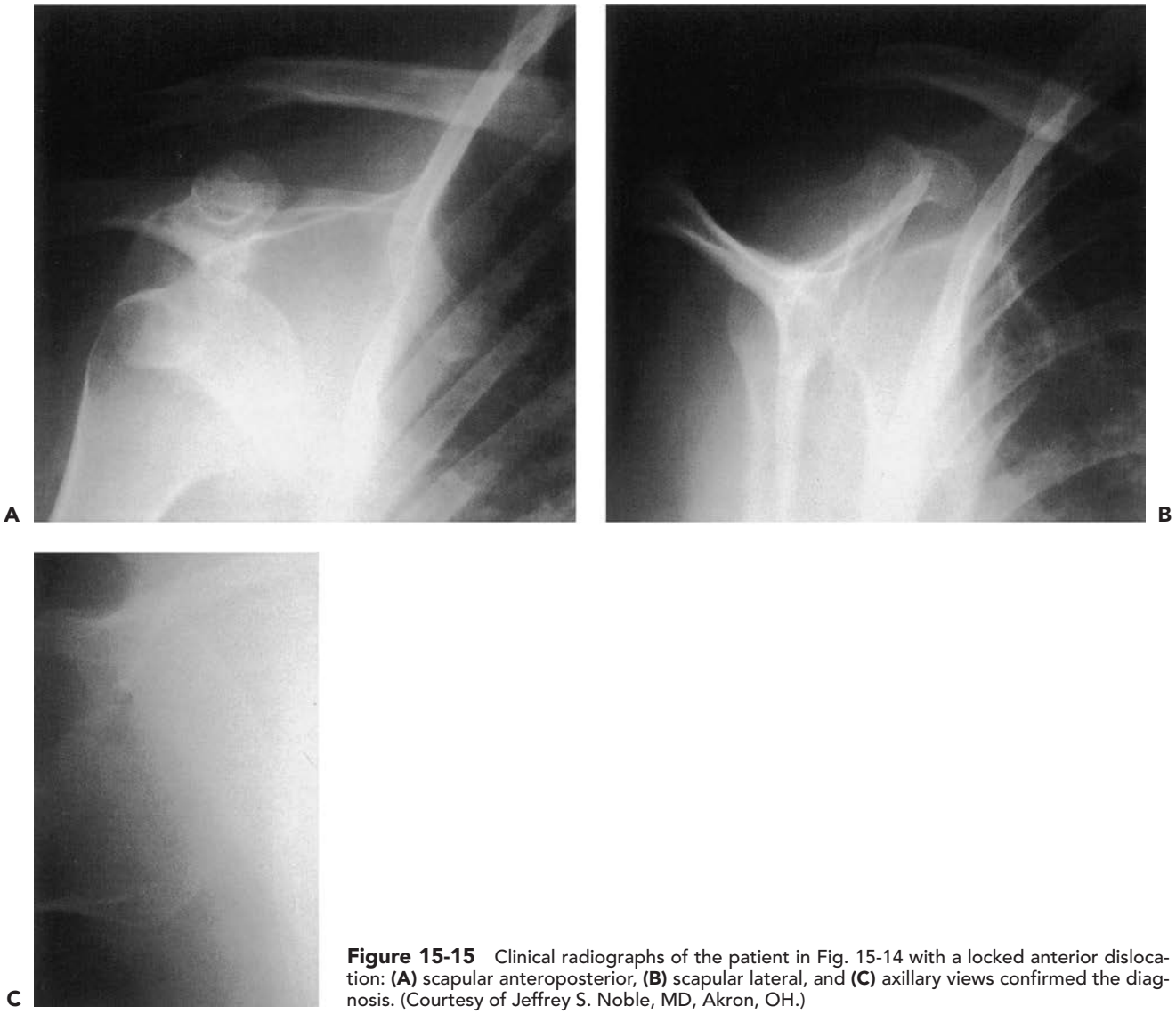


Figure 15-15 Clinical radiographs of the patient in Fig. 15-14 with a locked anterior dislocation: **(A)** scapular anteroposterior, **(B)** scapular lateral, and **(C)** axillary views confirmed the diagnosis. (Courtesy of Jeffrey S. Noble, MD, Akron, OH.)

results of untreated anterior shoulder dislocations are inferior to the results obtained after reduction, some patients have a surprisingly functional range of motion (Fig. 15-19). Patients treated with benign neglect must usually have a functioning contralateral shoulder to be able to function independently in their activities of daily living.

Closed Reduction

The success of closed reduction is very unlikely in a locked anterior dislocation more than 3 weeks old.[12,36] Because the incidence of vascular injuries reported with closed reductions performed in the setting of a chronic injury is high, we do not recommend an attempt at closed reduction if the injury is older than 3 weeks.[4] This holds especially true for older patients. If a closed reduction is to be attempted, careful evaluation of preoperative radiographs must be done to ensure that there is not an associated fracture of the tuberosities, humeral neck, or glenoid. If an associated fracture is present, it should be addressed, as described in the treatment of locked posterior dislocations. To perform a closed reduction, slow gentle traction under general anesthesia or scalene block is required. Torque on the arm must be kept to a minimum to prevent humeral fracture.

Open Reduction

If the impaction fracture of the humeral head involves more than 25% of the articular surface or the dislocation is more than 3 weeks old, the patient will require an open reduction. As previously stated, surgical exposure in chronic anterior dislocations requires careful handling of soft tissues to prevent damage to the neurovascular

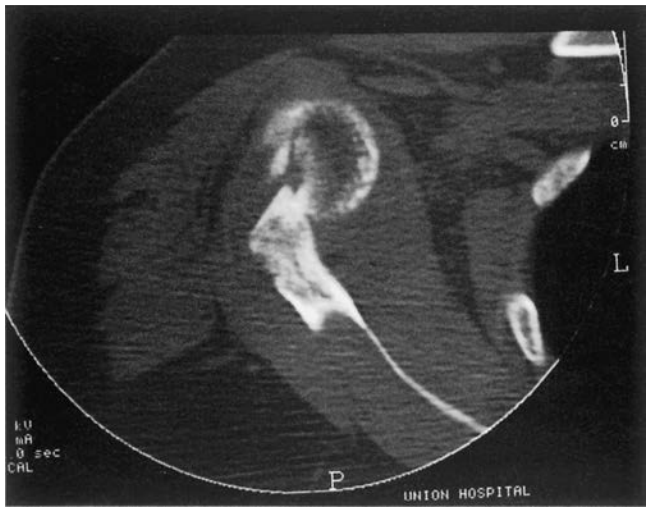


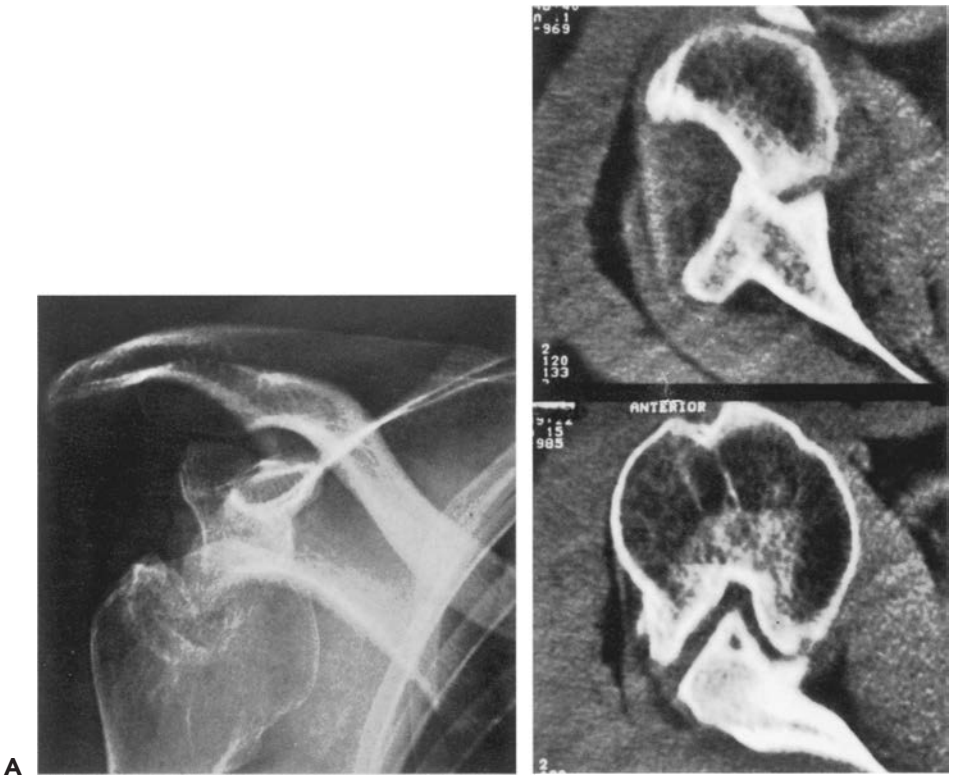
Figure 15-16 Computed tomography scan of the patient in Figs. 15-14 and 15-15 with a locked anterior dislocation. The glenoid was relatively preserved, with a Hill-Sachs lesion of approximately 25%. (Courtesy of Jeffrey S. Noble, MD, Akron, OH.)

structures. The anterior labrum and glenoid will also require special attention to regain stability after reduction.

We will first describe the surgical approach to the shoulder and then we will discuss the surgical management of the capsulolabral complex and the deficient glenoid. This will then be followed by a discussion of the surgical options for treatment of the humeral head defect.

Surgical Approach

A long deltopectoral approach is employed for anterior dislocations. The incision extends from just proximal to the coracoid to just medial to the deltoid insertion. An osteotomy of the coracoid process with its attached conjoined tendon is often necessary to aid in exposure of the medially displaced humeral head in chronic cases. If an osteotomy is necessary, the coracoid may be predrilled and the hole tapped prior to performing the osteotomy. This will ensure anatomic alignment of the coracoid at the time of reduction. At the time of closure, the coracoid is fixed with a partially threaded cancellous screw. Alternatively, the coracoid can be reattached with interfragmentary sutures; this does not provide rigid fixation nor anatomic reduction but is often sufficient for stable healing. After the osteotomy is complete, the conjoined tendon is reflected medially to allow identification of the neurovascular structures. These structures must be gently retracted to avoid injury. Release of the superior portion of the pectoralis major tendon is helpful to gain access to the



A B

Figure 15-17 Right shoulder of a bilateral, locked anterior dislocation: There are severe changes seen on the humerus and the glenoid by **(A)** conventional radiographs and **(B)** computed tomography scan. (From de Laat EA, Visser CP, Coene LN, Pahlplatz PV, Tavy DL. Nerve lesions in primary shoulder dislocations and humeral neck fractures. A prospective clinical and EMG study. *J Bone Joint Surg Br* 1994;76:381–383, with permission.)

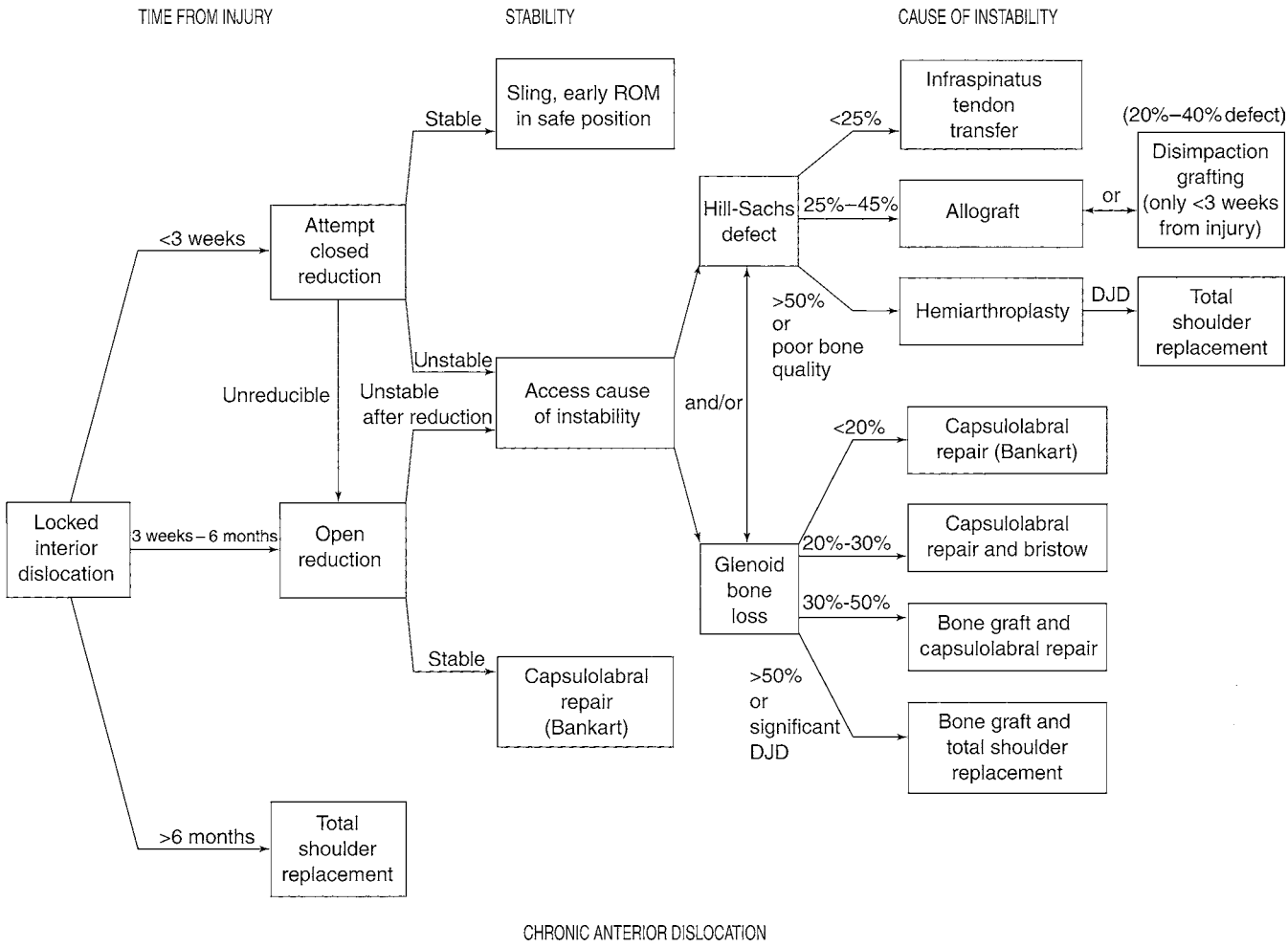


Figure 15-18 Algorithm for the treatment of chronic, locked anterior dislocations. DJD = degenerative joint disease; ROM = range of motion.

inferior portion of the subscapularis tendon and axillary nerve.

The subscapularis is then carefully dissected. Its anterior surface is freed of any adhesions to ensure that the brachial plexus is not adherent to it. The axillary nerve must be identified and freed from the inferior border of the subscapularis. For chronic dislocations requiring extensive dissection in this area, the axillary artery is at risk of injury and may require repair. Identifying an available vascular surgeon to be immediately available to scrub into the case is suggested in case severe bleeding occurs. This added precaution is particularly important in older persons in whom the vascular tissue tends to be more friable and at greater risk for tearing during dissection. Once free of adhesions, the entire subscapularis tendon is released just medial to its insertion into the lesser tuberosity.

The next step is the capsulotomy. The capsulotomy is performed by releasing the capsule from its humeral insertion. This is in contrast to the vertical capsulotomy performed during the approach to the posteriorly dislocated shoulder. The anterior capsule is attenuated by the dislocated humeral head. The inferior capsule, on the other hand, is contracted and tethers the humeral neck to the anteroinferior glenoid.[11] Before releasing the inferior capsule, the axillary nerve must be clearly identified and retracted to prevent injury. Once take-down of the capsule is performed, a Cobb elevator can be used to reduce the humeral head. Scar and granulation tissue must be débrided from the glenoid to allow reduction.

Once reduction is obtained, a posterior capsulotomy is performed along the glenoid margin. This maneuver allows internal rotation of the arm and helps to prevent the tendency toward redislocation caused by the tight posterior capsule. A humeral head retractor is often required to allow access to the posterior capsule. After the capsulotomy is completed, a Cobb elevator is used to ensure that the capsule is free from the posterior aspect of its glenoid insertion.

Locked anterior dislocations of the shoulder require reconstruction of the anteroinferior capsulolabral complex

Figure 15-19 Nonoperatively treated patient with a chronic, locked anterior dislocation who refused surgery: **(A)** Plain radiograph demonstrates the osteopenia associated with a chronic dislocation. The patient has decreased functional capacity **(B–D)** of the right arm, but is able to perform her daily activities with only mild discomfort.

(the Bankart lesion), as described by Rowe and Zarins in 1981.[34] A soft tissue Bankart lesion can be repaired with suture anchors placed onto the glenoid margin (Fig. 15-20). A bony Bankart lesion requires fixation of the bony fragment and its attached labrum back onto the glenoid. If there is marked loss of the glenoid surface by erosion, then a reconstruction of the glenoid may be required.

Glenoid Reconstruction

In cases of glenoid bone loss, a bone graft may be required to reconstruct the anterior glenoid rim. Smaller defects involving less than 20% of the glenoid surface may be reconstructed with a capsulolabral repair, reinforced with a Bristow procedure. A standard Bankart repair is performed, using suture anchors to reattach the capsulolabral complex to the margin of the remaining glenoid. The tip of the coracoid with its attached conjoined tendon is then fixed in an extraarticular fashion to the anteroinferior portion of the glenoid with a bicortical screw (Fig. 15-21). This bone block acts as a secondary restraint to anterior translation. The capsule and subscapularis tendon are then repaired in typical fashion.

If the lesion involves greater than 20% of the glenoid, then reconstruction with a tricortical iliac crest bone graft is required. The reconstruction must create a stable surface for the humeral head to articulate. Reconstruction of the glenoid is performed by first exposing its anterior surface

Figure 15-20 The patient in Figs. 15-14, 15-15, and 15-16 was reduced with an open reduction and take-down of the coracoid process for exposure: **(A)** Postreduction radiograph reveals a concentric reduction of the glenohumeral joint with repair of the capsulolabral complex. Follow-up clinical radiographs show excellent return of **(B)** forward elevation and **(C)** rotation after return of the axillary nerve. (Courtesy of Jeffrey S. Noble, MD, Akron, OH.)

by subperiosteal dissection. A burr is used to roughen the anterior glenoid to create an adequately flat surface for grafting. The size of the defect is then estimated, and a size-matched corticocancellous piece of iliac crest is harvested. The cortical surface of the graft is placed flush with the articular surface of the glenoid. The graft is then fixed to the anterior glenoid by partially threaded cancellous screws. The screws are placed parallel to the joint surface in a lagged fashion and in an anteroposterior direction. The donor graft is fashioned with a burr to allow a smooth transition from the glenoid cartilage to the graft reconstruction. The anteroinferior capsulolabral complex is then attached to the graft, using suture anchors (Fig. 15-22).

In the presence of degenerative arthritis or defects involving more than 50% of the glenoid surface, a total shoulder replacement is the treatment of choice. In this instance, the resected humeral head is used to reconstruct the glenoid. This reconstructed surface is used to support the glenoid component in a total shoulder replacement.

Postoperative rehabilitation for glenoid reconstruction must protect the anterior glenoid. A sling is worn for approximately 6 weeks and external rotation and motions posterior to the plane of the scapula are avoided.

Goga reported on 10 cases that he treated with an open reduction followed by a Bankart repair reinforced with a Bristow procedure.[14] He also utilized an acromiohumeral pin for 4 weeks. Outcomes evaluated using the method proposed by Rowe and Zarins: there were three excellent, five good, and two fair results.

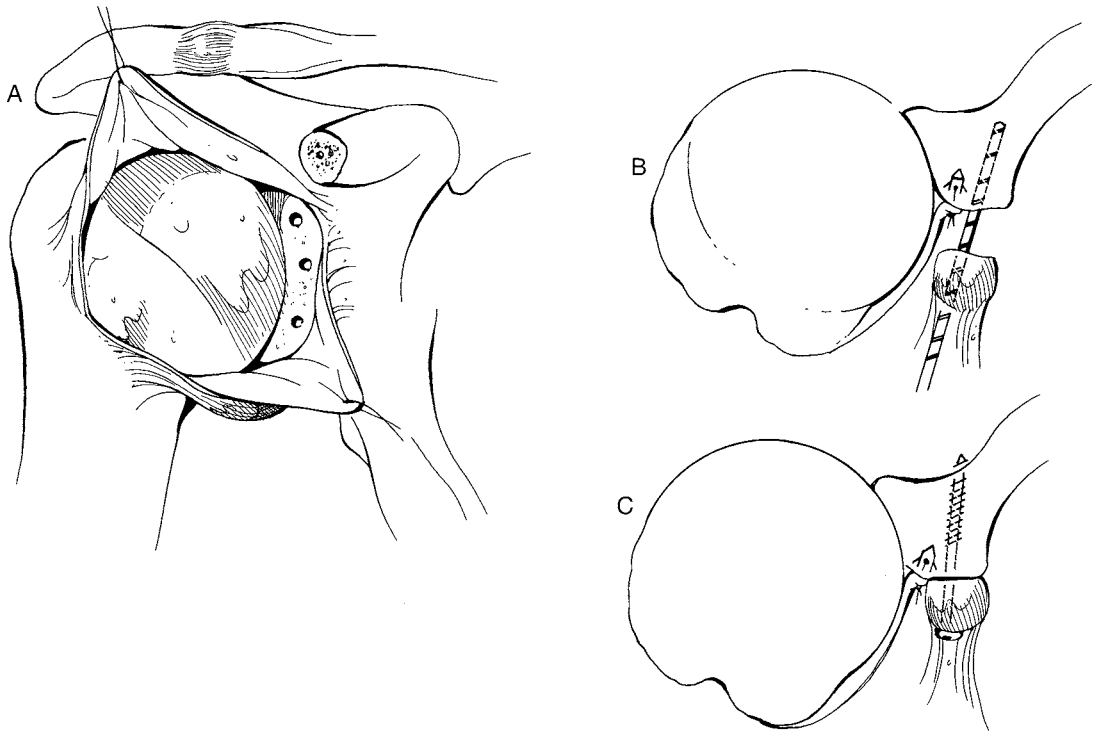


Figure 15-21 With moderate glenoid bone loss, an anterior capsulorrhaphy and coracoid transfer are performed. **(A)** The anchors are placed at the glenoid rim and the sutures are passed through the capsule and labrum, but not yet tied. **(B,C)** The glenoid defect is decorticated and the distal 1.5 cm of the coracoid are transferred to the defect and secured with a partially threaded screw.

Reconstruction of the Humeral Head Defect

As seen in the treatment of locked posterior dislocations of the shoulder, the choice of treatment will greatly depend on the size of the humeral head impaction fracture and the time from injury. Small Hill-Sachs lesions involving less than 25% of the humeral head often do not require attention. However, if the shoulder remains unstable after reduction, then transfer of the infraspinatus tendon may be required to obtain stability.

Defects larger than 25% of the humeral head should be addressed if the reduction of the glenohumeral joint is to be maintained. If the defect involves between 25% and 45% of the articular surface, the choices for treatment are the following: disimpaction with bone grafting, if the injury is less than 3 weeks old and there is adequate bone stock; allograft reconstruction, if the injury is more than 3 weeks old and the bone stock is adequate; or shoulder arthroplasty, if there is marked osteopenia or articular cartilage wear. Any defect involving more than 50% of the humeral head should be treated with an arthroplasty.

Disimpaction and Bone Grafting

If the humeral head defect involves between 20% and 40% of the humeral head and the injury is less than 3 weeks old, then disimpaction and bone grafting can be attempted. The indications and intraoperative evaluation of the humeral head are similar to those described for the posterior locked dislocation. In treating the Hill-Sachs lesion, the cortical window is created in the lesser tuberosity. Disimpaction and bone grafting are then carried out as described in Fig. 15-9.

The patient is placed in a sling for 3 weeks. Rehabilitation is begun on postoperative day 1. Gentle range of motion is begun, with care taken to avoid placing the arm in a position that would place stress on the disimpacted area. The positions to avoid should be determined at the time of surgery. In general, these will be abduction and external rotation. These motions should be avoided for approximately 6 weeks, until the graft has had time to consolidate.

Transfer of the Infraspinatus Tendon (Defects Less Than 25%)

The glenohumeral joint is exposed, as described previously. The shoulder is reduced and the size of the defect confirmed. The shoulder is then taken through a range of motion, and the position of instability is noted. If the shoulder is unstable at less than 90 degrees of abduction and 90 degrees of external rotation, an infraspinatus

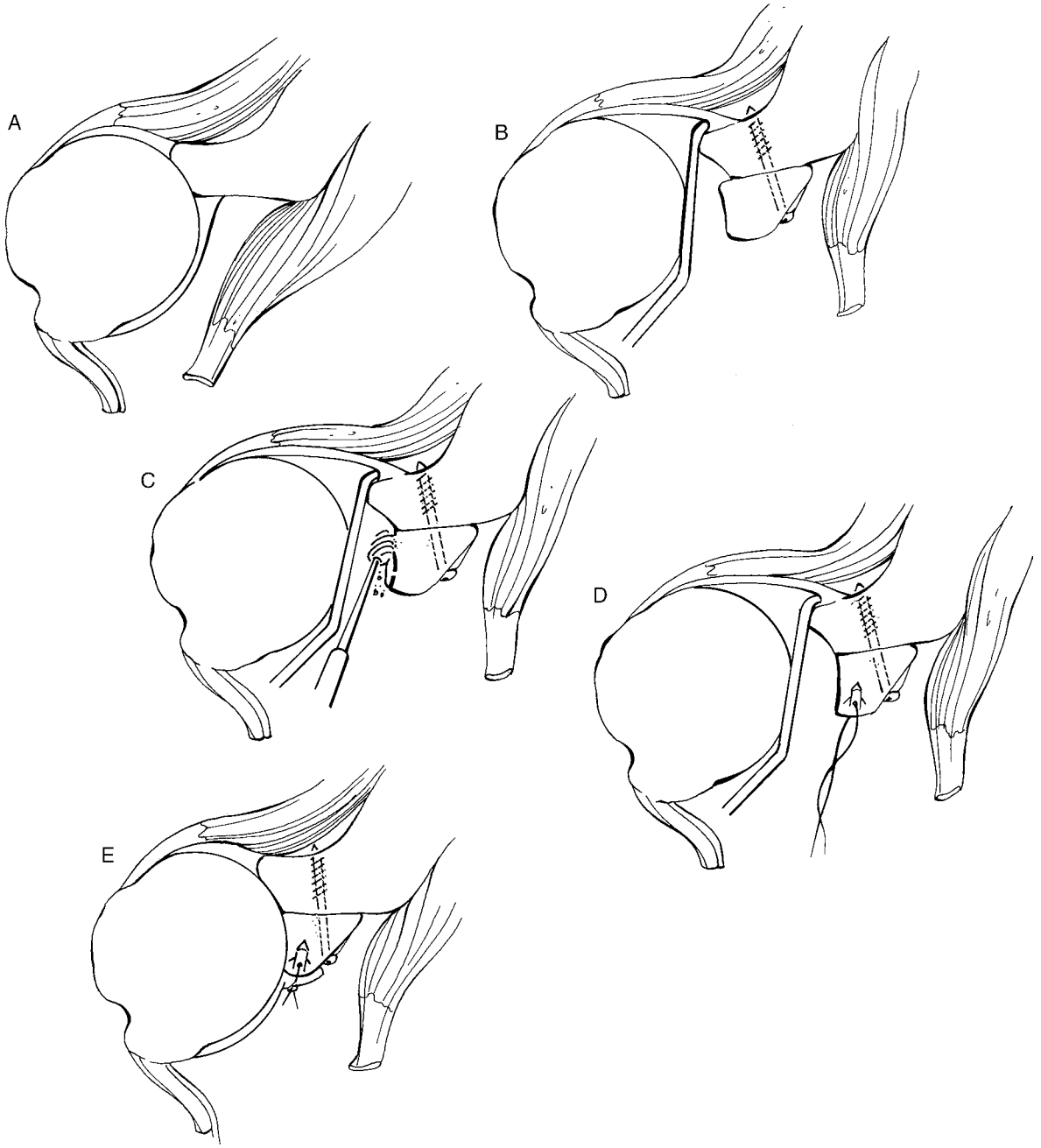


Figure 15-22 **(A)** For large anterior glenoid defects an iliac crest graft is used to reconstruct the glenoid defect. **(B)** The defect is decorticated and made flat. A bicortical iliac crest graft is placed with its cancellous surface against the cancellous bone of the defect and secured with two partially threaded screws. **(C)** The position of the graft, which overhangs the face of the glenoid, is burred down to make a smooth contour with the native glenoid. **(D)** Suture anchors are placed into the rim of the graft. **(E)** The limbs of each suture are passed through the capsule and labrum, thereby repairing the ligaments to the new glenoid rim.

transfer is performed through a posterior approach. The Hill-Sachs defect is débrided and the infraspinatus is released from its insertion and reattached into the base of the defect by transosseous sutures.

Postoperative rehabilitation is centered around early passive motion that avoids the position of recurrent dislocation. In anterior dislocations, the position to avoid is motion posterior to the plane of the scapula, such as abduction and external rotation. On postoperative day 1, pendulums and supine forward elevation with the arm in internal rotation are begun. After 6 weeks, the patient is allowed to begin stretching in external rotation and abduction.

Strengthening is reserved for approximately 10 to 12 weeks after surgery, and after a full stable range of motion is obtained.

Arthroscopic Repair

In 2003, Yanmis et al. presented their technique and preliminary results using arthroscopy to assist in the treatment of locked anterior dislocations of the shoulder.[42] They felt that the addition of an arthroscopic evaluation of the shoulder could aid in reduction of the shoulder, be used to clear the joint of fibrotic tissue, and assess the status of the articular surfaces. Any associated labral detachment could then be repaired arthroscopically. The success of this method of treatment is substantially dependent on the pliability of the soft tissues. Therefore, it is less likely to be successful if the dislocation has been present for more than 6 weeks. If preoperative evaluation indicates that the impaction of the humeral head involves less than 25% and that the only reconstruction that will be required is that of the detached labrum, arthroscopic management may be possible. However, advanced arthroscopic skills are required. Therefore, the applicability of this method to more than a small number of highly skilled arthroscopists is questionable.

Allograft Reconstruction

If the Hill-Sachs lesion involves between 25% and 45% of the humeral head, then allograft reconstruction can be performed.[12,41] For this technique to be successful, the patient must have good bone stock and a suitable articular cartilage. Historically, defects of this size have been addressed with infraspinatus transfer into the defect, structural bone grafting of the defect, or humeral head replacement. More recently, Gerber has reported success treating this type of defect with allograft reconstruction[12] (Fig. 15-23). He stressed that allograft reconstruction requires good bone

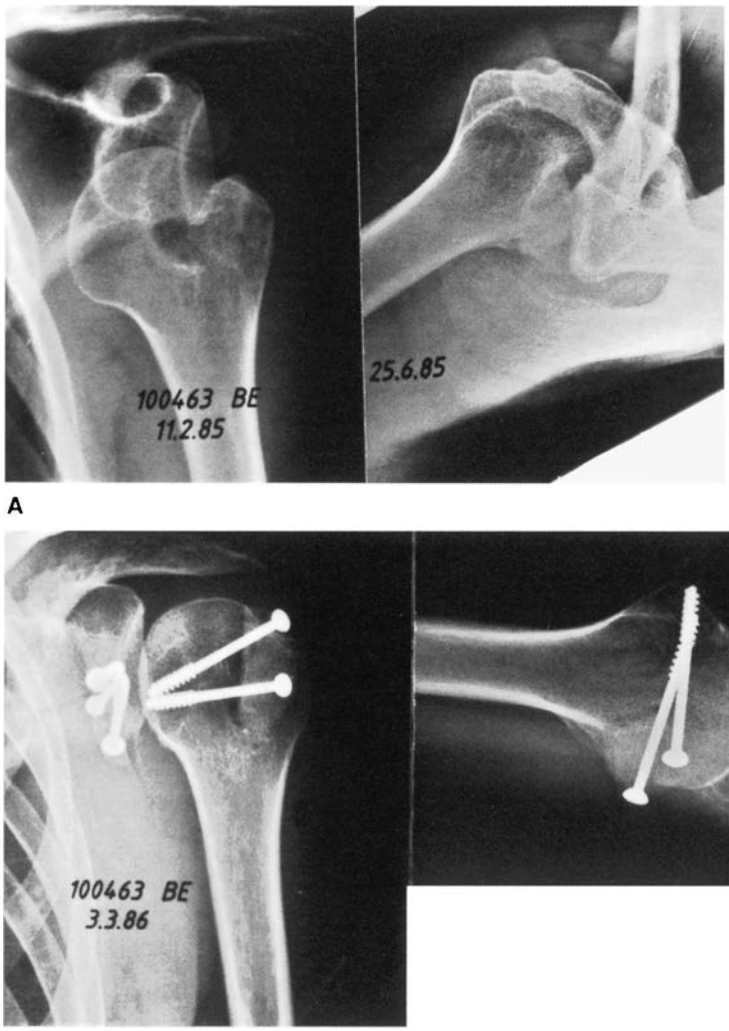


Figure 15-23 Radiographs of a patient with **(A)** a severe humeral head defect that was reconstructed with **(B)** an allograft and the glenoid augmented with a modified Bristow bone block. (From de Laat EA, Visser CP, Coene LN, Pahlplatz PV, Tavy DL. Nerve lesions in primary shoulder dislocations and humeral neck fractures. A prospective clinical and EMG study. *J Bone Joint Surg Br* 1994;76:381–383, with permission.)

stock in the remaining humeral head to prevent collapse. Dislocations of more than 6 months often have poor bone stock, and collapse of the humeral head following reduction is common.[11,33] If osteopenia is present, then humeral head replacement would be the better option in these patients.

Allograft reconstruction of a posterolateral Hill-Sachs lesion from an anterior approach requires extensive exposure. To allow access to the posterior aspect of the humeral head, the entire subscapularis insertion needs to be detached. The ascending branch of the anterior circumflex humeral artery must be protected to prevent vascular compromise to the humeral head. The capsule must then be released around its circumference. The arm can then be brought into adduction, external rotation, and extension to gain access to the posterior head. The allograft is fashioned to fit the defect and is fixed into place with either a headless compression screw such as the Herbert screw or a counter sunk 3.5-mm screw.

Postoperatively, the patient is placed in a sling. Rehabilitation consists of gentle pendulums and passive supine elevation, with avoidance of external rotation beyond 0 degrees for 6 weeks. At 6 weeks, the patient is allowed full motion of the shoulder and light strengthening is begun. No heavy lifting is allowed for 3 months.

Gerber has reported good results in a small series of patients treated with this procedure. Postoperative elevation averaged 145 degrees with a Constant score averaging 70%.[12]

Prosthetic Reconstruction

In patients with greater than 50% involvement of the articular surface of the humeral head, shoulder arthroplasty is the treatment of choice. Arthroplasty is also the treatment of choice in patients with marked osteopenia or loss of articular surface integrity. Some authors have actually reported collapse of the articular surface of the humeral head after reduction of a long-standing dislocation.[11,33] Therefore, a humeral head replacement will often be needed for dislocations that are older than 6 months.

When performing a humeral head replacement to treat a locked anterior dislocation, the surgeon should attempt to place the prosthesis in approximately 30 degrees of retroversion. If the glenoid is deficient, it must be reconstructed using autograft bone from the resected head. If the defect in the glenoid is larger than 50% of the glenoid surface or the remaining glenoid is arthritic, then placement of a glenoid component is required.

Any rotator cuff tears encountered during prosthetic replacement should be repaired.[11,33] The rotator cuff aids in stability of the shoulder and prevents superior migration of the humeral head. The repair is performed after the head is excised and before prosthetic fixation.

Flatow and colleagues reported on nine chronic anterior dislocations of the shoulder treated with total shoulder replacement, with four excellent, four satisfactory, and one unsatisfactory result.[11] The surgically treated group far outscored the nonsurgically treated group, with average active forward elevation to 147 degrees and average active external rotation to 69 degrees. All were able to function well with activities of daily living.

Some authors suggest that the version of the prosthesis can be adjusted to compensate for the chronic dislocation and aid in preventing a recurrent dislocation.[11,32] Pritchett and Clark reported a series of seven patients for whom the retroversion was increased 30 to 50 degrees, with no episodes of recurrent dislocation. All patients improved, with five good and two poor results.[32]

Postoperative rehabilitation is instituted on day 1. Gentle range of motion anterior to the plane of the scapula, with avoidance of provocative positions, is performed for 4 to 6 weeks. If the shoulder remains unstable at the end of the procedure, then the shoulder can be immobilized in a sling for 3 weeks to allow the soft tissues to scar. After 3 weeks, the sling is removed and gentle range of motion is begun. In this scenario, the position of instability is avoided for 6 weeks. No heavy activity is allowed for 3 months.

Resection of the Humeral Head

Resection is undertaken only for intractable pain in a shoulder that can not be reconstructed. In Rowe and Zarins' series, three out of four resections resulted in only fair results.[35] Although reported series are small, resection is inferior to nonoperative treatment, as far as functional outcome is concerned.

SUMMARY

Locked dislocations of the shoulder are rare injuries. Because of this, there are only a small number of series addressing the management of these difficult injuries. In this chapter, we have attempted to summarize the treatment recommendations given in the literature and the results obtained using each method. We have added to this information our own approach to the treatment of locked dislocations of the shoulder. For injuries involving less than 20% or more than 50% of the humeral head, the choice of treatment is rather straightforward. However, injuries involving 20% to 50% of the humeral head require that the surgeon understand the benefits and limitations of each of the treatments discussed. The percentages of humeral head involvement given for determining which treatment to use, as well as the time from injury listed, are only meant to serve as a guide during decision making. The surgeon must make his or her surgical choice based on the individual patient factors discussed and on the findings at the time of surgery.

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16 Complications of Instability Surgery

Mark D. Lazarus *Michael Walsh*

The only way to prevent complications is to not perform surgery.[268]

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INTRODUCTION

Rarely do we as surgeons proceed as confidently as when we operate for recurrent instability. For traumatic instability, a surgeon can confidently inform his or her patient that the patient's chances of returning to near-normal function are greater than 90%. When instability surgery fails, however, that surgical confidence can deflate as fast as a punctured balloon. Faced with the patient who has suffered multiple failures of instability surgery, we as surgeons can become thoroughly humbled. This chapter will explore the common causes of failure after instability surgery, how to avoid these failures, and techniques for recovery.

OPEN REPAIRS FOR ANTERIOR INSTABILITY

Recurrent anterior glenohumeral instability occurs most often following a significant traumatic incident and is often referred to as recurrent traumatic instability. This traumatic event usually takes place with the arm in an abducted, extended, and externally rotated position. Glenohumeral dislocation reproduced in the laboratory demonstrates a severe and explosive event, supporting the destructive pathology noted in the shoulders of these patients. As many as 84% to 97% of these patients will have avulsions of the anteroinferior capsulolabral complex from the glenoid rim (the so-called Bankart or Perthes lesion).[16,166,168,225,259] A wide array of factors can be responsible for failure of this repair (Table 16-1).

Recurrence of Instability

Incidence

The most common complication reported after repair for anterior instability is recurrence of instability.[35,98,200,221,228,294] The exact rate of instability recurrence depends on the specific surgical correction employed at the time of the index procedure, but ranges from 3% to almost 50%.[18,75,168,169]

Etiology

Numerous causes may be responsible for recurrence of instability after previous surgical repair (Table 16-2).

TABLE 16-1
CAUSES OF FAILURE OF OPEN ANTERIOR INSTABILITY REPAIRS

Recurrence of instability	Subscapularis failure
Loss of motion	Hardware complications
Capsulorrhaphy arthropathy	Neurovascular injury

TABLE 16-2
ETIOLOGY OF RECURRENT INSTABILITY AFTER ANTERIOR REPAIR

Decision-making errors	Anatomic factors
Incorrect diagnosis	Glenoid concavity defect
Incorrect surgical procedure	Residual capsular laxity
Voluntary instability	Anterior capsular deficiency
Surgical errors	Hill-Sachs lesion
Technical errors	
Other	
Severe recurrent trauma	

Although discussed individually, several of these situations may coexist in the same patient. In the patient with instability recurrence, the revision surgeon must consciously rule out each of these causes before choosing a course of action.

Incorrect Diagnosis

In an ideal world, every patient with recurrent traumatic anterior instability would present to the surgeon with a radiograph demonstrating the dislocation. In the absence of a radiograph, the patient would give a clear history of instability events directly related to positioning the arm in abduction, extension, and external rotation. Unfortunately, as is usually true in medicine, patients rarely read the textbook before giving their history. Deciding on the direction of instability, or differentiating unidirectional traumatic from multidirectional instability, can sometimes be a confusing task, and the diagnosis should be made by thorough history, examination, and radiographic analysis.

Accurate determination of the direction of instability is crucial for obtaining satisfactory results after instability surgery. Even Hippocrates recognized the importance of performing the corrective procedure at the precise location of instability and the worsening that might result from misdiagnosis:[4]

> For many physicians have burned the shoulders subject to dislocation, at the top of the shoulder, at the anterior part where the head of the humerus protrudes, and a little behind the top of the shoulder; these burnings, if the dislocation of the arm were upward, or forward, or backward, would have been properly performed; but now, when the dislocation is downward, they rather promote than prevent dislocations.

If a patient with recurrent anterior instability is incorrectly diagnosed as having posterior instability, a posterior repair would result in continued or worsened anterior instability.[18,35,101,143,168,294] Subsequent appropriate anterior repair would result in an excessively tight shoulder. Similarly, if an anterior repair is performed, but the true diagnosis

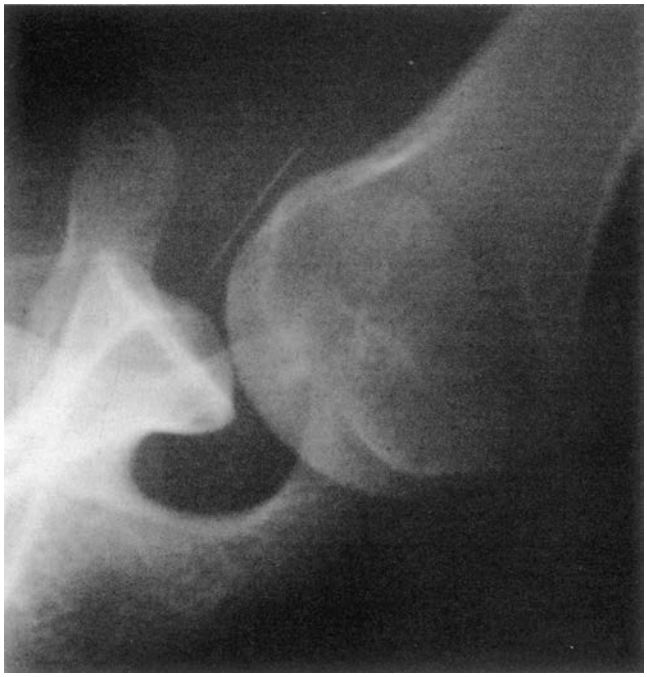


Figure 16-1 Axillary radiograph of a 24-year-old man who underwent a Putti-Platt procedure for presumed recurrent anterior instability when the true diagnosis was recurrent posterior instability. Symptomatic posterior instability was worsened by the anterior repair. (Courtesy of Douglas T. Harryman II, University of Washington Medical Center, Seattle, WA.)

is multidirectional or posterior instability, posterior displacement may be worsened (Fig. 16-1).

Hawkins and Hawkins reviewed cases of recurrent instability after surgical repair and attempted to retrospectively assign a cause to the repair failure.[98] In 12 of 31 patients, the authors believed that the primary cause of surgical failure was in the diagnosis, usually a confusion of direction of instability or type of instability. McAuliffe et al., in an analysis of 36 patients with failed instability repairs, found that 15 of those patients had recurrence secondary to misdiagnosis.[175] Norris and Bigliani determined that 9 of 42 patients with recurrent instability were incorrectly diagnosed, including four who underwent surgery on the incorrect side of the shoulder.[198] Rockwood and Gerber thought that the cause of recurrent, postoperative instability in 68% of 57 reported patients was failure to recognize multidirectional instability.[221] Finally, Burkhead and Richie analyzed 23 patients with postoperative instability and determined that, for 5 of 23, the diagnosis of multidirectional instability had been missed.[35]

Surgery for glenohumeral instability will also result in failure if the underlying diagnosis is not instability.[198] Through recent teachings, we have correctly become more alert to the potential of instability as a cause of shoulder pain in the young population. However, not all shoulder pain in the young patient indicates occult instability. Unless the diagnosis of glenohumeral instability is truly indicated by history and physical examination and is demonstrated by an examination under anesthesia, instability surgery should not be performed.

Incorrect Surgical Procedure

In most patients with recurrent anterior instability, the pathologic defect is a tear of the anteroinferior glenoid labrum and origin of the inferior glenohumeral ligament, necessitating a repair of this defect to provide stability.[16,166,168,225,259] In the literature, however, more than 150 different operative procedures have been described in the treatment of recurrent traumatic anterior instability. The "anatomic" surgical correction restores the labrum to its preinjury anatomic position. Other surgical techniques to correct instability are "nonanatomic." As a general rule, these alternative procedures do not address the inherent pathology and are associated with a greater likelihood of instability recurrence.[110,168,169] These procedures attempt to compensate for labral or capsular pathology by osseous blocks or soft tissue tightening or advancements. No attempts are made to correct labral pathology. Examples include the Putti-Platt, which shortens the subscapularis; the Bristow or Laterjet, which osteotomizes and transfers the coracoid to the glenoid rim and blocks translation; or the Magnuson-Stack, which advances the subscapularis. Despite advances in arthroscopic treatment of instability, open treatment remains a reliable method with low recurrence. It may, indeed, be the preferred treatment for pathology not adequately treated by arthroscopic means including soft tissue or bony deficiency. Although surgeries that involve bone blocks, subscapularis reefing, and musculotendinous slings may be technically easier than a Bankart repair, these procedures do not address the pathologic anatomy; therefore, they have higher rates of postoperative instability.

Rowe et al. reviewed the results of surgical correction after a prior failure of instability surgery.[228] In 84% of their patients, they found residual, unrepaired Bankart lesions at the time of revision surgery. With revision surgery, usually a Bankart repair, they were able to restore stability in 92% of patients. Hawkins and Hawkins found residual anteroinferior labral detachments in most of their patients who underwent surgery for recurrence of instability.[98]

Revising non-Bankart instability operations can often be a formidable challenge. These procedures are all usually accomplished through an anterior deltopectoral approach. If a surgeon is contemplating revision surgery when he or she did not perform the index procedure, review of the operative note from that index procedure is crucial to determine the nature of the index operation and possible difficulties revision might entail. Radiographs should also be evaluated for hardware and other hints as to the index operation.

Particular note is made of revision of procedures that involve transfer of the coracoid process (Bristow and Laterjet

operations). Coracoid transfer procedures carry historic recurrence rates ranging from 2% to 33.5%. In addition, arthritis may occur in up to 60% of patients. Subscapularis shortening secondary to dense scar formation has also been reported with subsequent loss of external rotation. Several new studies, however, have shown improved results. Hovelius et al.[109] reviewed 118 cases over 15 years and found a 3.4% recurrent rate with high patient satisfaction. Allain et al.[5] reviewed 95 Laterjet procedures over 14 years. There were no redislocations and an 88% satisfaction rate. Although nonanatomic repairs such as the Laterjet or Bristow technique have enjoyed some recent success, these techniques are not commonly performed unless severe bone loss or irreparable capsulolabral deficiency exists.

Young and Rockwood described in detail the surgical difficulties encountered in revising a failed Bristow procedure.[292] Specifically, loss of normal anatomic landmarks, extensive scar formation, and subscapularis deficiency all made surgery difficult. Matsen et al. have coined the phrase "lighthouse of the shoulder" to describe the coracoid process, meaning that the coracoid can guide a surgeon between the "safe" lateral side of the conjoined tendon and the "suicide" medial side.[166] Revision surgery without this lighthouse, as in after coracoid transfer procedures, is technically demanding and dangerous.

Surgical results after a previous Bristow operation may also be less gratifying than after other surgical procedures. For those patients with recurrent instability, Young and Rockwood obtained good or excellent results in 8 of 13.[292] Because of the associated problems of scar formation, hardware problems, and articular degeneration, the overall success rate at revision surgery was only 50%.

Technical Error

As much as we all wish to perform the perfect surgery in each and every case, there are times when the procedure does not proceed exactly as anticipated. The goal of the Bankart procedure is repair of the anterior glenoid labrum and inferior glenohumeral ligament (IGHL) to the glenoid rim. Although the surgeon may set out to perform a Bankart repair, difficulty with the exposure or unfamiliarity with the anatomy may result in the surgical goals being left unaccomplished.

It may be difficult to initially determine the cause of recurrence of instability in these cases. The index operative note may describe a Bankart-type repair as intended. With the increased use of suture anchors during Bankart-type repairs, the location of the anchors on a radiograph can be of assistance (Fig. 16-2). If the integrity of the anteroinferior glenoid labrum and IGHL are not restored, a Bankart repair was not accomplished and instability recurrence is predictable.

Technical errors leading to instability recurrence also plague non-Bankart repairs. For the Bristow procedure, correct placement of the transferred coracoid tip is essential to success.[108] If the coracoid tip is secured too medial or superior on the glenoid neck, recurrent instability would be expected. If the procedure is to be successful, the transferred coracoid process must be placed at the anteroinferior quadrant to act as a bone block against anteroinferior humeral translation. A surgeon cannot rely on the "sling" of conjoined tendon to prevent humeral subluxation.[235]

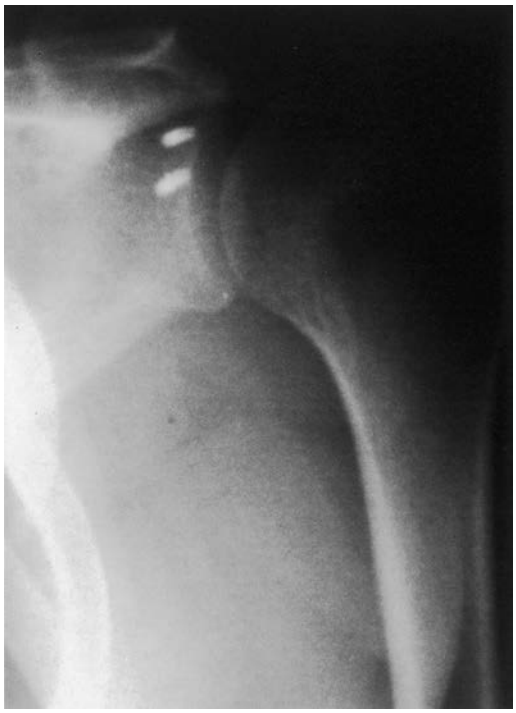


Figure 16-2 Radiograph of a 19-year-old man who had two previous "Bankart" repairs for recurrent, traumatic anterior instability. Placement of suture anchors in this superior position is unlikely to result in repair of the inferior glenohumeral ligament origin.

Failure to Restore Glenoid Concavity

The shoulder is often described as inherently unstable owing to the shallow glenoid fossa. Relative to the acetabulum, the glenoid fossa is shallow. However, just as a golf ball on a tee, the humeral head gains a great degree of stability from its position within a concave glenoid fossa, and should actually be considered inherently stable. Howell and Galinat determined the depth of the glenoid fossa to be 9 mm in the superior-to-inferior direction and 5 mm in the anteroposterior direction.[112] Half of the depth was attributed to the effect of the glenoid labrum, with the other half coming from the combined effect of the bony glenoid structure and the peripherally thickened chondral surface. This depth confers a great deal of stability to the glenohumeral articulation.

Even without ligamentous support, glenoid concavity plays a surprisingly effective role in providing glenohumeral stability. Lippitt et al. defined the *concavity–compression* mechanism of glenohumeral stability as the combined stabilizing effect of a spherical humeral head contained in

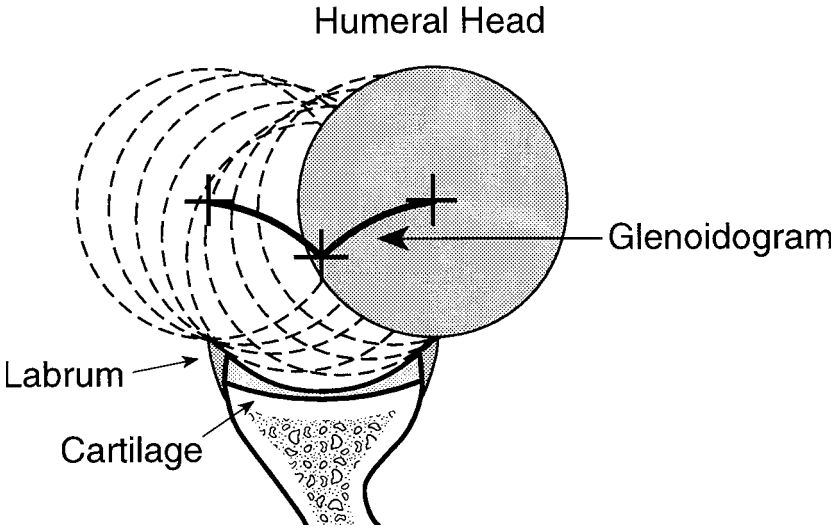


Figure 16-3 The "glenoidogram" defines the glenohumeral stabilizing effect of glenoid concavity. For the humeral head to translate anteriorly, posteriorly, or inferiorly out of the glenoid, it must initially move laterally, against the stabilizing force of the rotator cuff.

a deep glenoid fossa, held there by muscular compressive action.[154] These investigators used a graphic representation, referred to as the glenoidogram, to define glenoid depth. The glenoidogram demonstrates that lateral displacement of the humeral head is first necessary before anterior translation can occur (Fig. 16-3). They also used the *stability ratio,* a factor defined by Fukuda et al. as the humeral translating force required to cause a glenohumeral dislocation divided by the humeral compressive load maintaining stability, multiplied by 100, to analyze the effectiveness of glenoid concavity in promoting stability.[73] Normal glenoids were noted to have significant concavity, equating with an ability to provide a tremendous stabilizing effect (stability ratios as high as 63%). Excising the glenoid labrum caused a reduction in the stability ratio of approximately 20%.

To relate the concept of concavity–compression more directly to the diagnosis of recurrent traumatic anterior instability, Lazarus et al. measured glenoid concavity and stability ratios before and after creation of an anteroinferior glenoid chondral–labral defect.[144] This defect was designed to be similar to that which might be found after numerous anterior glenohumeral dislocations (Fig. 16-4). This investigation demonstrated significant reduction in the effectiveness of concavity–compression after creation of the instability defect. Glenoid concavity and stability ratios could be normalized with a simulated surgical reconstruction of the glenoid concavity (Fig. 16-5). Halder[90] confirmed previous studies that suggested that the stability ratio and effective depth of the glenoid socket

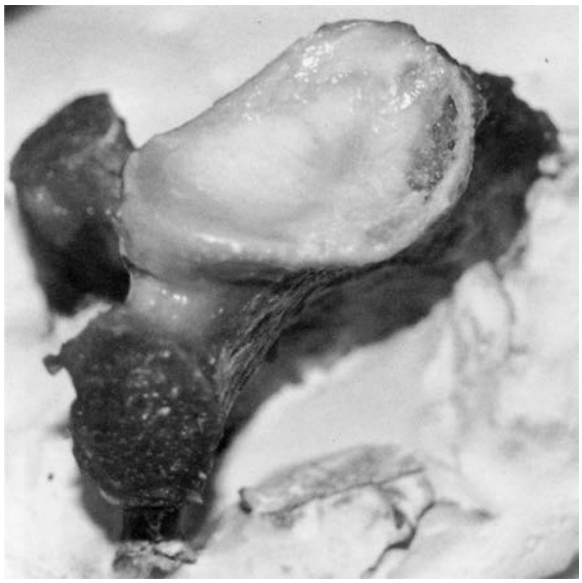


Figure 16-4 Surgically created anteroinferior glenoid labral and chondral defect to simulate the findings of glenoid concavity loss in recurrent, traumatic anterior instability.

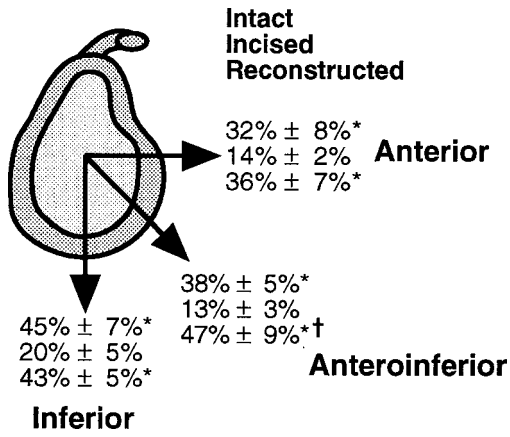


Figure 16-5 Stability ratios (humeral translating force/compressive load × 100) for intact specimens, those with surgically created anteroinferior chondral–labral defects, and those surgically reconstructed. Notice that, by restoring and even enhancing glenoid concavity, stability ratios can be corrected (*$P < 0.0001$ vs. incised, $P < 0.0001$ vs. intact).

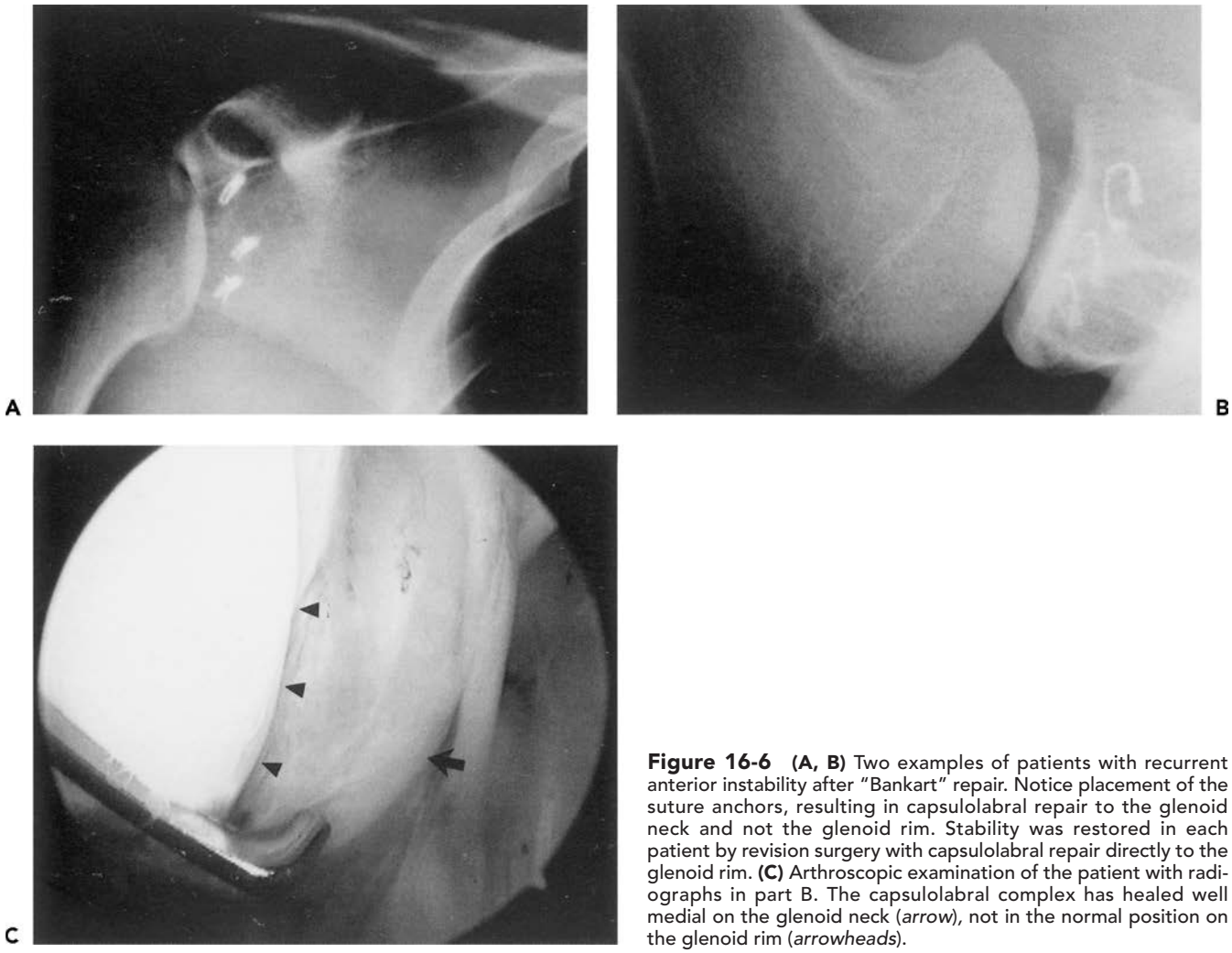


Figure 16-6 **(A, B)** Two examples of patients with recurrent anterior instability after "Bankart" repair. Notice placement of the suture anchors, resulting in capsulolabral repair to the glenoid neck and not the glenoid rim. Stability was restored in each patient by revision surgery with capsulolabral repair directly to the glenoid rim. **(C)** Arthroscopic examination of the patient with radiographs in part B. The capsulolabral complex has healed well medial on the glenoid neck (*arrow*), not in the normal position on the glenoid rim (*arrowheads*).

were linearly correlated. As compressive loads increased, the stability ratio decreased. Finally, Itoi[121] performed a cadaveric study in which osteotomy of the anterior–inferior glenoid was performed and then the Bankart lesion repaired. Stability to anterior translation was decreased significantly after the repair if the osseous lesion exceeded 21% of glenoid width. Severe external rotation loss of 25 degrees per centimeter of defect was observed.

The normal glenoid labral attachment is directly to the glenoid rim. Instability repairs that result in a capsulolabral repair to the glenoid neck and not to the glenoid rim fail to restore the normal glenoid architecture and can result in surgical failure. This type of repair is more commonly seen with the use of auxiliary fixation devices, particularly tacks, staples, and suture anchors (Fig. 16-6). Either because of their size or because of the surgeon's reluctance to place these devices near the articular surface, use of these tools can result in medial placement and subsequent inadequate repair. Although the integrity of the IGHL may be restored with this type of repair, normal glenoid concavity is diminished and the concavity–compression mechanism of stability is impaired (Fig. 16-7).

Revision surgery in cases of medial capsulolabral repair requires an attempt to restore normal glenoid anatomy and depth. Usually, the anterior capsular complex is found healed to the neck of the glenoid and requires mobilization. Care is taken to preserve the thickness of the glenoid labrum and capsular tissue. Once this tissue is freed from the glenoid neck, traditional repair to the rim of the glenoid is accomplished. For revision surgery, we recommend the technique of Thomas and Matsen.[259] A standard deltopectoral approach is used with a low axillary incision. Scar and adhesions between the overlying deltoid, acromion, and conjoined tendon and the underlying rotator cuff and humeral head are mobilized. Often these adhesions are extremely thick and require a sharp incision. Care should be taken not to be too superficial and risk injury to the axillary nerve as it travels on the deep surface of the deltoid. Scar on the superficial surface of the subscapularis should be excised only up to the lateral border

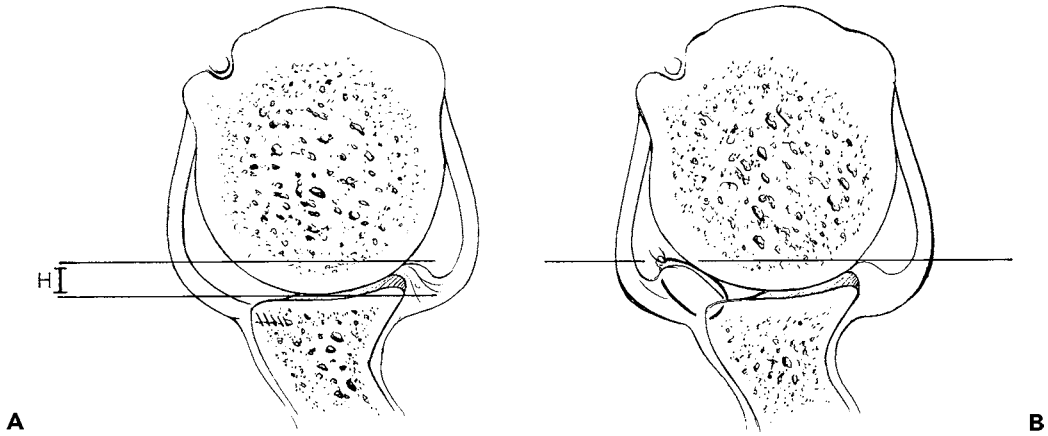


Figure 16-7 **(A)** Capsulolabral repair to the glenoid neck fails to restore normal glenoid concavity. **(B)** Capsulolabral repair to the glenoid articular margin restores normal glenoid concavity.

of the conjoined tendon. Subscapularis mobilization is completed after complete incision of the tendon.

The subscapularis and capsule are sharply incised as a single unit, approximately 1 cm medial to the lesser tuberosity. A small elevator can be passed through the rotator interval and deep to the subscapularis and capsule before incision, to gauge the combined thickness, protect the long head of the biceps, and protect the underlying humeral articular surface. After capsulotomy, the subscapularis can be completely mobilized by using traction sutures to pull the tendon laterally and cause adhesions on the undersurface of the conjoined tendon to present themselves lateral to the tendon.

Upon capsulotomy, capsulolabral repair to the glenoid neck is found. This repair is mobilized by either a small periosteal elevator or sharp dissection. The anterior, nonarticular glenoid surface is burred, using the burr to contour a new glenoid rim (Fig. 16-8A). Three to four bone tunnels are created using a 1.5-mm wire-passer burr and a 3-0 angled curette. Each of these holes is filled with a no. 5 Ethibond suture, passed with a no. 5 curved Mayo needle (Fig. 16-8B). Finally, the trailing medial edge of capsule is repaired with simple suture passes, taking care to mobilize the capsule superiorly with repair (Fig. 16-8C). The subscapularis and capsulotomy are closed end to end. We prefer an "all inside" repair performed through transosseous bone tunnels in the anterior glenoid rim because we believe this type of repair has a bunching effect on the labrum, further enhancing anterior glenoid concavity.[166,186,259] Capsulolabral repair with suture anchors can also be successful, as long as the anchors are placed directly on the glenoid rim. Typically in these situations, repair of the capsule and labrum to its true anatomic position is successful in achieving glenohumeral stability.

Postoperatively, these patients are managed as a primary instability repair. A sling is worn for 3 weeks, the patient removing the sling five times each day to perform supine active assisted forward elevation to 90 degrees and external rotation to neutral. At 3 weeks postoperative, the sling is discontinued, and full motion is permitted. At 6 weeks postoperative, resistance exercises are begun, including rotator cuff strengthening and scapular stabilization. Noncontact athletics are permitted at 3 months postoperative, with full contact at 6 months.

Larger concavity defects occur with fracture of the glenoid rim. It has been well reported that glenoid rim fractures are associated with recurrent anterior instability.[18,106,140,168] Treatment options in the face of a glenoid rim fracture include excision of the fragment and capsulolabral repair to the remaining glenoid rim versus direct internal fixation of the anterior glenoid fragment. Most authors recommend repair of the fragment if it constitutes more than 25% of the glenoid width.[18,294]

The accurate preoperative evaluation of a potential clinically significant osseous glenoid lesion is critical. Special radiographic studies, including a Garth view and West Point axillary view, should be obtained. Even with good-quality images, however, only large or distinct osseous fragments may be visible. A computed tomography (CT) scan may be needed to adequately visualize and size a glenoid rim defect. Sugaya et al.[253] used CT to evaluate 100 shoulders with recurrent unilateral glenohumeral instability and found that 90 had pathologic lesions. Fifty had osseous defects and 40 had blunting of the normal glenoid contour suggesting compression or erosion at the glenoid rim. Glenoid rim deficiency of less than 25% of the articular width can be effectively treated by simple repair of the capsulolabral complex into the defect. If the defect involves more than 25% of the glenoid articular width, however, it must be repaired or reconstructed for successful revision surgery. Failure to account for and reconstruct an anterior glenoid rim fracture can be a cause of instability recurrence after surgery[18] (Fig. 16-9A).

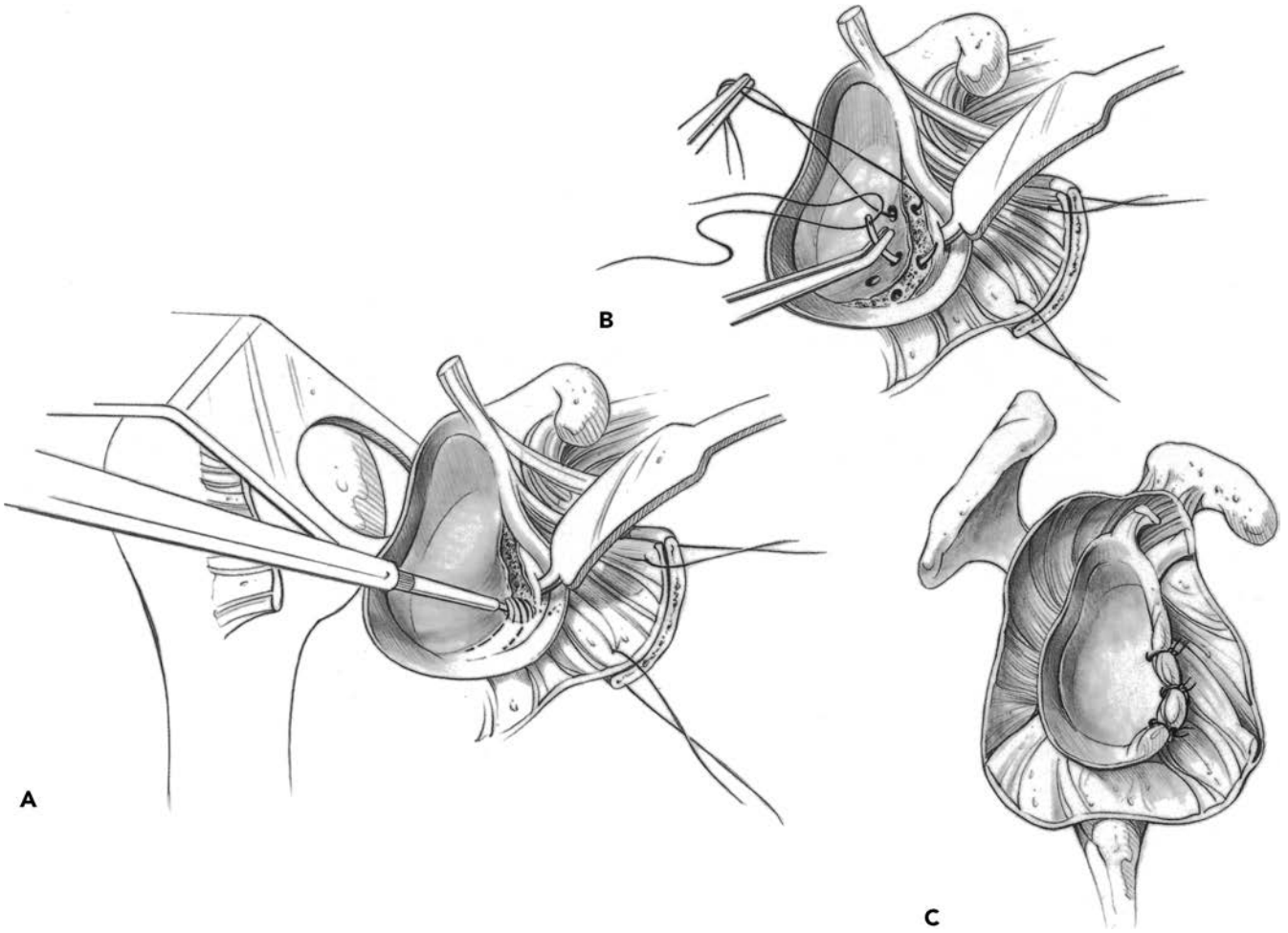


Figure 16-8 **(A)** The previous medial capsulolabral repair is elevated from the glenoid neck. A high-speed burr is then used to create a lip to the typically rounded anterior glenoid rim. **(B)** Bone tunnels are made in the glenoid rim and each receives a no. 2 braided, nonabsorbable suture. **(C)** By passing the sutures in simple fashion (all-inside technique), the labrum is bunched on the glenoid rim, restoring glenoid concavity.

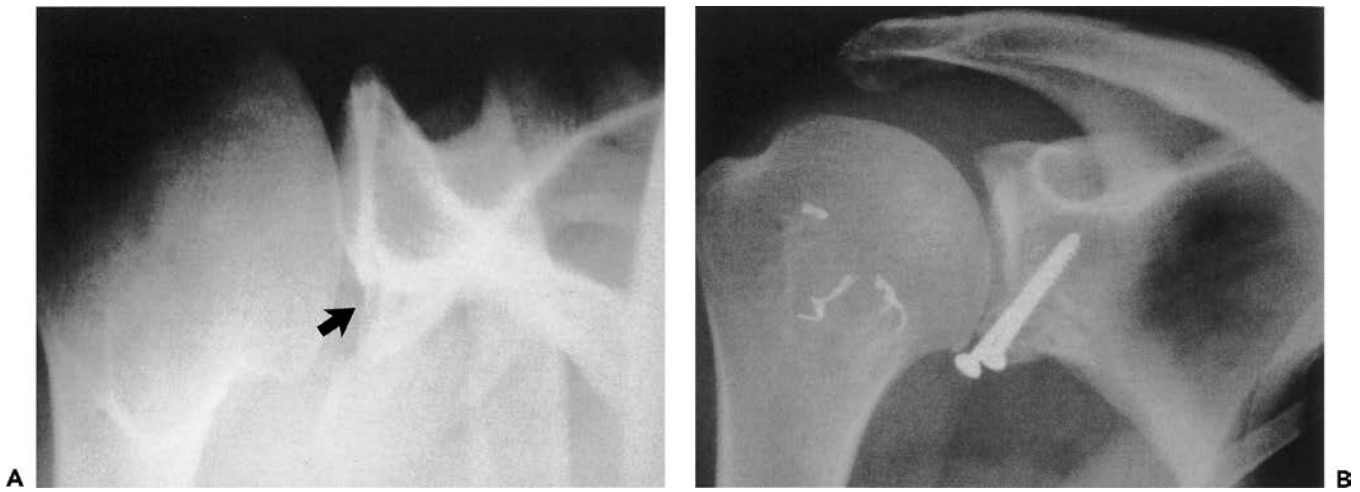


Figure 16-9 **(A)** Garth apical oblique view demonstrating a large anteroinferior glenoid osseous defect (*arrow*). **(B)** Same patient after repair of the osseous lesion. (Courtesy of Douglas T. Harryman II, University of Washington Medical Center, Seattle, WA.)

Gerber has reported work on the effect of loss of the anteroinferior glenoid rim on stability.[76] Instead of measuring the percent loss of the glenoid rim, he recommended measuring the shortest distance from the glenoid rim just superior to the defect to the intact rim at the inferior aspect of the defect. In his study, if this distance was greater than 15 mm, the stability to translational force was decreased by over 60%. More importantly, the stability could not be corrected by ignoring the osseous lesion and repairing the capsule and labrum into the defect.

If, during revision surgery, the residual bone fragment appears viable, direct internal fixation of the fragment should be performed (Fig. 16-9B). In these situations, we alter the standard Bankart approach by dividing the subscapularis and capsule as two distinct layers. The subscapularis is divided approximately 1 cm medial to the lesser tuberosity. Then, using sharp dissection or an electrocautery, the subscapularis tendon is elevated, in a medial direction, from the underlying capsule. The tendon must be freed in a medial direction past the glenoid rim fragment. The anterior capsule is then incised, allowing visualization of the glenoid articular surface. The glenoid fragment is usually found partially healed to the anterior glenoid neck and requires mobilization (Fig. 16-10A). The osseous bed is then prepared by a high-speed burr, and the fragment is repaired with bicortical lag screws, taking care to not leave the screws long posteriorly where they may injure the suprascapular nerve (Fig. 16-10B). Usually the anterior glenohumeral ligaments are attached to the osseous fragment and, therefore, are restored with union of the repair.

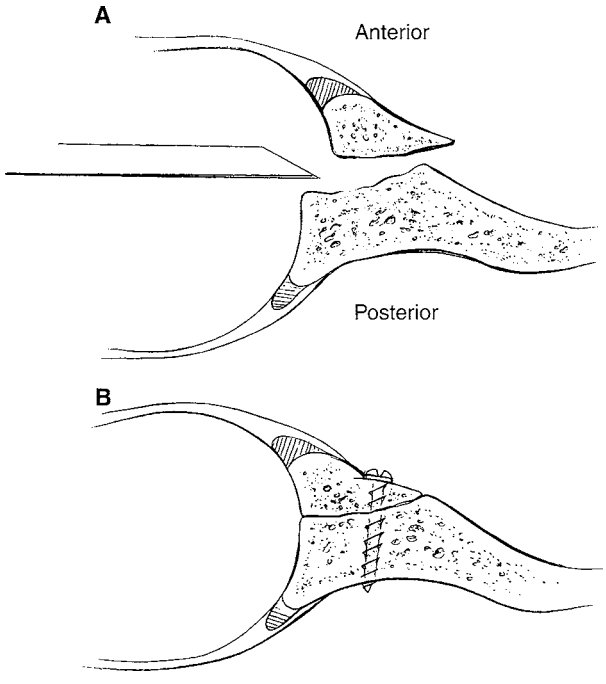


Figure 16-10 **(A)** For repair of a large osseous Bankart lesion, the bone fragment is mobilized from its partially healed position on the anterior glenoid neck. **(B)** Fixation is by two 3.5-mm bicortical lag screws or 4.0-mm cannulated screws.

If, however, the fragment has eroded or appears nonviable, the glenoid rim must be reconstructed. Gerber has discussed reconstruction of the glenoid rim by placement of autogenous iliac crest graft, secured with bicortical screws, and fashioned to match the curvature of the native glenoid.[76] In a preliminary report, he found that the technique restored stability in 12 of 13 patients, with the final patient having questionable recurrence of subluxation only. In a similar study, Gill et al.[78] reconstructed the anterior glenoid with iliac crest bone graft in 12 patients with CT-confirmed anterior glenoid bone loss. There was no recurrence in any patient at average 2-year follow-up. The only patients who developed progressive degenerative change were those who had some degree of degeneration preoperatively. Other authors echo these results. Bodey and Denham,[27] Haaker et al.,[89] and Hutchinson et al.[116] all reported high patient satisfaction with a low recurrence rate for iliac crest grafting. Warner[273] reported on 12 patients with anterior–inferior glenoid deficiency who underwent intraarticular reconstruction of the anterior–inferior glenoid utilizing tricortical iliac crest autograft and capsular shift. At follow-ups ranging from 24 to 61 months, there were no reported cases of instability and CT confirmed osseous union absent arthritic changes.

Our preference in these difficult situations is to use autogenous bone to fashion a graft for the rim, similar to Gerber. The surgical approach is as described earlier. The remaining atrophic glenoid fragment is excised, taking care to preserve the full length of the anterior capsuloligamentous structures. A pine cone burr is used to freshen and prepare the bed. A tricortical graft is harvested from the iliac crest. With a pine cone burr, the graft is fashioned to approximately conform to the curvature of the glenoid. If sufficient capsular length exists to place the graft in an extracapsular position, sutures of no. 2 Polydek are placed under the graft for later capsulolabral repair (Fig. 16-11A). The graft is then secured with bicortical lag screws, taking care to closely observe the articular surface for screw penetration and to not leave the screws long posteriorly where they may irritate the suprascapular nerve. Finally, the pine cone burr is again used to complete fashioning of the graft in situ (Fig. 16-11B). With use of the previously placed sutures, the capsule is repaired directly to the native glenoid rim, leaving the bone graft in an extracapsular position (Figs. 16-11C and 16-12). If capsular repair to the host glenoid–graft junction will result in excessive loss of external rotation, drill holes are placed in the rim of the graft and the capsulolabral complex is repaired to the rim of the graft. The subscapularis and capsule are then closed side to side. The postoperative regimen is as previously described for Bankart repair.

An alternative to bone grafting is a Bristow or Laterjet coracoid transfer. Either of these procedures would be technically easier than bone grafting of the anterior glenoid

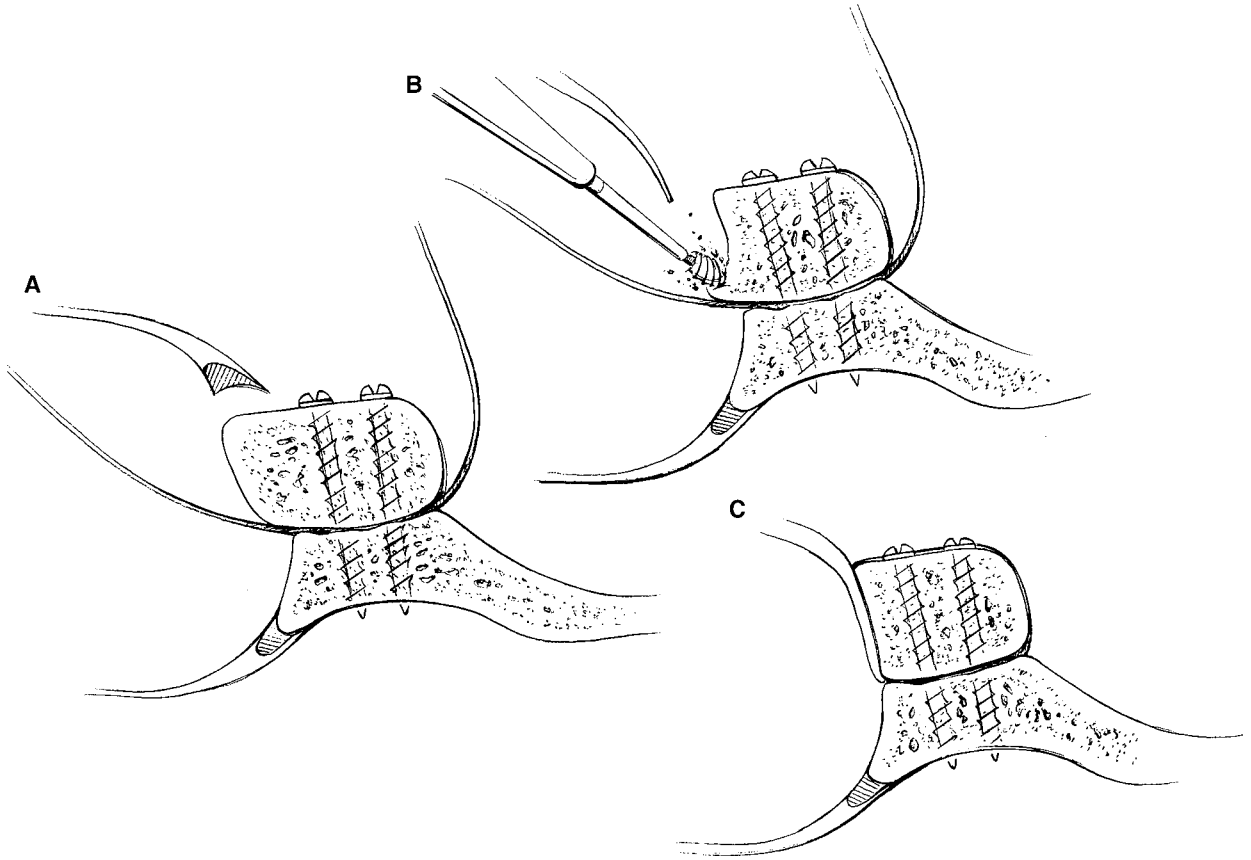


Figure 16-11 **(A)** For a large osseous Bankart lesion that requires grafting, a tricortical graft is taken from the iliac crest and applied to a prepared bed on the anterior glenoid neck. Sutures are placed under the graft for later capsular repair. **(B)** The graft is burred to conform to the curvature of the normal glenoid. **(C)** When burring is completed, the anterior capsule is repaired over the graft using the previously placed sutures. This repair leaves the graft in an extracapsular, supportive position.

rim. Coracoid transfer procedures, however, provide only a block to dislocation and do not reestablish the glenoid curvature, thereby leaving the patient susceptible to continued anterior subluxation.

Finally, an osteochondral allograft can be used to reconstruct large anteroinferior glenoid defects. Swarm and Lazarus[254] reported on the use of this procedure in two patients, successfully eliminating instability in both (Fig. 16-13). Advantages of allograft over autogenous iliac crest graft include the ability to near anatomically size the glenoid rim, lack of donor-site morbidity, the presence of a smoother articulating surface, and the presence of labral and capsular tissue on the graft to assist in the reconstruction of combined osseous, chondral, labral, and capsular deficiencies. Clearly, the disadvantages are the theoretical possibility of disease transmission and the cost.

Residual, Abnormal Anteroinferior Capsular Laxity

In the science of shoulder instability, there is currently ongoing debate over the presence or absence of plastic deformation of the IGHL in patients with recurrent traumatic anterior instability and, if present, its clinical significance. Bigliani et al. demonstrated plastic deformation of the IGHL before the ultimate failure by avulsion from the glenoid rim during stress to failure of the IGHL complex.[24] Reeves had previously confirmed these findings in older cadaveric shoulders, but found isolated capsular avulsion without plastic deformation in shoulders of younger cadavers, within the age group most likely to suffer recurrent traumatic anterior instability.[214] Speer et al.[246] studied cadavers with simulated Bankart lesions and found increased translation anteriorly and inferiorly that were small, averaging only 3.5 mm. The authors concluded that some degree of capsular injury must occur for instability recurrence.

On the contrary, McMahon et al. presented work that demonstrated capsulolabral avulsion in 8 of 12 cadaveric shoulders when the application of stress was performed with the arm positioned in the critical abducted and externally rotated position.[182] More importantly, the amount of plastic deformation of the anterior capsule was only 2.3 mm, a finding of questionable clinical significance.

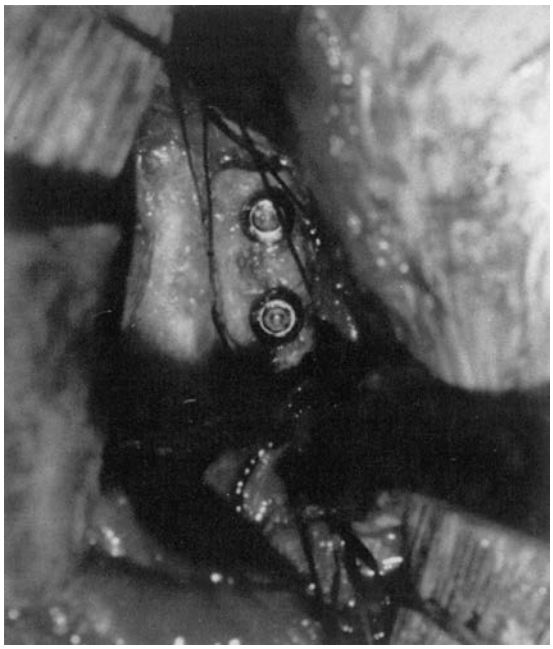


Figure 16-12 Intraoperative photograph of autogenous iliac crest graft used to reconstruct an anterior osseous glenoid deficiency. Note placement of sutures under the graft for later capsulolabral repair. (Courtesy of Douglas T. Harryman II, University of Washington Medical Center, Seattle, WA.)

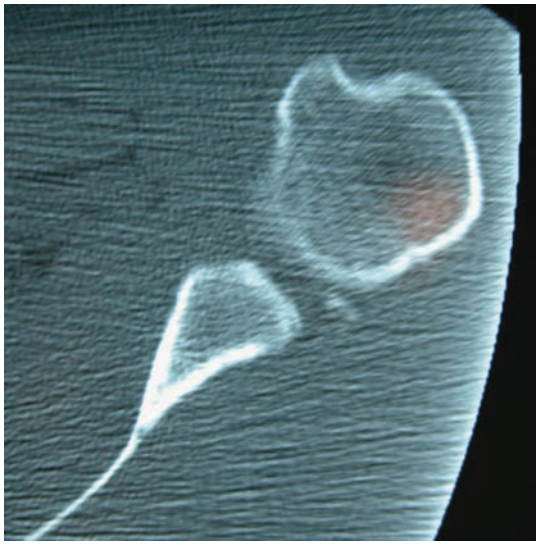
These studies leave in question the occurrence of stretch of the IGHL as a component of recurrent traumatic anterior instability. In addition, if such plastic deformation was confirmed in the laboratory, the clinical significance is still unclear. For instance, a period of immobilization after initial dislocation might be expected to lead to scar and contracture of interstitial IGHL injury, not lengthening. Also, if a concomitant Bankart lesion was present, repeated dislocations would be expected to occur with less stress applied to the interstitial fibers of the IGHL.

Several authors have blamed residual capsular laxity as the cause of recurrent anterior instability after Bankart repair.[35,98,175,221,228,282,292,294] At the time of revision surgery, the presence of a redundant or patulous anteroinferior capsule was noted by these investigators. This finding was purely observational and no specific measurements were recorded. Rowe et al. described a method whereby the arm is placed in adduction and in 90 degrees of external rotation and the anteroinferior capsule is grabbed with a clamp and pulled upward.[228,294] If the capsule can be mobilized greater than 1 cm, this indicates residual capsular laxity. All of these authors described some type of capsular shifting or imbricating procedure as an integral component of the revision repair.

The difficulty in judging residual capsular redundancy, however, is differentiating capsular stretch from normal capsular laxity. As discussed previously, Speer et al. found only minor increases in laxity after creation of a Bankart lesion.[246] This result seems to indicate that plastic deformation of the IGHL is a necessary component of recurrent traumatic anterior instability. Laxity and instability, however, are two properties that are not only different, but also they may not be related. Laxity is defined as the amount of movement of the humeral head across the glenoid fossa in response to a small applied translational force (for instance, the force applied by an examiner during a drawer or sulcus test). The maximum amount of humeral translation in a given direction is a shoulder's *laxity.* Glenohumeral *instability* is the inability to keep the humeral head centered within the glenoid fossa. Lippitt et al., performing in vivo measurements of laxity in shoulders of normal subjects and those of patients with recurrent instability, failed to demonstrate differences in laxity between these groups[152] (Fig. 16-14). Hawkins et al., who used radiographic examination under anesthesia in normal subjects and those with unstable shoulders, also found significant overlap in laxity between the two groups.[112] Finally, Sperber and Wredmark found no differences in intracapsular volume or capsular elasticity in unstable shoulders when compared with normal shoulders.[278] Therefore, whether or not residual capsular laxity exists as a cause of postoperative instability remains controversial. Moreover, the exact method by which a surgeon can intraoperatively differentiate residual capsular redundancy from normal capsular laxity is equally unclear.

Finally, glenohumeral dislocation under load does not occur as a gradual event, as replicated by measuring translational laxity, but occurs as a sudden and explosive event, with minimal translation before the moment of sudden dislocation (Fig. 16-15). The absence of significant translation before the moment of dislocation implies that glenohumeral articular congruity and glenoid concavity are the major determinants of glenohumeral stability, with ligamentous integrity serving as the final checkrein against dislocation.[61,144,154,166,245] The clinical significance of relating laxity measurements to glenohumeral instability, therefore, is unknown.

Most cases of recurrent traumatic anterior instability are attributable to the presence of an anteroinferior capsulolabral avulsion from the glenoid rim and are successfully managed by repair of this defect without any further capsular tensioning.[16,166,168,169,259] There are situations, however, for which true and demonstrable clinical instability exists, yet either a Bankart tear is not present, the tear appears too small to account for the patient's degree of instability, or there are obvious findings of capsular injury (Fig. 16-16). In these cases, some plication or shifting of the anteroinferior capsule may be necessary to restore stability. However, considering the unavoidable capsular tightening that occurs simply by repairing a Bankart lesion and side-to-side closure of a capsulotomy, combined with the typical postoperative scarring and contracture of the anterior soft tissues, residual or unrepaired anterior capsular laxity as a cause of postoperative instability should be considered a diagnosis of exclusion.



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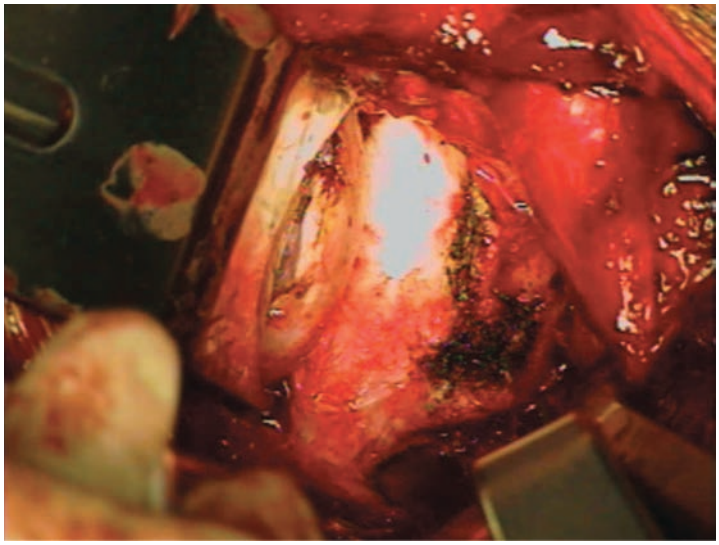

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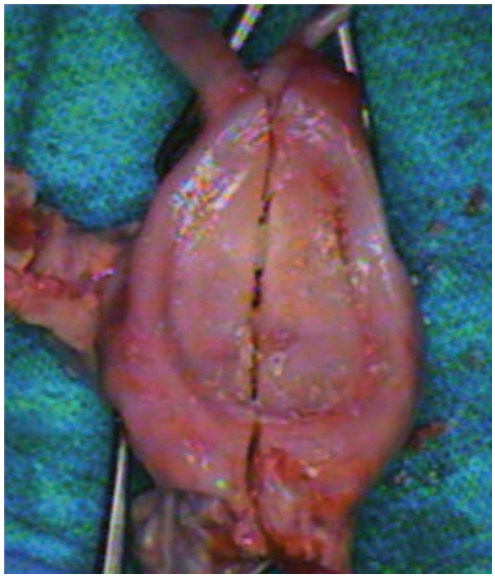

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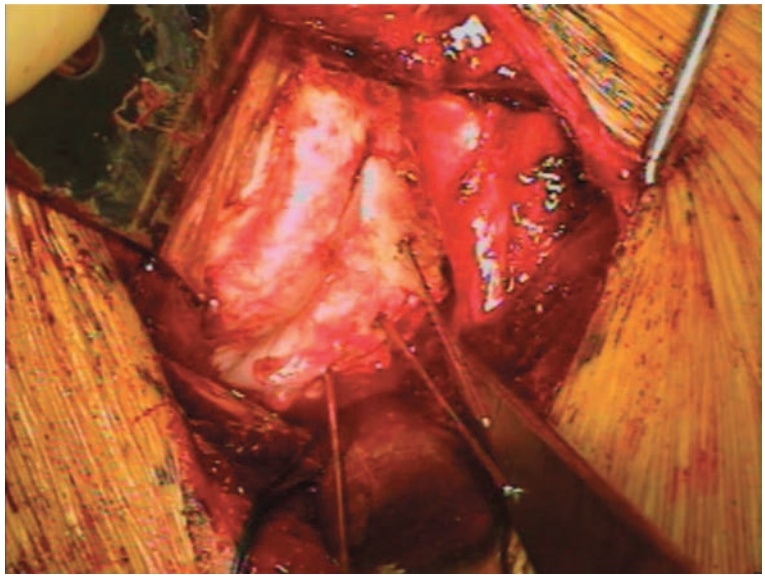

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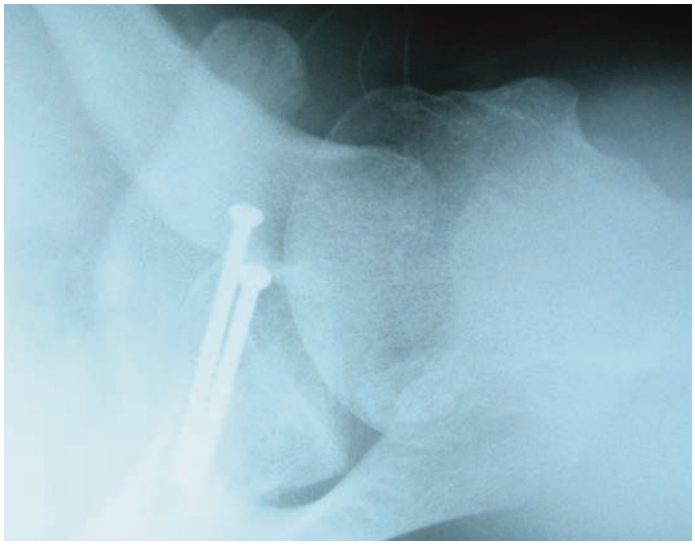

E

Figure 16-13 CT scan **(A)** and intraoperative photograph **(B)** of a patient with recurrent anterior instability after three prior stabilization attempts. Notice the large anteroinferior osseous defect. Stabilization was achieved with use of a glenoid osteochondral allograft **(C)**. Intraoperative photograph **(D)** and postoperative radiograph **(E)** after graft placement.

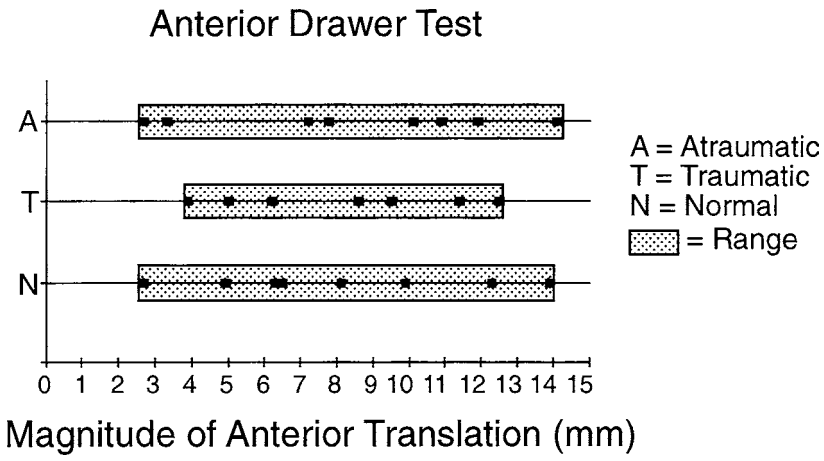

Figure 16-14 In vivo laxity to anterior drawer test in normal subjects and patients with either traumatic or atraumatic instability. Notice the significant overlap in laxity between these groups. (Permission from Lippincott Publishers, Lippitt SB, Harris SL, Harryman DT II, Sidles JA, Matsen FA III. In vivo quantification of the laxity of normal and unstable glenohumeral joints. *J Shoulder Elbow Surg* 1994;3:215–223.)

Residual capsular laxity as a cause of recurrence of instability after surgical repair should be considered in the following situations:

1. When true and clinically demonstrable glenohumeral instability was present at the time of the index operation and either no or a small Bankart tear was noted at entrance into the glenohumeral joint
2. When an appropriate and technically correct Bankart repair was performed at the time of the index operation and, at revision surgery, that repair is still intact despite true and clinically demonstrable instability
3. When obvious and large differences in translation on anterior drawer testing compared with the patient's contralateral shoulder are present in the absence of a large Bankart tear
4. When either no or a small Bankart tear is present in the patient with a history of repetitive microtrauma, such as throwing, swimming, or gymnastics

When other causes of instability recurrence are ruled out and one or more of the previously mentioned conditions exist, stability can be restored by an anteroinferior capsular shift.[7,168,282] There are several descriptions in the literature on the technique of capsular shift, and the exact procedure is not critical (see Chapter 12). What is important is to contract the excessively lax anterior and inferior capsule without overtightening the shoulder.

Anterior Capsular Deficiency

One reason for the wonderful results after primary instability surgery is tissue quality. Typically, patients requiring

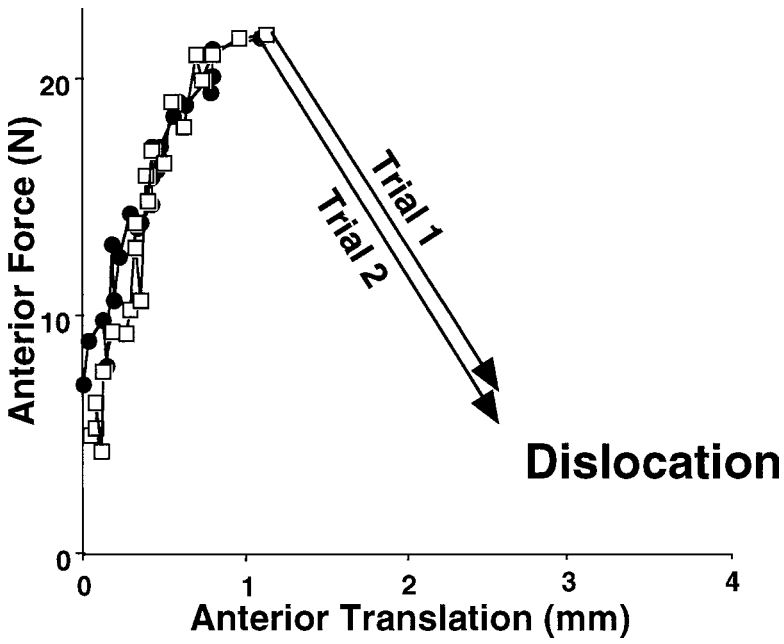

Figure 16-15 Translation before dislocation in a loaded, cadaveric shoulder. Even in an older cadaveric shoulder, there is minimal humeral translation before the explosive moment of dislocation. (Permission from JBJS, ref. 81.)

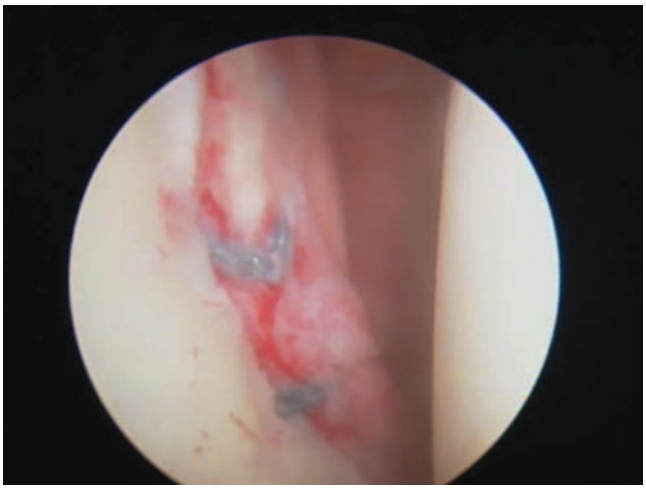

Figure 16-16 Arthroscopic image of a patient who suffered recurrent anterior dislocations after a prior arthroscopic Bankart repair. The labrum was completely healed but capsular disruption occurred lateral to the labral attachment.

surgery for recurrent traumatic anterior instability have thick, healthy anterior capsular tissue that permits a robust repair. After repeated attempts at anterior repair, however, the anterior glenohumeral capsule may become a thin, attenuated remnant, incapable of being repaired. Preoperative workup may reveal subscapularis deficiency in these individuals (discussed later). In these difficult cases, the surgeon needs both a specific plan of attack as well as availability of alternatives to the usual capsuloligamentous repair.

Revision surgery begins by defining the humeroscapular motion interface, that plane between the underlying rotator cuff and the overlying deltoid, acromion, and conjoined tendon.[166] Initially, the surgeon may be fooled by the intact and often thickened clavipectoral fascia. However, this fascia will not move as one with the humerus during humeral rotation, being clearly differentiated from the subscapularis. All scar and adhesions within this interface are excised and the axillary and musculocutaneous nerves are located. At this juncture, the amount and quality of the subscapularis and anterior capsule can be assessed. A Joker or Freer elevator can be passed through the rotator interval and deep to the combined anterior capsule and subscapularis to gauge the combined thickness of these structures.

Occasionally, when incising the clavipectoral fascia, it instantly becomes clear that the anterior capsule and subscapularis are completely deficient. In these situations, reconstruction of the capsule is necessary. Capsular reconstruction begins by identifying the interval between deficient and normal capsule. Typically, the posteroinferior capsule is normally present. By performing a limited posteroinferior capsular shift, this normal posteroinferior capsule can be mobilized to reconstruct the anteroinferior quadrant.

Gallie and LeMesurier recommended use of fascia lata to reconstruct the anterior glenohumeral capsule as a treatment for recurrent instability.[67] Iannotti et al.[117] studied seven patients with recurrence of anterior instability due to capsular deficiency treated with capsular reconstruction using 2-cm strips of the Iliotibial band folded on one another. The most superior strip recreated the rotator interval, while the middle and inferior limbs recreated the middle glenohumeral ligament and anterior–inferior glenohumeral ligament, respectively. Bony glenoid defects were excluded. At 45-month average follow-up, there were no reports of recurrent instability and range of motion was maintained. Lazarus and Harryman described use of semitendinosus allograft to reconstruct the superior (SGHL) and middle (MGHL) glenohumeral ligaments[143] (Fig. 16-17). Using a pine cone burr (4 mm in diameter), holes are created in the glenoid and the humeral head, these holes corresponding to the origin and insertion sites of the SGHL and MGHL. A single strip of autograft tendon is passed from lateral to medial to recreate the SGHL and then from medial to lateral to reconstruct the MGHL. With the humeral head reduced and the arm in neutral rotation, the reconstruction is tightened. Patients must be aware that they will have permanent motion restrictions, sacrificing normal motion range for stability. Postoperatively, a sling is placed for 3 weeks, with abduction and external rotation isometrics being the only exercise program. After 3 weeks, the sling is removed three to five times each day for gentle supine active assisted forward elevation and external rotation exercises. Particularly with external rotation, patients are shown the neutral rotation point and are cautioned against attempting to stretch beyond this range. At 6 weeks postoperative, the sling is discontinued, and a gentle rotator cuff and scapular strengthening program is begun. No lifting of greater than 10 lb (4.5 kg) is permitted for 6 months. In addition, patients with heavy-labor occupations or those involved in contact sports are encouraged to discontinue these activities permanently. If necessary, the graft can also be used to reconstruct a deficient labrum and capsule simultaneously (Fig. 16-18). Lazarus and Harryman reported use of this procedure in 17 patients with capsular insufficiency and recurrent, disabling instability after multiple attempts at anterior stabilization. In 70% of cases, the procedure was successful in eliminating instability.[143] Thorough knowledge of the scapular and humeral attachments of the superior, middle, and anterior band of the inferior glenohumeral ligaments is essential to the success of this reconstruction.[264]

Hill-Sachs Deformity

Posterolateral osteochondral impression fracture of the humeral head, the so-called Hill-Sachs deformity, is a common finding in patients with recurrent instability.[107,111,168,169,275] The role of this defect in causing continued anterior instability after surgical repair, however, is

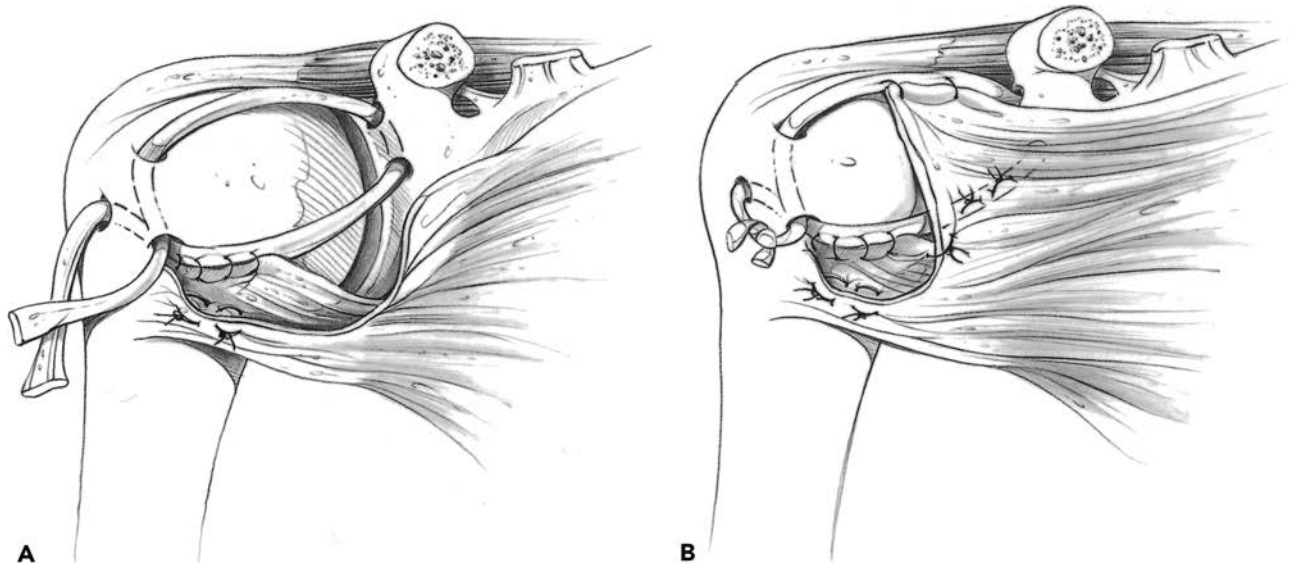

Figure 16-17 **(A)** Technique to reconstruct severe loss of anterior subscapularis and capsular tissue. The posteroinferior capsule is shifted to reconstruct the anteroinferior quadrant. Autogenous tendon graft is then woven through drill holes in the glenoid and humerus. This graft recreates the superior and middle glenohumeral ligaments. **(B)** The graft is secured with the glenohumeral joint located and the arm in neutral rotation. Remnant subscapularis tendon can be secured to the shifted capsule and the tendinous autograft.

unclear. Rowe et al. found Hill-Sachs lesions in 76% of shoulders that had recurrence of instability after surgical repair.[231] Previously, Rowe and colleagues had noted a slightly higher incidence of redislocation in those patients who had moderate or severe Hill-Sachs deformities.[228]

It is rare for a humeral head defect to play an isolated role in recurrent anterior instability. The combination of a large humeral head defect and a glenoid rim defect, however, dramatically decreases the amount of humeral rotation necessary to engage the osteochondral lesion onto the anterior glenoid rim, potentially leading to glenohumeral dislocation (Fig. 16-19). A surgeon who is considering the effect of a Hill-Sachs lesion should obtain a CT scan preoperatively to measure the dimensions of the defect to help select the most appropriate surgical option.

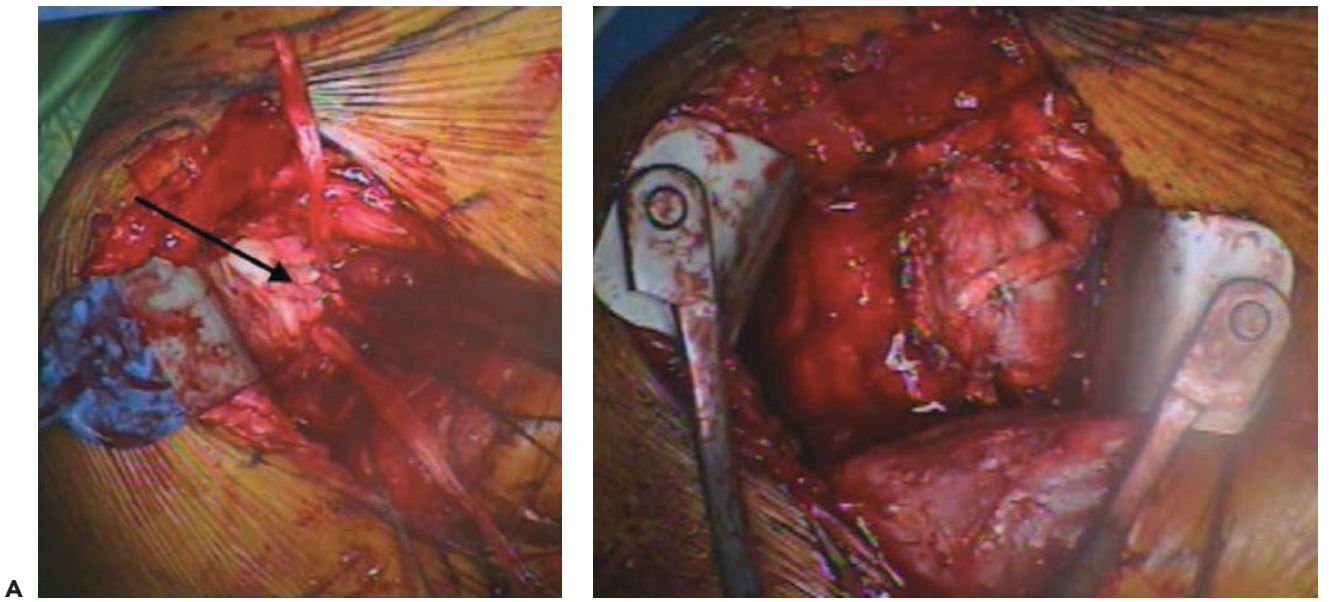

Figure 16-18 A semitendinosus graft used to reconstruct a deficient labrum **(A)** and anterior capsule **(B).**

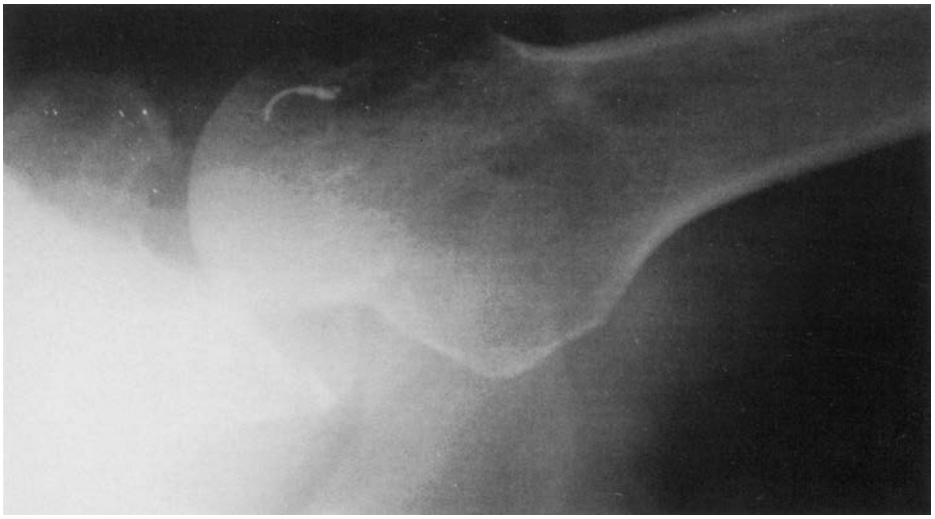

Figure 16-19 Axillary radiograph of a 26-year-old man with continued anterior instability after a Bankart repair and capsular shift procedure. The combination of a large Hill-Sachs deformity and osseous Bankart lesion results in contact of the Hill-Sachs lesion with the glenoid rim with minimal glenohumeral rotation.

At surgery, when a posterior Hill-Sachs lesion comes in contact with the anterior glenoid rim before terminal external rotation, several solutions exist. The simplest and often the most effective answer is to limit the patient's external rotation by a slight imbrication of the capsule or subscapularis. Surprisingly little decrease in external rotation is required to exclude the Hill-Sachs lesion from contact. Each centimeter of capsular or tendinous shortening diminishes humeral rotation by approximately 20 degrees.[94]

Another method to account for a large Hill-Sachs lesion is to render the defect extraarticular. Connolly has reported transfer of the infraspinatus with a portion of the greater tuberosity into the defect, thereby leaving the lesion extraarticular.[43] Subsequently, with humeral external rotation, the lesion can no longer contact the anterior glenoid rim.

Humeral osteotomy has also been described as a method of treating a large Hill-Sachs defect. Weber et al.[3] have reported humeral rotational osteotomy as a treatment for severe posterolateral humeral head defects, the goal being to rotate the Hill-Sachs lesion away from potential glenoid contact.[303] They reviewed 207 rotational osteotomies performed for large (greater than 4 cm) defects of the humeral head. They reported a redislocation rate of 5.7% after this procedure, but it is important to recognize that they include subscapularis shortening as part of their technique.

On rare occasions, the posterolateral humeral defect will be so severe that it involves more than 40% of the articular surface of the head. In this situation, the lesion will contact the anterior glenoid with minimal external rotation. To preserve any glenohumeral external rotation while providing stability, some extension of the humeral articular surface is required. Gerber and Lambert have reported on the successful treatment of a reverse Hill-Sachs deformity with placement of an osteochondral allograft into the defect.[75] We have had some success in the treatment of large Hill-Sachs deformities by reconstruction of the defect using an osteochondral femoral head allograft (Fig. 16-20). Miniaci et al.[205] reported on 18 patients treated with allograft for engaging Hill-Sachs lesions greater than 25% of the articular surface. Simultaneous capsulolabral repairs were undertaken. At a mean of 50 months postoperatively there were no redislocations, and two patients experienced partial graft collapse requiring hardware removal.

In rare cases, a humeral hemiarthroplasty or total shoulder arthroplasty (TSA) is indicated for older patients with more than 45% of the articular surface damaged or concomitant glenoid arthritis. Flatow[65] studied nine patients treated with TSA for large posterolateral defects of the humeral head and found that eight of nine had satisfactory results.

Voluntary Instability

In 1973, Rowe et al. published their comprehensive and classic description of voluntary glenohumeral instability.[227] In that study, patients with underlying psychiatric dysfunction did poorly with all types of treatment. For most patients, a rehabilitation program for muscle strength and coordination was recommended.

In any patient for whom multiple surgical reconstructions have failed to provide stability, voluntary instability should be considered. It is important to recognize, however, that having the ability to volitionally subluxate or dislocate the shoulder does not necessarily place a patient in this category.[63,100,267] Wall and Warren separated volitional instability into positional and muscular types.[267] The positional group can cause an instability event by arm movement. The muscular group can cause instability by simple muscle contraction. Surgery in this second group is unlikely to be successful. Neer has long discussed the difference between the patient who can cause an instability event and the one who either has great secondary gain in the event or who has a desire to cause instability.[195,267] It is in this second group that surgery should be avoided.

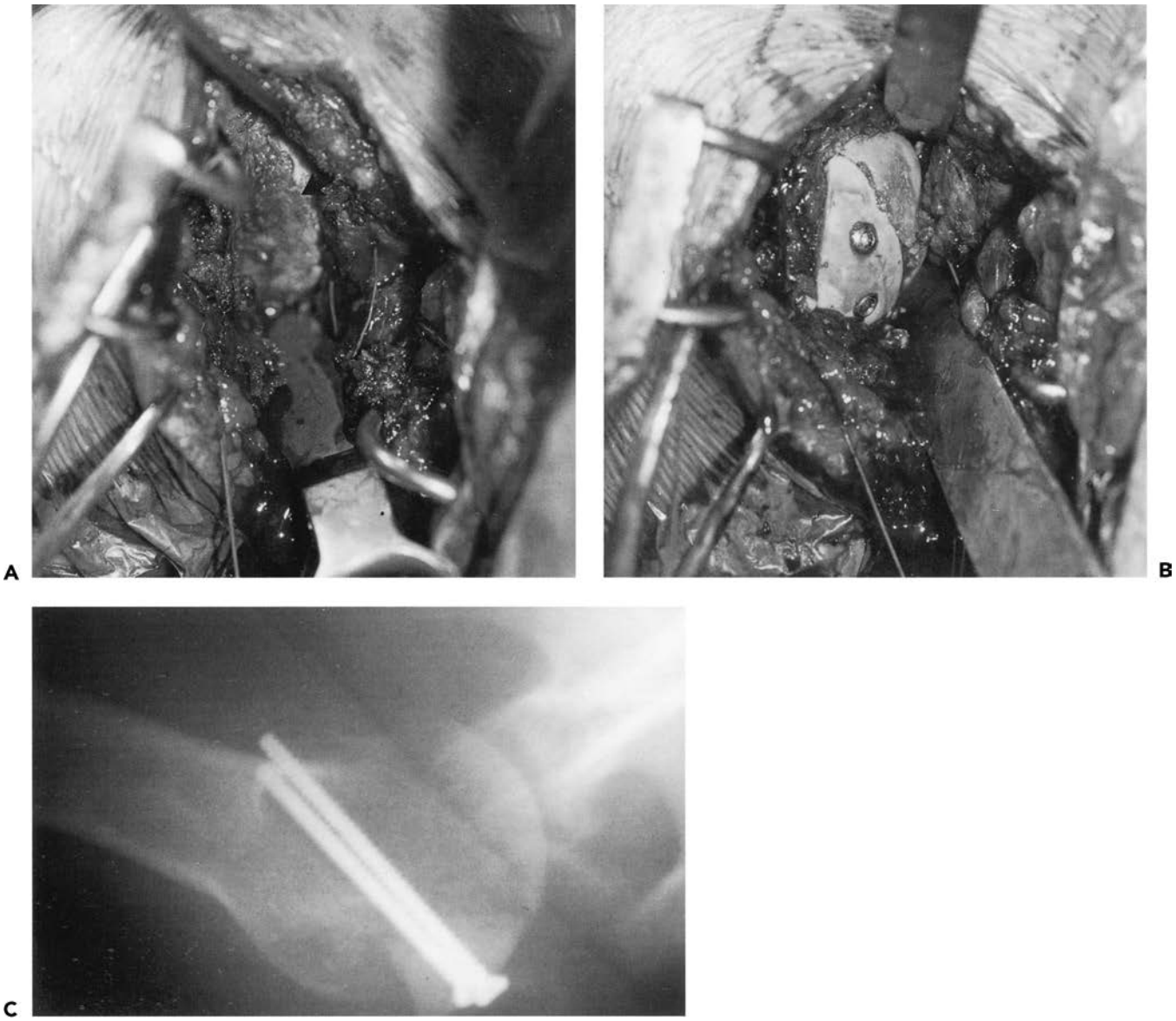

Figure 16-20 **(A)** Intraoperative photograph of a large Hill-Sachs lesion (*arrow*) seen through a posterior approach. The normal humeral articular surface (*medial*) is to the right. **(B)** Reconstruction of the Hill-Sachs lesion with placement of an osteochondral allograft. **(C)** Postoperative radiograph demonstrating extension of the humeral articular surface by the allograft.

Severe Traumatic Event

The literature is somewhat unclear about the role of trauma in causing failure of an instability repair. Some investigators have found recurrent trauma in the face of an adequate index repair to be a significant cause of instability after repair.[35,98] Others have determined that failure of instability repair typically occurs secondary to a failure of diagnosis or technique, not secondary to trauma.[175,228]

Clearly, if significant force is applied to the hand or arm while in a vulnerable abducted, extended, and externally rotated position, failure of even the strongest of repairs can occur. Trauma as the sole cause of failure, however, should be a diagnosis of exclusion. Only when misdiagnosis and persistent structural pathology are ruled out can significant trauma be blamed for instability recurrence.

Loss of Motion

Possibly the most common nonphysiologic outcome after surgery for recurrent traumatic instability, but infrequently reported as an actual complication, is loss of motion. In fact, limiting external rotation to a certain value is often stated as a desired surgical goal.[168] Often, some imbrication or "shifting" of the anterior capsule is recommended to correct presumed plastic deformation. If, however, a normal length of fibrotic, shortened capsule

is imbricated at the time of Bankart repair, motion restriction will occur.

Limitation of a patient's normal, physiologic glenohumeral external rotation may have serious consequences. Minor deficits in external rotation can lead to a decrease in a patient's ability to assume the late-cocking position and, therefore, can decrease the velocity of a throw or serve.[104,157,262] Major reductions in external rotation may not only limit functional activities, but can have disastrous consequences. Harryman et al. described obligate humeral translation, whereby the tightened anterior capsule at terminal external rotation forces the humeral head to translate posteriorly.[93] Hawkins and Angelo reported on seven patients in whom previous Putti-Platt procedures with overtightening of the anterior structures resulted in glenohumeral arthritis.[96] When the anterior structures are too tight, obligate humeral translation occurs with minimal external rotation, forcing the humeral head in a posterior direction and creating a shear force on the posterior glenoid, resulting in articular degeneration. This degenerative process, which Matsen has referred to as capsulorrhaphy arthropathy, is not only related to the Putti-Platt procedure, but can also be seen in any patient who has nonphysiologic, asymmetrical tightening of the capsule or rotator cuff tendon unit.[166]

The treatment for an excessively tight anterior capsulorrhaphy is to reverse the altered biomechanics. Specifically, abnormally shortened anterior soft tissues must be lengthened. If the original surgical procedure involved an imbrication or transfer of the subscapularis, a subscapularis release can be performed as described by MacDonald et al.[162] The technique begins with release of the subscapularis from its insertion (original or transferred). The humerus is then externally rotated to the desired position. With external rotation, the subscapularis tendon is drawn medially. At the point of desired external rotation, the subscapularis tendon is repaired to the underlying glenohumeral capsule.

If capsular contracture is also anticipated or if greater than 20 degrees of rotational correction is necessary, a coronal Z-plasty of the combined subscapularis and anterior capsule is required.[166,195] The technique is begun by passing a small elevator through the rotator interval and posterior to the anterior glenohumeral capsule to gauge the combined thickness of the subscapularis and capsule. Using sharp dissection and beginning approximately 1 cm medial to the lesser tuberosity, the superficial subscapularis tendon is cut in a longitudinal fashion until 50% of the combined subscapularis–capsular thickness is divided. Then, in the coronal plane, the superficial subscapularis is elevated off the underlying deep capsule. This coronal division is taken medially to a point just lateral to the palpable glenoid rim. The deep layer is then longitudinally incised, making sure to remain lateral to the glenoid labrum. The medial capsulolabral glenoid attachment should not be disturbed during this technique. At arthrotomy, débridement of osteophytes, loose bodies, and hypertrophic synovium can be accomplished. By repairing the lateral edge of the superficial flap to the medial edge of the deep flap, as much as 2 cm of length (40 degrees of rotation) can be gained. To date, we know of no patient who has redeveloped anterior instability regardless of the amount of external rotation gained by this method.

Capsulorrhaphy Arthropathy

Capsulorrhaphy arthropathy occurs after surgical stabilization when overtightening the anterior capsule and subscapularis leads to internal rotation contracture. Repeated attempts at external rotation against anterior capsular restriction may lead to capsulorrhaphy arthropathy via altered glenohumeral mechanics.[96,166] Typical findings are severe glenohumeral destruction, eccentric posterior glenoid wear, and posterior subluxation (Fig. 16-21). Anterior capsular release and subscapularis lengthening is recommended if the joint surfaces are intact. For established capsulorrhaphy arthropathy with subluxation, subscapularis lengthening and anterior capsular release are unlikely to provide any long-standing benefit. If symptoms warrant, the most successful treatment is shoulder arthroplasty. Because of the tendency toward posterior glenoid deficiency and subluxation, humeral hemiarthroplasty alone may lead to continued posterior subluxation. Therefore, despite the often young age of these unfortunate patients, total shoulder replacement is usually indicated.

Neer et al.'s work on total shoulder arthroplasty included those shoulders that had prior instability surgery. Approximately 7% of the total shoulder arthroplasties were performed on patients with prior instability surgery.[194] The average age for arthritis after instability was 38 years versus 60 for primary osteoarthritis.[195] Sperling et al.[251] studied 31 patients treated with total shoulder arthroplasty or hemiarthroplasty for capsulorrhaphy arthropathy at average 7 years follow-up. For both groups improvements were noted in pain relief, external rotation, and abduction. Revision rates were high, with 8 of 21 patients with total shoulder arthroplasty requiring revision and 3 of 10 hemiarthroplasties requiring revision. The authors concluded that pain relief and increases in motion could be achieved, but that revision rates remained high.

Bigliani et al. reported on 17 patients who underwent shoulder arthroplasty for end-stage capsulorrhaphy arthropathy.[25] The average time between instability repair and arthroplasty was 16 years. At a mean follow-up of 3 years, 77% of patients obtained a satisfactory result. Although these results are good, they are less than what would be expected after arthroplasty for osteoarthritis. Specific surgical problems were distorted anatomy, anterior soft tissue contracture, and posterior glenoid deficiency. Green and Norris[83] studied 19 patients with advanced glenohumeral arthritis after failed anterior

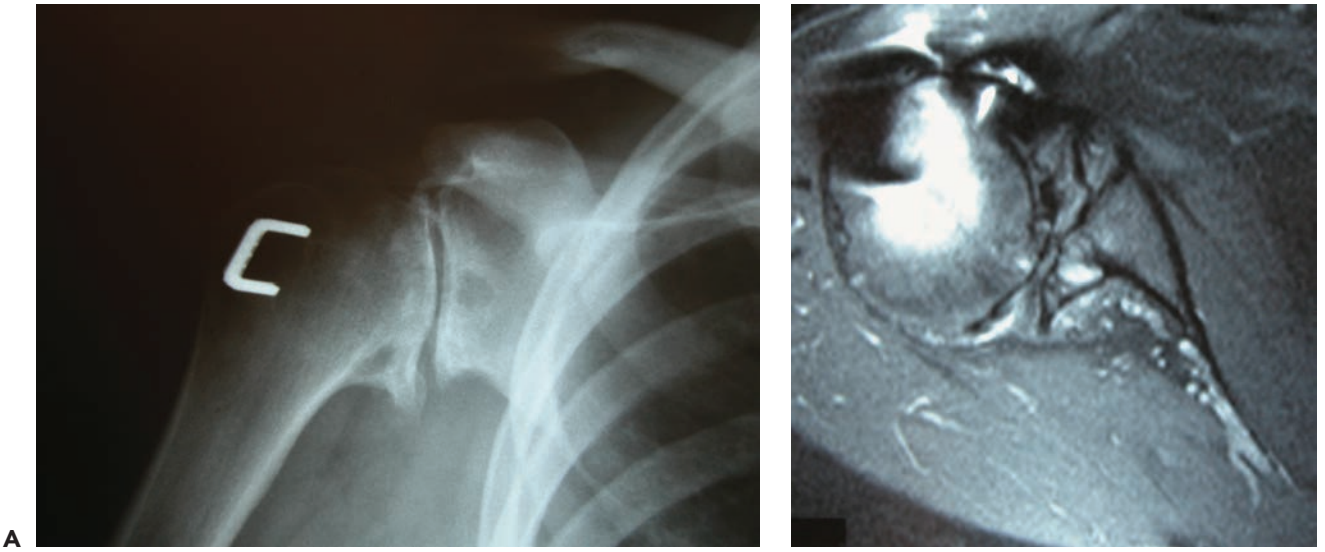

Figure 16-21 Capsulorrhaphy arthropathy in a patient who underwent a Magnuson-Stack procedure (lateral transfer of the subscapularis) twelve years prior **(A).** Notice the typical posterior subluxation and eccentric glenoid wear seen on magnetic resonance imaging scan **(B).**

instability procedures. Severe internal rotation contracture and posterior glenoid bone loss were the most common findings. Active and passive motion improved in all planes and subjective rating of pain showed improvement in 94% of patients treated with total shoulder arthroplasty or hemiarthroplasty.

Subscapularis Failure

Rupture of the subscapularis after an anterior instability repair is increasingly recognized as a cause of surgical failure and postoperative disability.[86,295] Patients usually present with pain that fails to subside postoperatively, weakness, tenderness over the lesser tuberosity, apprehension, and possibly recurrent instability. Physical examination reveals an increase in passive external rotation relative to the normal side, weakness or pain on active internal rotation, and inability to perform a lumbar lift-off test as described by Gerber and Farron.[72] The diagnosis can be made on physical examination alone but, if uncertainty remains, magnetic resonance imaging (MRI) may be helpful (Fig. 16-22). Usually the patient will give a history of persistent postoperative pain that fails to abate during the normal rehabilitation period or a traumatic event in the early postoperative period that initiates symptoms.

The initial step in the management of subscapularis repair failure is prophylaxis. Greis et al. have questioned whether early, aggressive mobilization after instability surgery may lead to an increase in subscapularis failure.[96] Motion exercises within the first 3 to 6 weeks after instability repair should not be performed to the point of tension on the subscapularis repair.

Sachs et al.[231] studied 30 patients treated for unidirectional anterior instability with open Bankart repair. Seven of the 30 had incompetent subscapularis muscles at 4-year follow-up. Of the patients with incompetent subscapularis, only 57% rated their results as good or excellent and would have the surgery again. The authors concluded subscapularis function was the ultimate determinant of success and warned that aggressive rehabilitation should be avoided.

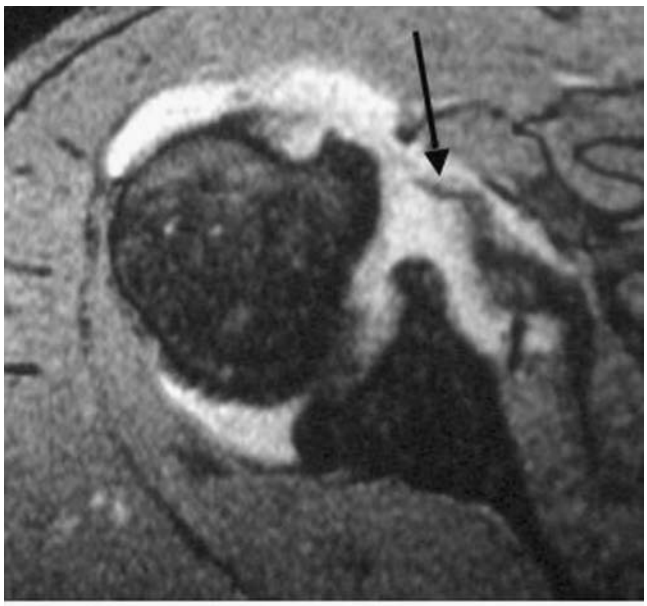

Figure 16-22 Magnetic resonance imaging scan demonstrating subscapularis failure after prior open stabilization.

Mobilization and repair of the ruptured subscapularis tendon can be a challenging undertaking, especially if the patient presents late after subscapularis failure.[295] The subscapularis tendon will often retract under the conjoined tendon, where it adheres to the surrounding tissues. Subsequent attempts at mobilization can be extremely hazardous to important neurovascular structures. Also, the upper, middle, and lower subscapular nerves usually insert more lateral than would be expected.[42,293] Dissection medially on the superficial surface of the subscapularis may result in denervation.

Repair of the ruptured subscapularis, once it is adequately mobilized, can also be formidable. If sufficient quality tendon tissue remains on the muscle and bone insertion, direct tendinous repair is performed. If the lower half of the subscapularis tendon remains intact, it is separated from any remaining capsule and transferred superiorly. Usually, the humeral side has only a remnant of tissue and repair of the tendon through bone tunnels at the articular margin adjacent to the lesser tuberosity is required. On occasion, we have found patients who have extremely poor subscapularis tissue. In these circumstances, we have reinforced the subscapularis repair with a Dacron graft or xenograft (Fig. 16-23). The graft is used primarily to protect and augment the tendon repair or reinforce large defects. We do not use the graft to span a full-thickness defect, but only to reinforce thin tissue.

The optimal treatment of an irreparable, full-thickness subscapularis defect is controversial. Wirth et al. have recommended superior transfer of the clavicular head of the pectoralis major to restore stability, with excellent results.[314] Resch et al.[216] studied 12 patients with irreparable tears of the subscapularis treated with transfer of the upper one-half to two-thirds of the pectoralis major tendon routed under the conjoined tendon. Average follow-up was 28 months, with 9 of 12 demonstrating excellent or good results. Jost et al.[125] transferred the entire pectoralis tendon anterior to the conjoint tendon in 30 repairs, with 25 very satisfied or satisfied with the results. Less favorable results were obtained when an irreparable supraspinatus tear was also present. Klepps et al.[136] performed a subcoracoid pectoralis transfer of the upper half to two-thirds of the tendon in 14 patients. Decreased pain and improved forward flexion were seen in 9 of 14 patients.

Hardware Complications

Any foreign object placed within the vicinity of the glenohumeral joint has the potential to loosen and migrate. The great range of the glenohumeral joint and significant soft tissue tensile loads put enormous demands on any fixation device. Hardware failure or loosening can be seen at any point in the postoperative period, from within weeks to years after surgery. Metal that was safely away from the articular surfaces at placement can later become a constant articular gouge during glenohumeral motion. More seriously, loose hardware in the shoulder can migrate significant distances to threaten vital structures.

The largest series in the literature of problems in the shoulder after placement of hardware was reported by Zuckerman and Matsen.[296] Screws and staples placed to secure a transferred coracoid or to plicate an anterior capsule were usually at fault. Most of the patients required reoperation and 41% had significant chondral injury noted

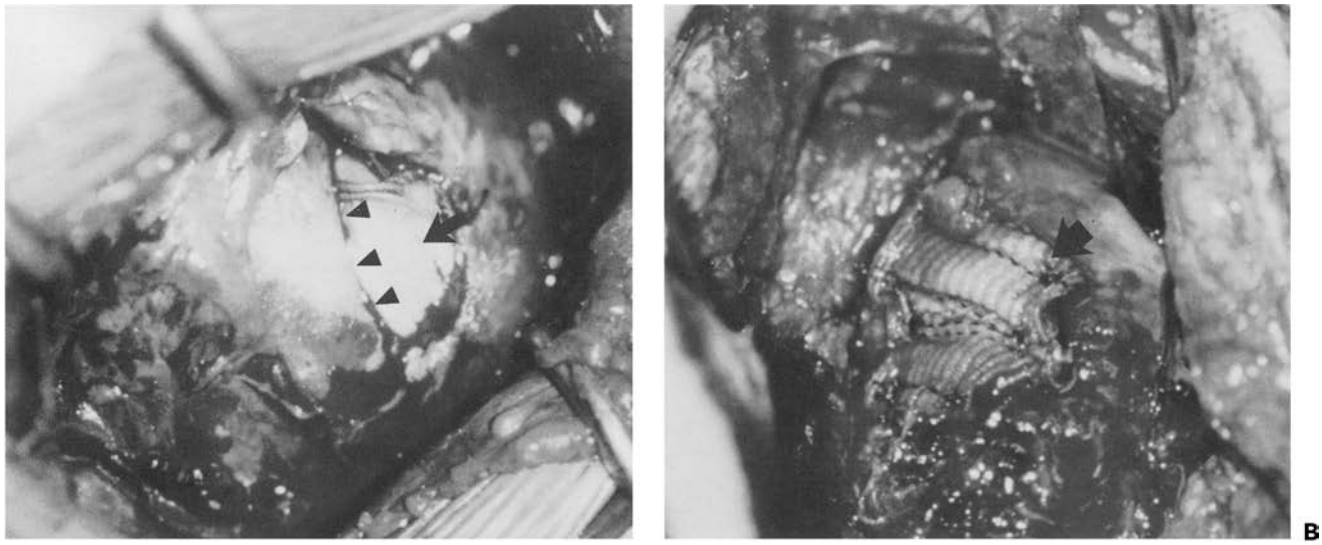

Figure 16-23 **(A)** Intraoperative photograph of the left shoulder of a patient who had failure of the subscapularis repair after a previous Bankart repair. The *arrowheads* demonstrate the retracted subscapularis edge with the arrow showing the underlying exposed humeral head. **(B)** Repair of the subscapularis and reinforcement of thin tissue with a Dacron graft (*arrow*).

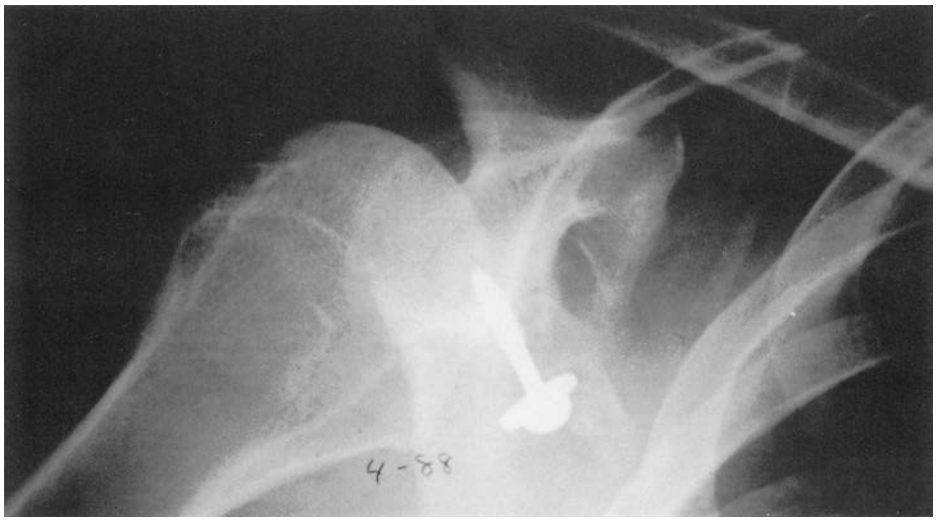

Figure 16-24 A 26-year-old man, 3 years after a Bristow reconstruction. He developed recurrent dislocations and was noted to have a loose screw and coracoid nonunion. (Courtesy of Frederick A. Matsen III, University of Washington Medical Center, Seattle, WA.)

at reoperation. Norris and Bigliani noted "avoidable" hardware complications in 40% of the patients in their series of failed instability repairs.[198] In particular, coracoid screws placed during the Bristow operation have caused problems[14,56,119,199,296] (Figs. 16-24 and 16-25).

The simplest method of preventing complications of hardware is to not use it at all. All anatomic repairs for anterior instability can be performed without implanted devices. Although metal suture anchors may shorten operative time and are safe in virtually all cases, they are not without risk; anchors can loosen or become prominent, hastening chondral degeneration.

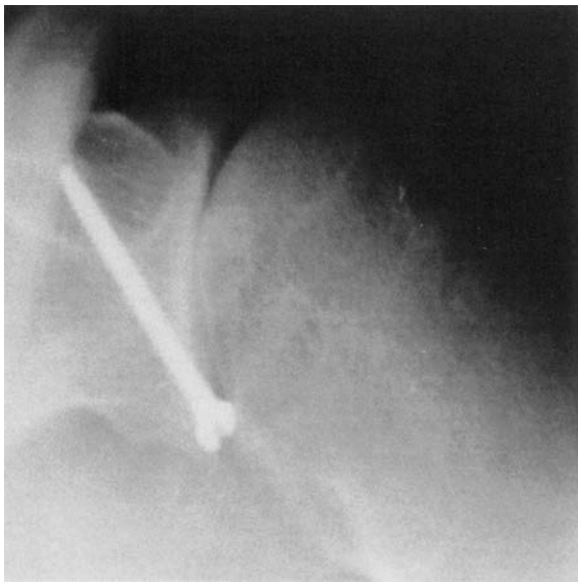

Figure 16-25 A 36-year-old man, 12 years after a Bristow reconstruction, presented with increased pain and decreased range of motion. The screw head has begun to erode the humeral articular surface.

If the patient requires revision surgery, creating strong bone tunnels in an anterior glenoid that has been filled with suture anchors can be a formidable challenge. Often, suture anchors are placed medial to the anatomic glenoid rim. During revision, we have had success using the residual bone defect after removal of the suture anchor as the nonarticular side of the bone tunnel for suture placement. We will often place a small amount of cancellous autograft into the remaining defects (Fig. 16-26). Fracture of the glenoid rim, however, is a greater risk during revision surgery requiring anchor removal.

Neurovascular Injury

Anterior instability surgery may jeopardize the anterior neurovascular structures. Richards et al. have reported on neurologic injury after anterior instability surgery, specifically Putti-Platt and Bristow operations.[219] The most common nerve involved was the musculocutaneous nerve, with the axillary second (Fig. 16-27). Flatow et al. have performed anatomic dissections of the musculocutaneous nerve and found the nerve to enter the conjoined tendon as near as 2 cm to the coracoid process.[60] The nerve, therefore, is particularly at risk during procedures that involve coracoid osteotomy or transfer.

Because of its direct relation to the inferior glenohumeral capsule, the axillary nerve is at particular risk during instability surgery.[158] Richards et al. reported two axillary nerve injuries in their series, one of these caused by a suture around the nerve.The single best way to avoid injuring the axillary nerve is to constantly be sure of its location. We always directly localize the nerve after incision of the clavipectoral fascia, as recommended by Matsen et al.[168] Throughout the procedure the nerve is palpated and localized. Finally, before closure, the nerve is once again palpated or visualized. This final step is critical in postoperative

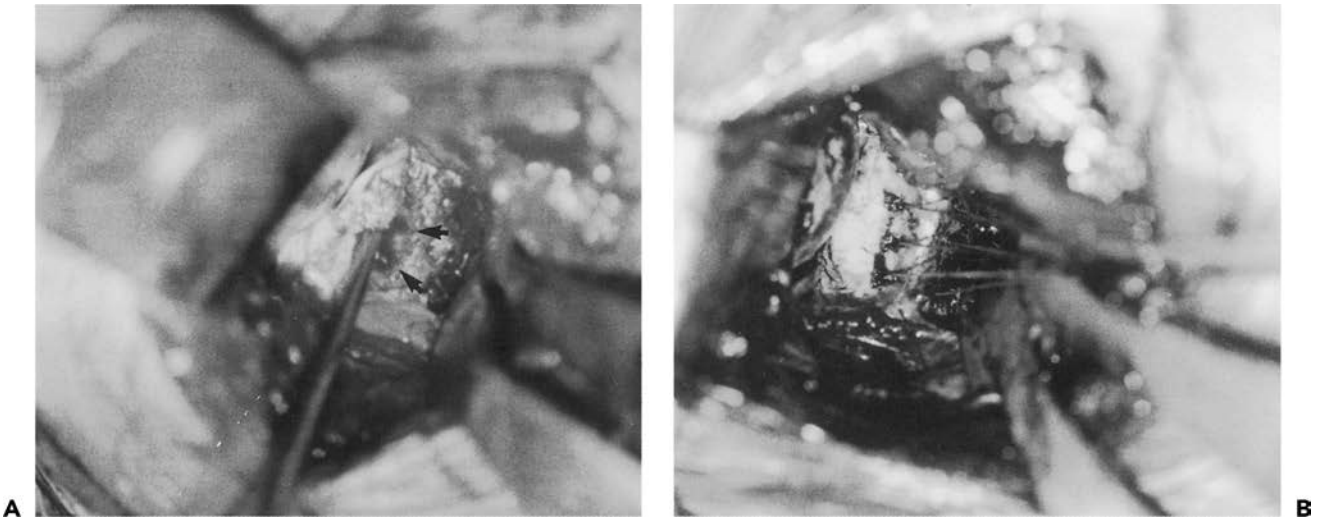

Figure 16-26 **(A)** Intraoperative photograph of a right shoulder during revision Bankart surgery after previous suture anchor repair. Because of their location and size, the suture anchors required removal, leaving large osseous defects in the anterior glenoid neck (*arrows*). **(B)** The osseous defects were used as the nonarticular side of the bone tunnels and a nonabsorbable, braided suture has been passed for capsulolabral repair. The defects are then packed with cancellous graft.

decision making should the patient awake with an axillary nerve palsy. If the nerve was palpably or visually intact at the end of the procedure, the surgeon can be much more comfortable with a provisional diagnosis of neurapraxic injury and may wait for recovery.

Ho and Cofield recently reported an incidence of neurologic injury in patients undergoing instability surgery of 8.2%.[105] These were predominantly neurapraxic injuries of the brachial plexus. All patients showed signs of neurologic recovery by 3 months after surgery and, at a mean of 8.4 years of follow-up, 19 of 24 patients had complete recovery.

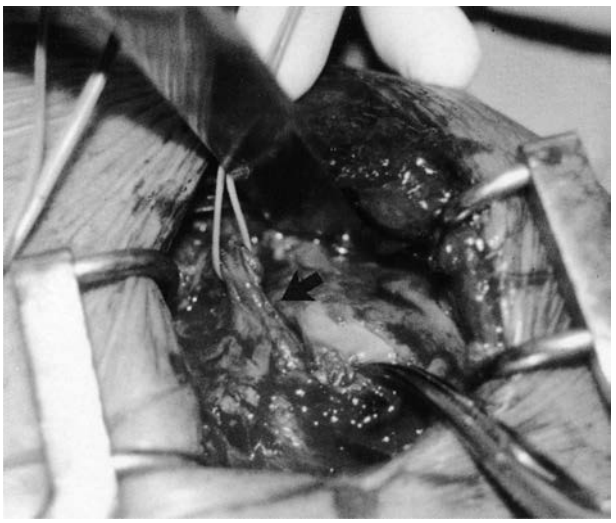

Figure 16-27 A 28-year-old man with complete absence of musculocutaneous nerve function after a reported revision Bankart repair with capsular shift. On exploration, a subscapularis repair suture (hemostat) was found around the musculocutaneous nerve (*arrow*).

We advocate that if the procedure lasts longer than expected, the retractor should be occasionally released to restore blood flow to the brachial plexus.[94]

If postoperative examination reveals a mononeuropathy involving the axillary or musculocutaneous nerve, and the nerves were not palpated and known to be intact before closure, an electromyography (EMG) study should be performed immediately. EMG within the first 24 hours of injury should be able to demonstrate axonotmesis. If axonotmesis is confirmed, immediate nerve exploration with removal of offending suture or nerve repair is indicated. Because most injuries are neurapraxic, any mononeuropathy involving nerves that were palpably intact at closure or any polyneuropathy can be treated with observation. Neurologic deficit that does not reverse itself within 6 weeks postoperative requires an EMG. If the EMG is consistent with neurapraxic injury, further observation is appropriate and, as demonstrated by Ho and Cofield, clinical signs of neurologic recovery should become obvious by 3 months.[105] Should the findings be consistent with axonal interruption, however, immediate exploration with possible repair or cable graft is necessary.

Vascular injuries secondary to instability surgery are exceedingly rare. Most of the reports in the literature concern late axillary artery problems secondary to loose Bristow screws.[14,56,119] Both Artz and Huffer[14] and Iftikhar et al.[119] described axillary artery pseudoaneurysms caused by loose Bristow screws. The diagnosis was initially made clinically by neurologic loss associated with the compressive effects of the pseudoaneurysm, with the diagnosis confirmed by arteriography. In both of their reports, despite prompt diagnosis and exploration, permanent neurologic loss ensued.

OPEN REPAIRS FOR POSTERIOR INSTABILITY

Posterior shoulder instability represents approximately 5% of all instability cases.[220] About half of these patients will recall a distinct injury to the shoulder. Dislocation is rare, however, with approximately 25% with a documented posterior shoulder dislocation requiring reduction in an emergency room.[220] Open surgical procedures for posterior instability can be divided into osseous, soft tissue, or combination.

Bony procedures include glenoid osteotomy, posterior bone block (reverse Eden-Hybbinette), and proximal humeral rotation osteotomy. Soft tissue procedures include posterior capsular shift, reverse Bankart repair, and reverse Putti-Platt procedures. Combination procedures incorporate elements of both categories.

Recurrence of Instability

The actual incidence of recurrent instability after open posterior repair has been reported as extremely variable and dependent on the index procedure. Rates as high as 30% to 50% have been reported.[260] There may be several reasons to explain the high recurrence rate. Unlike anterior instability, there appears to be no "essential lesion" of posterior instability. Recurrent instability is thought to be a pathologic process incompletely understood with several causes making treatment more difficult. As would then be expected, the results of surgery for unidirectional posterior instability are variable and often much less gratifying than those for anterior instability.

Boyd and Sisk reported their results of posterior capsular imbrication, with and without posterior transfer of the long head of the biceps tendon, and found no recurrences at longer than 2 years postoperative.[28] Hurley and colleagues, on the other hand, reported a recurrence of instability rate of 72% after soft tissue reconstruction for posterior instability.[114] When Hawkins et al. reviewed their cases of posterior capsular and infraspinatus imbrication, they found a recurrence rate of 83%.[100] Regardless of the technique used, the overall recurrence rate in that study was 50%. Tibone and Ting reported recurrence of instability in 6 of 20 patients who underwent staple posterior capsulorrhaphy.[261] Bigliani and colleagues reported the results of posteroinferior capsular shift in 35 patients with posterior instability.[23] At a mean follow-up of 5 years, only four patients suffered from recurrence.

Most authors, however, cite posterior capsular redundancy as the most common intraoperative pathology. The wide recurrence range likely also depends on the sample group. The results of treatment are not often reported on one well-defined subgroup. Besides patients with unidirectional posterior instability, many patients with MDI have painful posterior subluxations, making this their primary direction. There is no general agreement regarding classification of posterior instability. Multiple variables, such as trauma, MDI, and voluntary or involuntary components, are difficult to integrate into a reproducible accepted classification system. Nevertheless, posterior instability has been broadly classified into acute or chronic. Chronic can be further subdivided into "locked" (missed) and recurrent voluntary or recurrent involuntary. Structural abnormalities including labral injury or rotator interval injury may exist. Structural abnormalities also may include bony defects like increased glenoid retroversion or posterior glenoid erosion.

The causes of failure after posterior instability repair mimic those of anterior repairs. There are, however, several failure mechanisms unique to posterior repairs (Table 16-3). The causes of recurrent posterior glenohumeral instability as well as the best surgical correction remain uncertain.

Incorrect Diagnosis

Many of the causes for postoperative instability are similar to those after anterior reconstruction. Specifically, accurate diagnosis is critical. Because recurrent anterior instability is much more common in frequency than recurrent posterior, it is easy to misdiagnose a posterior dislocator simply on a statistical basis (see Fig. 16-1). Multidirectional instability must also be excluded as a diagnosis. Wolf et al.[285] studied open posterior capsular shift and labral repair in 44 shoulders from 2 to 22 years of follow-up for posterior instability. His study group consisted primarily of traumatic posterior subluxors or dislocators. Recurrence of instability was found in 19% of patients. All patients had examination under anesthesia and it was documented that 11 cases had MDI. In those cases with MDI recurrence was 40% versus 10% for those patients with posterior inferior instability. The authors emphasized the need to document MDI at the time of surgery. Burkhead and Ritchie[35] reviewed 23 patients with postoperative instability and found 5 of 23 patients had missed MDI. Rockwood and Gerber[221] found missed MDI in 68% of their study patients with postoperative instability.

Once MDI, scapular winging, and anterior instability have been ruled out, the examiner must first attempt to answer two questions. First, what is causing the symptoms? Is it true posterior instability or is there another potential cause of the symptoms? Second, what is the pathology that

TABLE 16-3
CAUSES OF FAILURE OF OPEN POSTERIOR REPAIR

Recurrence of instability
Degenerative arthritis
Coracoid impingement syndrome

accounts for the symptoms? These are best assessed using clinical examination and history.

Clinically, recurrent subluxation typically presents as achy activity-related pain exacerbated by overuse. A history of trauma and volitional instability should be sought and habitual dislocators should undergo psychologic examination. Other potential causes for posterior shoulder pain include suprascapular nerve compression, Bennet lesion (posterior glenoid spur), quadrilateral space syndrome, osteoarthrosis, tumor, scapular winging, or cervical radiculopathy.

Physical examination attempts to reproduce symptomatic posterior translation. The signs, however, may be nonspecific. Athletes may have nonpathologic posterior humeral head translation of 50% and little discomfort. If the patient has painful posterior translation, the position of the arm and scapula should be noted. Patients with true posterior instability typically demonstrate subluxation with the arm in 80 to 90 degrees of forward flexion. Bidirectional instability patients typically require 110 to 120 degrees of forward flexion. Any inferior component to the instability is noted as it implies either rotator interval laxity or a redundant inferior capsule.

Regardless of the test performed, it is critical to demonstrate reproducible symptomatic posterior instability in the clinic setting and to confirm the direction of instability in the operating room with examination under anesthesia.

Failure of Operative Technique

The most common surgical error is a failure to address all components of the instability.[24,220,274] Open procedures for posterior instability typically involve fixation of labral tears, posterior capsular shift, and, in some instances, posterior glenoplasty to correct retroversion. The treatment, whether open or arthroscopic, should address all lesions of the shoulder.

Capsular laxity, at present, is felt to be the primary cause of recurrent atraumatic posterior instability. A redundant capsule is the most common finding at surgery. Cadaveric and biomechanical studies have reinforced the importance of the inferior glenohumeral ligament and rotator interval when evaluating posterior instability. Warren et al.[274] demonstrated in cadaveric sections damage to the anterior–superior capsule with posterior dislocation. They coined this injury the "circle" concept, emphasizing capsular injury on both sides of the capsule for dislocation to occur. Blaiser et al.[26] demonstrated that the coracohumeral ligament contributed to posterior stability with the arm forward flexed to 90 degrees in neutral rotation. He also demonstrated that the IGHL contributed to posterior instability with the arm in internal rotation.

Lesions of the labrum have been primarily implicated as contributing to traumatic posterior instability. Lippitt et al. reported that loss of the posterior labrum reduced resistance to posterior–inferior glenohumeral translation by 20%.[151] Lesions of the superior labrum also contribute to instability. Cadaveric studies by Pagnani et al.[205] demonstrated increased anterior–posterior translation in cadavers with superior labrum from anterior to posterior (SLAP) tears. Wolf et al.[285] studied open posterior capsular shift and labral repair in 44 shoulders from 2 to 22 years of follow-up. Their study group consisted primarily of traumatic posterior subluxors or dislocators. Reverse Bankart lesions were found in 18 shoulders and were the most common finding at surgery. Recurrence of instability was found in 19% of patients.

More recently, Misamore and Facibene[186] reported on 14 athletes with unidirectional posterior instability treated with open posterior capsular shift and repair of labral defects. Thirteen of 14 patients had no instability or apprehension on follow-up. Fuchs et al.[65] found recurrence of 23% in 26 patients treated with posterior capsular shift with labral repair, but three of six recurrences were in patients having previously undergone instability procedures.

Despite variability in recurrence, posterior capsular shift with labral repair if necessary is the mainstay of open surgical treatment of traumatic posterior shoulder instability. Good or excellent results can be obtained over short and intermediate terms.

It is unclear how much of a role increased glenoid retroversion accounts for recurrent posterior subluxation.[29,65,74,115,183,212] If a patient suffers recurrence of instability after a soft tissue procedure, osseous abnormality should be investigated. Fuchs et al.[65] identified posterior IGHL complex redundancy in 68% and lesions of the posterior labrum in 50% of 26 patients treated with open posterior capsular shift and labral repair. Glenoid retroversion was 12 degrees in recurrent subluxors versus 6 degrees in stable shoulders. Some studies have confirmed increased glenoid retroversion in patients with posterior subluxation while others have discounted it.[29,74,115,183] The most common osseous procedure performed is posterior glenoid osteotomy. Preoperatively, a CT scan is indicated to accurately evaluate glenoid version. If greater than 10 to 20 degrees of retroversion is found, a posterior opening wedge osteotomy is indicated as part of the revision procedure (Fig. 16-28). Scott reported the use of posterior glenoid osteotomy to treat recurrent posterior instability.[236] In his initial report of three patients, one of the three suffered from anterior instability postoperatively. When using a similar technique, Kretzler reported a recurrence rate of posterior instability of 14%.[139] Hawkins used CT scan to analyze shoulders after posterior glenoid osteotomy.[99] He noted several potential complications, including undercorrection of version, intraarticular fracture, graft extrusion, and osteoarthritis. The rate of instability recurrence was 17%. Osseous procedures alone often leave a redundant inferior capsular pouch or labral pathology, which may account for their high rates of recurrence. As such, these techniques in isolation are no longer recommended.

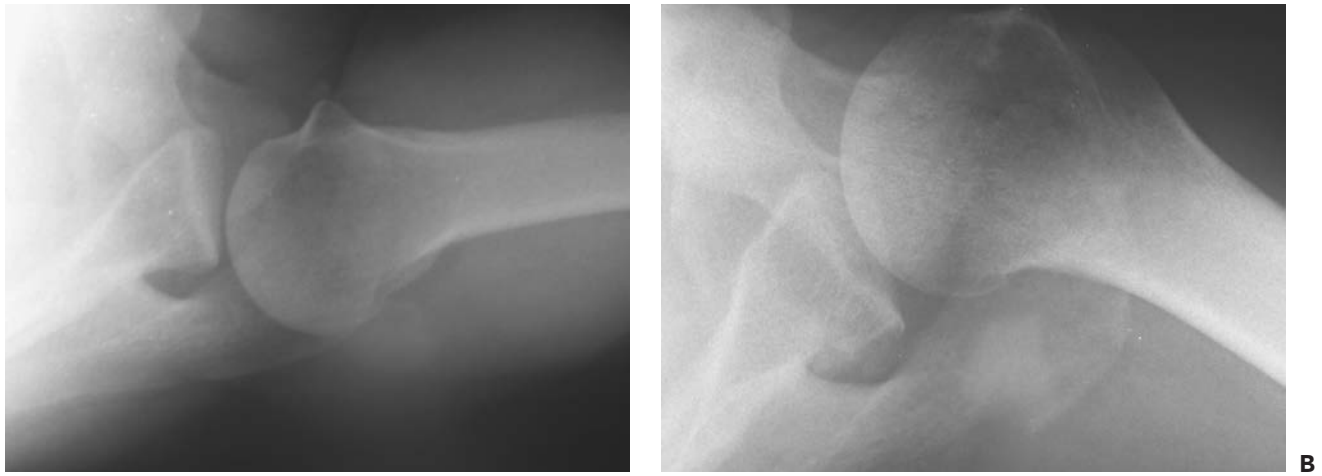

Figure 16-28 Preoperative radiograph of a patient that had failed multiple soft tissue stabilization attempts for posterior instability **(A).** Successful treatment with posterior glenoid osteotomy and posterior capsulorrhaphy **(B).**

Some investigators have reported better results and lower recurrence rates with posterior instability repairs while others have not. Fronek et al. reported 11 patients who underwent posterior capsulorrhaphy with or without posterior bone block and found recurrence of instability in only one of those patients.[63] In 1984, Hawkins et al. reported a recurrence of instability rate of 41% after posterior glenoid osteotomy, posterior capsular plication, and infraspinatus imbrication.[100]

Failure of Rehabilitation

Postoperative rehabilitation is individualized and often begins with immobilization for a period of 6 weeks. Rotator cuff strengthening and scapular stabilization is then undertaken. Full range of motion can be expected by 6 months. If rehabilitation progresses too rapidly, the potential to stretch the capsule exists, potentially leading to instability recurrence.

Miscellaneous Complications

Degenerative Joint Disease after Glenoid Osteotomy

The technique of posterior glenoid osteotomy is fraught with difficulty. Of particular note is the possibility of extension of the osteotomy into the articular surface. The classic technique is described as ending at the anterior glenoid neck such that the glenoid can be "cracked" open and hinged on the anterior cortex. Unfortunately, it is dangerously easy to enter the joint with the osteotomy cut or for the crack to propagate into the joint (Fig. 16-29). Entrance into the glenoid articular surface will predispose the shoulder to arthritic degeneration.

Johnston et al. described a case of glenohumeral arthritis requiring total shoulder arthroplasty in a patient 5 months after posterior glenoid osteotomy.[124] The authors blamed possible intraarticular extension of the osteotomy

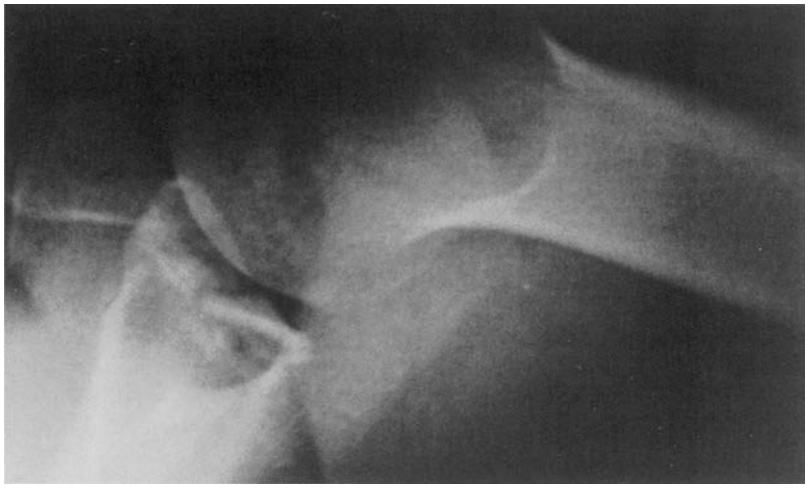

Figure 16-29 Intraarticular extension of a posterior glenoid osteotomy. This patient is at increased risk for necrosis of the isolated posterior glenoid fragment or for glenohumeral degeneration.

as the cause of the rapidly progressive chondral degeneration. Hawkins et al. reported avascular necrosis of an isolated posterior glenoid rim fragment after extension of the osteotomy into the joint.[100] Hawkins noted osteoarthritis in 1 of 12 patients after posterior glenoid osteotomy.[99]

The treatment of glenohumeral degeneration after posterior glenoid osteotomy is difficult. Ultimately, most of these patients will require total shoulder arthroplasty, regardless of their young age. Technically, restoring normal glenoid component version may require posterior support, including bone grafting.

Coracoid Impingement Syndrome

Posterior glenoid osteotomy is a surgical option in the treatment of recurrent posterior instability. Theoretically, if the glenoid version is overcorrected, the humeral head will have a tendency toward anteromedial orientation. This nonphysiologic alignment can result in contact between the anterior humeral head and the coracoid process, the so-called coracoid impingement syndrome (Fig. 16-30).

Gerber et al. reported a combined clinical and anatomic study on the consequences of posterior glenoid osteotomy.[73] They presented a case report of significant, symptomatic coracoid impingement after glenoid osteotomy treated with resection of the inferolateral aspect of the coracoid process. In a corresponding anatomic study, 13 cadaveric shoulders displayed evidence of coracoid impingement after posterior glenoid osteotomy. Humeral contact against the coracoid and coracoacromial ligament was effectively relieved in the cadaveric shoulders by resection of the coracoacromial ligament and inferolateral coracoid process.

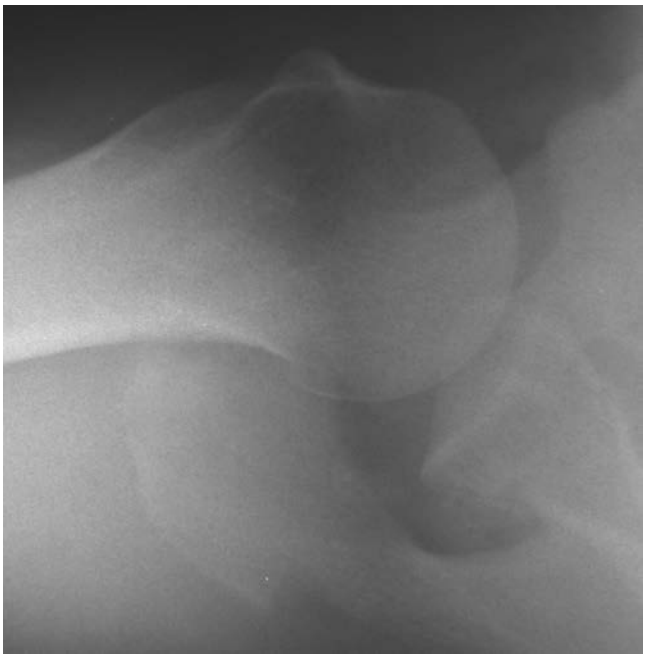

Figure 16-30 Coracoid impingement after posterior glenoplasty.

Summary

Operative treatment for recurrent posterior subluxation or dislocation carries a higher recurrence rate than unidirectional anterior instability. The poor results are believed to be secondary to inappropriate patient selection, incorrect diagnosis, failure to treat all the pathology associated with the instability, and poor compliance with rehabilitation. The optimal treatment to treat posterior instability is evolving, but a course of physical therapy is warranted before operative intervention is pursued. Good success can be achieved for patients with nonvolitional recurrent persistent symptomatic instability. A lesion-specific approach is most successful. Repair of posterior labral pathology and concomitant capsular shift is the mainstay of treatment, with glenoplasty reserved for patients with confirmed retroversion or posterior glenoid erosion contributing to the instability.

OPEN REPAIRS FOR MULTIDIRECTIONAL INSTABILITY

The literature on the surgical treatment of multidirectional instability is not as vast as that for traumatic instability. Yet, many of the complications that plague surgery for traumatic instability are analogous to those for multidirectional instability.

Recurrence of Instability

Overall, the rate of instability recurrence after capsular shift for an appropriate diagnosis of multidirectional instability is low. In 1980, Neer and Foster reported 40 patients who had undergone capsular shift for multidirectional instability, with only one patient suffering a recurrence.[193] Altchek et al. described recurrent instability (posterior) in 1 of 40 patients who underwent a T-plasty Bankart repair for multidirectional instability (anterior and inferior).[7] Cooper and Brems reported recurrence in 4 of 39 patients after capsular shift.[44] Most recently, Pollock et al.[210] treated 49 shoulders with open inferior capsular shift for MDI. There was a 4% recurrence after 61 months of average follow-up. Excellent or good results were found in 94% of their study group.

The causes of instability recurrence after capsular shift can be divided into mechanical and biologic. The capsular shift procedure is predicated on decreasing capsular volume in the appropriate regions, as defined by the patient's instability pattern. Preferential imbrication of one side of the capsule may result in instability in the opposite direction in patients with circumferential multidirectional

instability.[7] To correctly perform a capsular shift for patients unstable anteriorly, inferiorly, and posteriorly, the inferior humeral capsule must be released well past the inferior humeral margin, back to the posteroinferior quadrant. With subsequent superior capsular shift, anterior, inferior, and posterior capsules will all be equally tightened.

Although past investigators have concentrated on reducing the inferior capsular pouch, more recent attention has been directed toward the superior capsule, particularly in the treatment of inferior instability. Basmajian and Bazant hypothesized the primary importance of the superior capsule in preventing inferior instability.[19] Rowe and colleagues reported the presence of a defect in the rotator interval in patients with recurrent subluxations.[229] Recent basic science has further defined the rotator interval capsule (coracohumeral ligament and superior glenohumeral ligament) as the primary inferior stabilizer of the adducted shoulder,[92,270] and other investigators have noted the clinical importance of rotator interval repair.[197] Considering these data, imbrication of the rotator interval should be considered an integral aspect of the capsular shift procedure.

Of all the patients referred to our clinic with a diagnosis of recurrent instability after capsular shift procedure for multidirectional instability, the most common direction of persistent instability is inferior. This pattern of instability is also extremely disabling because carrying is a significant aspect of activities of daily living. Often, these patients can have dramatic sulcus signs even with stress by gravity alone (Fig. 16-31). Sometimes, because of repeated attempts at capsular shift, the patients are overtightened in the anterior and posterior directions, yet continue to display instability in the inferior direction, caused by uncorrected incompetence of the rotator interval (Fig. 16-32). Revision surgery, therefore, needs to primarily address this defect. We will usually imbricate the rotator interval capsule with the glenohumeral joint held located and the arm in approximately 20 degrees of external rotation. If the degree of inferior instability is severe, less external rotation is applied, sacrificing external rotation at the side for stability. Finally, since the long head of the biceps tendon parallels the course of the SGHL and CHL, a reverse tenodesis can be helpful. In this technique, the biceps tendon is left attached at its origin but is released distally. The tendon is then inserted into the humeral head through a bone tunnel with the apex of the tunnel at the insertion of the SGHL. The biceps is then sutured to itself with the humeral head reduced and the arm held in neutral external rotation.

Recurrence of instability after capsular shift can also occur in the anterior or posterior directions. If the patient indeed has recurrent multidirectional instability, consideration should be given to combined anterior and posterior approaches at the time of revision. Neer and Foster suggested this strategy in their initial description of the inferior capsular shift procedure for multidirectional instability.[193] Specifically, they recommended Bankart repair, if necessary, through an anterior approach, with a concomitant posterior approach for capsular shift. The technique is also useful, however, in the absence of a Bankart lesion. Through the posterior approach, complete access is gained to the posterior and posteroinferior capsule. From anteriorly, the anteroinferior, anterior, and rotator interval capsular regions can be addressed.

Postoperatively, these patients are placed in a neutral position brace with the arm positioned in neutral external rotation but with the entire humerus and shoulder girdle superiorly displaced. This brace is maintained for 6 weeks,

Figure 16-31 A 24-year-old woman after six previous surgical stabilization attempts. She has continued inferior instability, even to gravity stress. (Courtesy of Frederick A. Matsen III, University of Washington Medical Center, Seattle, WA.)

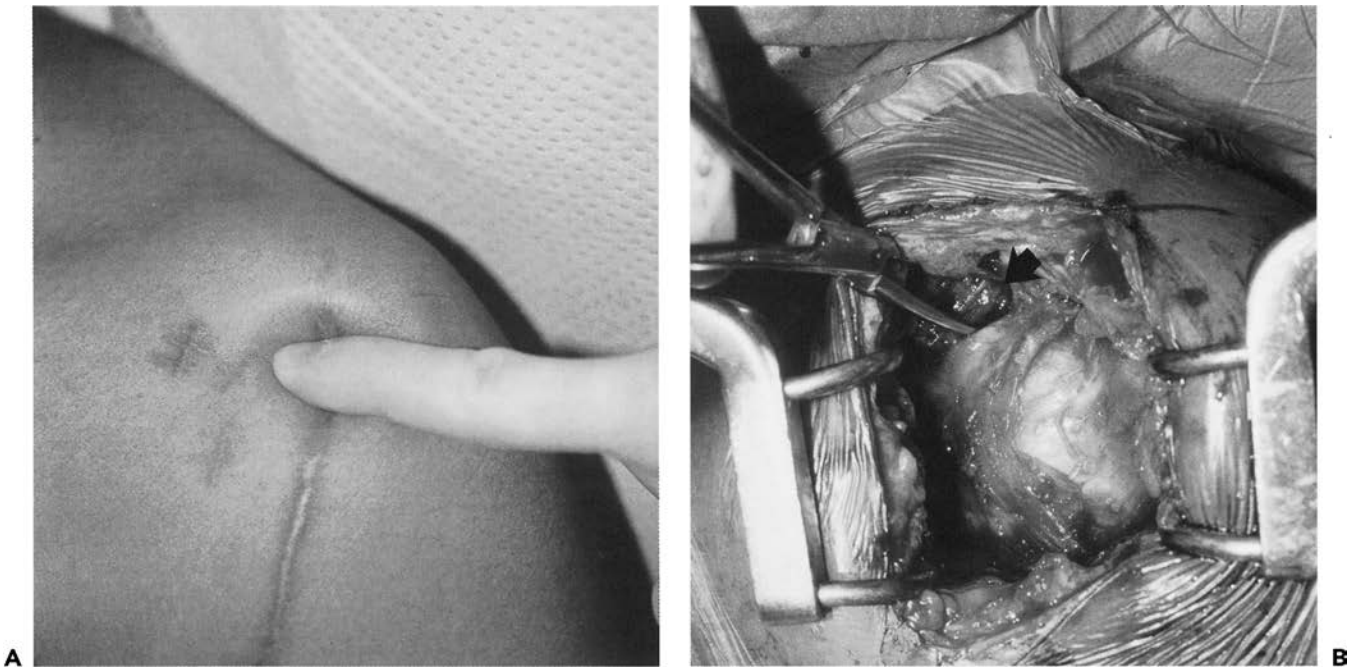

Figure 16-32 **(A)** Persistent inferior instability in a patient who had undergone four previous capsular shift procedures. The rotator interval was not addressed in any of the previous surgeries. **(B)** Intraoperative photograph of the same patient. The rotator interval capsule was noted to be completely deficient and the interval was widened (*arrow*).

with the patient performing abduction and external rotation isometrics in the cast. At 6 weeks postoperative, the cast is removed and a sling is placed. The patient is permitted to remove the sling for activities of daily living, but he or she is encouraged to wear the sling for activities during which the arm may be expected to hang to gravity. At 10 weeks postoperative, the patient is begun on a gentle, supine, active assisted motion program, rotator cuff strengthening against resistance, and a scapular stabilization and balancing program. Contact athletics and heavy lifting are restricted for 1 year.

Despite our mechanical knowledge of the ligamentous support of the glenohumeral joint, multidirectional instability is not a pure mechanical problem. It has long been recognized that patients with multidirectional instability are often systemically ligamentously lax. Neer and Foster discussed not only an increase in capsular volume, but also abnormal capsular compliance in these individuals.[193] McNab and McNab reported on the poor-healing potential in patients with multidirectional instability, implying an overall collagen defect in this disorder. Rodeo et al. found that, in patients with multidirectional instability, there was a decreased cysteine content of the capsule, possibly indicating a higher prevalence of less stiff collagen type III. In addition, the skin of these patients had smaller collagen fibril diameters, again suggesting an underlying defect of collagen.[223]

Patients with recurrence of instability after capsular shift often appear to have abnormal healing. Skin incisions seem to be wider than usual. The patient may say that there was an "opening" of the incision in the initial postoperative period, indicating wound dehiscence. On revision surgery, the amount of deltopectoral and humeroscapular interface scarring appears minimal, and can often be divided by finger dissection. Most importantly, the capsuloligamentous structures are almost always attenuated and deficient. Occasionally, this deficiency may also include the subscapularis tendon and may extend to the point of tissue absence.

We as surgeons do not yet have a good method of overcoming the biologic deficiency in these patients. We tend to treat these patients in a manner similar to that discussed earlier for traumatic instability with anterior capsular deficiency. The surgical approach begins by defining the humeroscapular interface and judging the presence and amount of anterior subscapularis and capsular tissue. In cases of anterior deficiency, we resort to an autologous tendon graft to reconstruct anterior capsuloligamentous defects (see Fig. 16-17).

End-Stage Instability

Rarely, a patient will continue to suffer from disabling glenohumeral instability despite numerous attempts at surgical reconstruction. Although this condition can occur after any type or direction of instability, it is most common with multidirectional instability. These persons usually have impairment of most if not all of their stabilizing

mechanisms, including loss of glenoid concavity, absence of capsuloligamentous restraint, severe Hill-Sachs lesions, and severe scapulohumeral dysfunction.

Diaz et al.[51] reported on eight patients who underwent fusion for end-stage instability. Each had an average of seven previous attempts at surgical stabilization. At a mean follow-up of 35 months there was no reported recurrence of instability. Time to fusion averaged 3.5 months. All patients had subjective improvement and the authors emphasized that this is a viable salvage treatment for refractory instability. Ruhmann et al.[230] studied 43 patients retrospectively for complications of arthrodesis with average follow-up of 6.7 years. Screw arthrodesis and screw and plate arthrodesis were utilized. Patients rated the surgery as excellent, good, or satisfactory in 91% of cases. There were no reported incidents of recurrent instability. Pseudoarthrosis averaged 10% to 15%.

Richards et al. have reported the results of glenohumeral arthrodesis for patients with terminal instability.[218] After fusion, four of six patients with atraumatic instability had not only poor results, but also continued to have the sensation of episodic instability, despite clinical and radiographic evidence of solid fusion. In patients with end-stage instability, we have also resorted to arthrodesis when all other reconstructive methods have failed (Fig. 16-33). Many patients with atraumatic instability are prone to also have problems of the scapulothoracic joint, particularly ptotic posturing, pseudowinging, and snapping scapulae. In this population, glenohumeral arthrodesis initiates or worsens scapulothoracic symptoms. Regretfully, despite the high incidence of continued symptoms, glenohumeral arthrodesis remains the only surgical option for the treatment of end-stage instability.

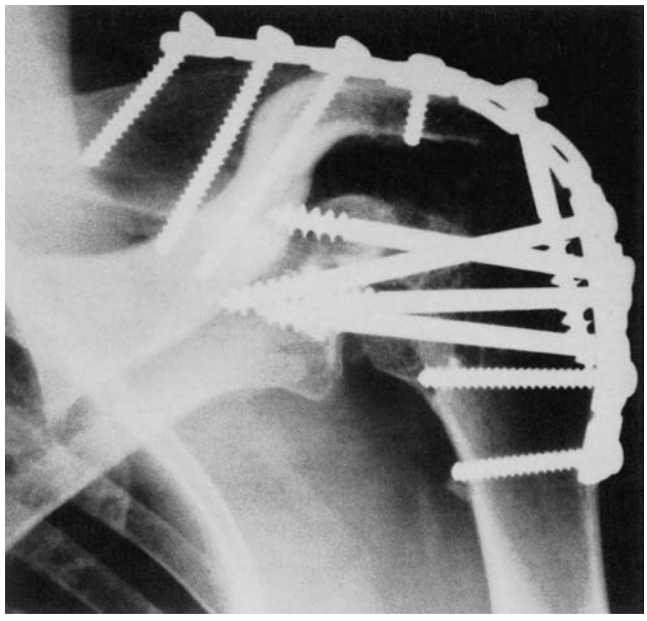

Figure 16-33 Glenohumeral arthrodesis for end-stage instability.

ARTHROSCOPIC REPAIRS FOR ANTERIOR INSTABILITY

Introduction

Over the past two decades shoulder arthroscopy has developed from a diagnostic tool into a valid treatment option for the management of shoulder instability. Arthroscopic repair offers the potential advantages of quicker surgery, with less surgical morbidity, better cosmesis, decreased postoperative pain, more selective anatomic repair, without violating the normal adjacent soft tissues, and recovery of more normal range of motion.[12,85,271] The shoulder, however, has been identified as the joint with the highest rate of arthroscopic complications.[35,242,243] Some of the previously mentioned complications of open instability repair apply equally, if not more so, to arthroscopic surgery. Recurrence rates of 0% to 70% for arthroscopic stabilization have been reported. Arthroscopic repairs are also subject to unique complications specific to the repair method (Table 16-4). Avoiding pitfalls of arthroscopic stabilization surgery requires diligence on the part of the surgeon and advanced arthroscopic skills.

Recurrence of Instability

Incidence

The literature is extremely varied on the recurrence rate of glenohumeral instability after arthroscopic repair. Much of the variability probably relates to the wide array of different arthroscopic repair techniques, differences in surgeons' experience, and short follow-up. When viewed as a whole, the reported recurrence rate of instability after arthroscopic repair ranges from 0% to 70%. Shaffer and Tibone reviewed recurrence of instability and found rates of 16% to 33% for staple capsulorrhaphy, 0% to 60% for transglenoid suturing , 0% to 37% for tack stabilization, and 0% to 30% for suture anchor repair.[238] Overall, this rate of recurrence of instability is higher for arthroscopic repairs when compared with open.[12,20,80,82,85,97,134,141,146,189]

Arthroscopic techniques for shoulder instability can be divided into several main categories: metal fixation such as

TABLE 16-4
CAUSES OF FAILURE AFTER ARTHROSCOPIC REPAIR

Recurrence of instability
Loss of motion
Neurovascular injury
Hardware complications

screws or staples, absorbable devices, suture-based repairs, and thermal treatment. Each technique offers unique advantages. However, each can also result in unique complications and causes for instability recurrence.

Initial arthroscopic Bankart repairs were performed by staple capsulorrhaphy. Johnson's staple repair technique, which formed the basis for many other subsequent approaches, had an initial failure rate of 21%.[123] This high recurrence rate was somewhat related to allowing patients early range of motion postoperatively. The failure rate was cut by a third with immobilization for 4 weeks postoperatively.

Lane et al.[142] retrospectively reviewed 54 patients undergoing arthroscopic staple capsulorrhaphy followed for 39 months. The found a 33% recurrence rate, and a 19% revision rate to open stabilization. Fifteen percent had loose staples on follow-up radiographs and only 43% returned to preinjury level of athletics. Other authors continued to report high recurrence rates after arthroscopic staple capsulorrhaphy.[31,46,87,97,123,142,172,213,243,278,281] The use of this technique is no longer recommended.

As a response to the problems of staple capsulorrhaphy, other investigators developed arthroscopic suture techniques,[12,20,40,52,62,71,80,82,141,189,190,196,206,224,266,277,290] based on the transglenoid open Bankart repair procedures of Reider and Inglis.[215] Some initial reports listed failure rates using this suture technique at less than 8%, but these rates have ranged from 0% to as high as 69% in both short and long-term follow-up.[12,20,40,62,71,80,82,85,146,189,215,240,271,288] Many studies have shown excellent short-term results (no recurrence of dislocation or subluxation) using the transglenoid technique in acute dislocators.[20,189] Recurrence rates, however, can be fairly significant after this technique. Landsiedle,[141] using this technique, showed 14% postoperative dislocation over 24 to 60 months (no mention of subluxation). Hubbell[113] studied 30 patients treated with arthroscopic transglenoid fixation and found a 17% rate of dislocation and 60% rate of subluxation. He concluded that open repairs were more successful in the young athletic population. Manta's review of 38 shoulders, stabilized with the arthroscopic transglenoid suture technique, demonstrated a 10% failure rate at 2 years and a 40% failure rate at a minimum 5 years of follow-up.[165]

Steinbeck and Jerosch[252] compared open Bankart reconstruction using bone anchors to transglenoid fixation arthroscopically and found that in 32 patients followed for 3 years the rate of redislocation for transglenoid fixation was 17% versus 6% for the open procedure. They concluded the higher dislocation rate was related to the poor condition of the inferior glenohumeral lesion at arthroscopy, less time of postoperative immobilization, high level of activity, and number of preoperative dislocations. Hayashida et al.[102] found similar reasons for redislocation. He followed 82 patients for 40 months and found contact sports participation, residual labrum and capsular tear, thin labrum–ligamentous complex, and a repair using less than four sutures to be positive predictors for recurrence of instability using the transglenoid repair.

Another method developed for arthroscopic Bankart repair utilizes absorbable tacks for labral attachment. A commonly used absorbable fixation device is the Suretac (Acufex Microsurgical, Mansfield, MA), composed of polyglyconate polymer. It is a cannulated tack with head and body molded separately and then attached to one another. The tack dissolves by hydrolysis and loses 50% of its strength by 2 weeks and 100% at 4 weeks. Although the device loses its strength as it resorbs, it acts as a temporary compressive fixation for what is thought to be a sufficient time to allow healing of the capsulolabral separation.[207,247] Even in experienced hands, the overall rate of instability recurrence is approximately 12% to 21%.[237,248] Speer and colleagues published results of arthroscopically assisted repair of the anterior aspect of the labrum with the use of a bioabsorbable tack. Their reported failure rate of 21% (11 patients) included four secondary to traumatic and seven resulting from atraumatic reinjury.[248] Common technical errors when using this device include medial placement of the anchor, inadequate abrasion of the glenoid rim, inadequate superior/medial shift of the IGHL, and inadequate compression of the capsular tissue to the glenoid rim. Any of these errors may lead to recurrent instability.

The other common method of arthroscopic labral repair involves use of suture anchors, either absorbable or permanent.[207,247,249,268,269,271] Arthroscopic Bankart repair with suture anchors have been utilized with success ranging from 70% to 93%.[15,118,130] Ide et al.[118] utilized absorbable anchors for acute Bankart repair in 55 athletes with no glenoid bone loss followed for 42 months with 93% success. Mazzocca et al.[174] studied collision athletes and found an 11% redislocation rate after a 37-month follow-up. Burkhart and DeBeer[33] noted that the rate of recurrence for this technique for athletic patients with anterior glenoid defects was 89%. Without significant bone loss the recurrence rate dropped to 6.5%, similar to Ide's study. Similarly, Bacilla et al.[15] reported 91% return to athletic competition without redislocation in 32 patients and Kim et al.[130] reported only 4% recurrent instability in a group of 167 patients (53% contact athletes) followed for 44 months. Koss et al.,[138] however, reported 30% redislocation in 27 patients followed for 40 months with failure associated with more than five preoperative dislocations. Guanche[98] reported 33% resubluxation or dislocation in 15 patients followed for 17 to 42 months.

Causes of Recurrent Instability after Arthroscopic Repair

The higher rate of recurrence of instability after arthroscopic procedures when compared with open ones may

TABLE 16-5
FACTORS AFFECTING RECURRENCE OF INSTABILITY AFTER ARTHROSCOPIC REPAIR

Incorrect diagnosis	Recurrence of Bankart tear
	Early mobilization
Anatomic factors	Strength of repair
Defects of glenoid concavity	
Residual capsular laxity	
Rotator interval defect	
Unrecognized humeral avulsion of glenohumeral ligament	

simply reflect our comparing apples and oranges. The wide array of lesions, differences in patient populations, surgical techniques, multiple scoring systems, and short-term follow-up make comparison of open and arthroscopic repairs difficult. As such, the causes of instability recurrence after arthroscopic repair are not necessarily the same as those after open repair (Table 16-5).

Incorrect Diagnosis

Some of the reasons for instability recurrence after arthroscopic repair are similar to those after open repair. Accurate diagnosis is essential to obtain a good outcome. Failure to identify the patient with MDI will lead to higher percentage failures. In the surgical suite, Oliashirazi et al.[204] found 83% sensitivity and 100% specificity to identify unilateral anterior instability using examination under anesthesia with the affected arm tested in neutral, 40 and 80 degrees of external rotation, and 20 degrees of abduction. They also added the directions anterior–inferior and posterior–inferior in 70 to 80 degrees of abduction to the more standard anterior, posterior, and inferior tests.

Manta and Pettrone found a high failure rate of transglenoid repair in females (70%), particularly those with multidirectional instability.[165] Landsiedle attributed failures to poor suture technique and poor patient selection, including those patients with nontraumatic dislocation and the absence of a Bankart or Hill-Sachs lesion.[141] Warner et al. suggested that success of the procedure may improve by selecting only patients with unidirectional, traumatic, anterior instability who have a discrete Bankart lesion and well-developed ligamentous tissue.[268] Speer and colleagues have commented that their high rate of recurrence (21%), when compared with open procedures for instability, was related to an underestimation of the degree of overall capsular laxity at the time of initial arthroscopic treatment in those patients with atraumatic instability.[248]

Recurrence of Bankart Tear

Either failure of healing of the Bankart lesion or recurrence may lead to postoperative instability. Warner and colleagues sought to clarify the issue of Bankart repair healing by analyzing a group of patients who underwent a "second-look" arthroscopy to evaluate and treat pain or recurrent instability following arthroscopic Bankart repair with the Suretac device. Second-look arthroscopy was performed at an average of 9 months for recurrent instability in seven patients, pain in six, and pain and stiffness in two. In the seven patients with recurrent instability, the Bankart repair was completely healed in three (43%), was partially healed in one (14%), and had recurred in three (43%). In the remaining eight cases with stable shoulders, the Bankart repair had completely healed in five cases (62.5%) and partially healed in three patients (37.5%).[268]

Mologne et al.[188] assessed failed arthroscopic Bankart repairs at open surgery and found that 40% of the redislocators had Bankart lesions at the time of surgery. The presence of the postarthroscopic Bankart lesion correlated significantly to redislocation.

Defects of Glenoid Concavity

As with open repair, failure to correct defects of glenoid concavity will result in higher rates of surgical failure. The glenoid has an average depth of 9 mm in the superior–inferior direction and 5 mm in the anterior–posterior direction. The labrum contributes as much as 50% of the socket depth. In general, concavity–compression results from the compressive force generated by surrounding muscles to center the humerus in the bony glenoid. A Bankart repair that places the labrum medially off the glenoid rim will result in a persistent concavity loss and can result in instability recurrence. Warner and colleagues analyzed eight cadaver shoulders that underwent arthroscopic repair of an experimental Bankart lesion using the Suretac device.[269] These shoulders were then dissected to reveal the placement of the Suretac and the adequacy of the Bankart lesion repair. There were several technical errors that occurred, including medial placement of the Suretac relative to the articular margin. As noted earlier, medial anchor placement fails to adequately restore glenoid contour and concavity and may lead to instability recurrence.

As with open surgery, failure to account for anterior glenoid osseous loss can result in failure of arthroscopic Bankart repair. Anterior inferior glenoid bone loss has been reported in 22% of first time dislocators and 73% of recurrent dislocators. Biomechanical investigations have been conducted to determine the effect of anterior glenoid bone loss on stability. Burkhart et al.[32,33,156] studied glenoid bone loss and found that 25% to 45% of the glenoid must be missing to create an "inverted pear" glenoid. He showed that arthroscopic Bankart repair resulted in a redislocation rate of 4% without bony defects and that the rate of redislocation increased to 61% if an inverted pear was

present. Bony defects in contact athletes resulted in an 89% redislocation rate versus a 6.5% redislocation if no glenoid defect existed. Burkart et al.[32] further showed that the degree of bone loss can be determined arthroscopically by measuring the distance from the bare spot of the glenoid to the posterior rim. This distance is almost identical to the distance from the bare spot to the anterior rim. The percentage of bone loss can therefore be determined. If greater than 25% bone loss exists, the author recommends consideration for glenoid osseous reconstruction or coracoid bone block procedure. Other authors have noted worse results after arthroscopic repair in the presence of either an osseous lesion of the anterior glenoid rim or severe labral deficiency.[47,84,165,206,266]

Residual Capsular Laxity

Failure to address residual capsular laxity has been thought to be an important cause of recurrent instability after arthroscopic repair. Guanche and colleagues have reported a 27% revision rate in arthroscopic cases that did not address capsular laxity as compared with 8% in open cases.[88] Both Landsiedle and Manta and Pettrone reported worse results in those patients with hyperlaxity.[141,165] Manta and Pettrone, Pagnani et al., and Walch et al. all documented failure to definitively address inferior laxity as a contributing factor to failure.[165,206,266] Speer and colleagues have recently reported on arthroscopic reoperations for recurrent anterior instability, in which seven of eight patients had completely healed Bankart lesions. In these patients, the anteroinferior aspect of the capsule was felt to be patulous in each patient.[248] Mologne et al.[188] assessed capsulolabral lesions in 20 patients having failed arthroscopic Bankart repairs. Forty percent had recurrent Bankart lesions and 75% were felt to have redundant anterior capsules. They concluded that capsular laxity, while difficult to quantify, should be addressed at revision surgery.

As discussed in the section on failures of open instability repairs, normal shoulders can have a great degree of capsular laxity, and the significance of residual and unrepaired capsular laxity remains unclear (see Fig. 16-14). Moreover, the exact techniques used to differentiate excessive capsular laxity from normal laxity are unknown and are difficult to perform even in the open setting. Kim et al.[138] described intraoperative methods for evaluating capsular redundancy. The capsule was redundant if thumb-to-forearm distance is less than 4 cm, the sulcus sign is greater than 2+, and a large anterior pouch is seen with insufflation of the joint. Because we do not arthroscope normally lax shoulders, we do not know how many normals would have arthroscopic signs of persistent laxity. Although it is easy to blame persistent laxity as a cause for failure after arthroscopic repair, it remains to be proved that residual laxity after arthroscopic repair is abnormal. Laxity does not necessarily equal instability; therefore, persistent laxity as a cause for instability is a diagnosis of exclusion. Proponents of plastic deformation of the capsule support a modified Bankart repair, which includes a limited capsular shift.[126] Adequate tensioning of the anterior capsule is accomplished by utilizing a shift of the anterior–inferior capsular tissue superior and lateral with the arm in a position of 20 degrees of external rotation. It is unclear, however, if their reported success rates are increased by the addition of the capsular shift.

Rotator Interval Defect

Selective cutting studies have confirmed the rotator interval's role in preventing inferior and posterior instability. For patients with acute anterior dislocation combined with inferior instability, imbrication of the interval is recommended if examination under anesthesia confirms inferior subluxation not resolved with external rotation of the arm and a normal examination of the contralateral unaffected extremity. Arthroscopic findings consistent with rotator interval pathology include redundancy between the supraspinatus and subscapularis and discrete tears of the superior glenohumeral ligament. Redundancy may be difficult to determine arthroscopically and exact criteria to define this don't exist. Additionally, fraying of the biceps tendon or upper rolled border of the subscapularis may be seen, though these findings are nonspecific. Failure to address a rotator interval defect in the patient with inferior instability can lead to recurrence. Speer et al. performed arthroscopic evaluation of eight patients with recurrence of instability after previous arthroscopic Bankart repair. In seven of the eight patients, the Bankart repair had healed. An open lesion of the rotator interval was seen in five of these seven patients.[248]

The Humeral Avulsion of Glenohumeral Ligaments Lesion

Avulsion of the capsulolabral complex at the glenoid (Bankart lesion) is a well-known cause of anterior shoulder instability. Wolf[318] and colleagues have reported on the importance of recognizing a lesser known entity, the humeral avulsion of glenohumeral ligaments (HAGL). HAGL lesions can exist in isolation or with other associated pathology. Recurrent instability without Bankart lesion should be considered a HAGL lesion until proven otherwise (Fig. 16-34). The incidence of recurrent instability from a HAGL lesion has been reported to range from 2% to 9.3%.

Wolf arthroscopically evaluated 64 shoulders with the diagnosis of anterior instability. Six shoulders had HAGL lesions (9.3%), 11 shoulders had generalized capsular laxity (17.2%), and 47 shoulders had Bankart lesions (73.5%). In other authors' experience the rates of HAGL lesions may be lower. Kon et al.[137] noted an incidence of 2.4% in his patient population. In patients with documented anterior instability without a demonstrable

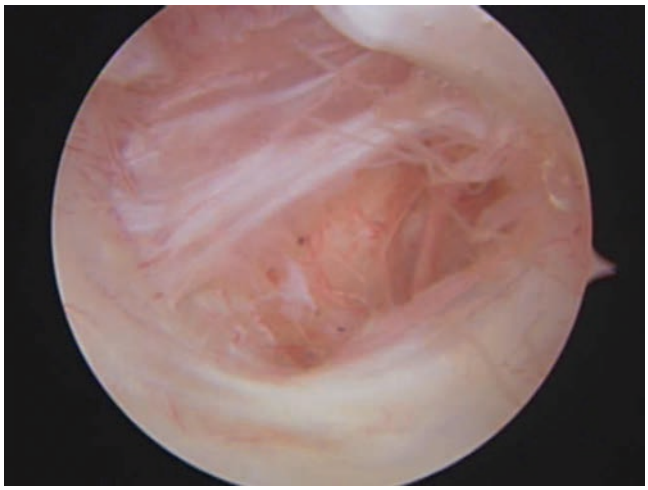

Figure 16-34 Humeral avulsion of the glenohumeral ligament in a patient who had recurrent anterior subluxations after arthroscopic Bankart repair.

Bankart lesion, a HAGL lesion should be ruled out. This lesion is readily recognized arthroscopically from the anterior portal or posterior portal using a 70-degree arthroscope, and its appropriate repair can restore anterior stability.[106] Failure to address a HAGL lesion may result in persistence of instability. Kon et al.[137] described three cases of all-arthroscopic repair of a HAGL lesion. The authors emphasized proper placement of the anchors on the humeral insertion, use of a 70-degree arthroscope, and a transsubscapular portal as critical to their success. Richards and Burkart[217] also described an all-arthroscopic repair of HAGL lesions. They emphasized the use of a 70-degree arthroscope and a "5 o-clock" transsubscapular portal.

Strength of Repair

Because recurrence of the Bankart lesion has been demonstrated as the major source of failure after arthroscopic repair, fixation strength of the repair technique probably plays some role in lesion healing. Arthroscopic repair techniques have varied in their ability to accomplish a robust fixation.

McEleney and others looked at the initial failure strength of eight repair techniques using a previously described canine model of Bankart repair. Intact capsule-to-bone complexes failed at the bony interface at 236 N. Traditional Bankart repair failed at 122.1 N (two sutures) and 74.7 N (one suture), Acufex TAG rod (Acufex Microsurgical, Mansfield, MA) at 143.5 N (two sutures) and 79.8 N (one suture), transglenoid suture technique (two sutures) at 166.6 N, Mitek GII (Mitek, Norwood, MA) (one suture) at 96.4 N, Zimmer Statak (Zimmer Inc, Warsaw, IN) (one suture) at 95.2 N, and Acufex bioabsorbable Suretac at 82.2 N. The two-suture repairs were statistically equivalent in strength to each other, as were the one-suture repairs and the Suretac device. Two-suture repairs were significantly stronger than one-suture repairs ($P < 0.01$). In the single-suture specimens, failure occurred by suture breakage in 46% (18 of 39) of specimens and soft tissue failure around the suture in 54% (21 of 39). Failure in the two-suture techniques occurred primarily by soft tissue failure (23 of 25). No device broke or pulled out of bone.[176]

Shall and Cawley evaluated three mechanical soft tissue fixation devices (SuperAnchor, Suretac, and the Instrument Makar [IM] Bioabsorbable Staple) in a cadaveric model by examining ultimate tensile failure and modes of failure in simulated Bankart repairs. They attempted to realistically evaluate the strengths of soft tissue reattachment procedures at the anterior glenoid under worst-case conditions—load to failure. The mean load at failure for the SuperAnchor was 217.32 N; for the IM Staple, 132.32 N; and for the Suretac, 122.37 N. The load at failure for the SuperAnchor was statistically greater ($P < 0.001$) than the IM Staple and Suretac. There was no statistical difference between load at failure for the Suretac and the IM Staple. The most common failure mode for the Mitek was suture breakage (71%). Anchor pullout from bone was the most common failure mode for the IM Staple (75%) and Suretac (94%).[239]

In a labral repair model, Gohkle and colleagues compared the strength of suture anchor repair to the standard Bankart procedure and the intact anterior capsule.[79] The mean load at failure for the suture anchor repairs varied from 90 to 115 N and was lower than in the standard Bankart procedure (127 N). All suture anchors demonstrated similar holding strength with the exception of an absorbable wedge ($P < 0.05$).[79]

When utilizing the suture anchor technique arthroscopically, recurrence may result from the failure to tie adequate knots arthroscopically. Lo and Burkhart[155] tested Ethibond and FiberWire arthroscopic knots and evaluated knot security and loop security. Knot security was defined as the ability of the knot to resist slippage and loop security as the ability to maintain a tight suture loop as the knot is tied. Several common arthroscopic knots were evaluated including the surgeon's knot, SMC, Tennessee slider, Nicky's knot, Roeder knot, Weston knot, and Duncan loop. A standard surgeon's knot demonstrated the highest force to failure (102 to 197 N) and tightest loop circumference. All sliding knots demonstrated force to failure of less than 75 N and loose loops. Loutzenheiser and Harryman also have demonstrated that some failures may relate to the challenge of tying secure knots arthroscopically. Many knots tied arthroscopically commonly consist of an initial slip knot to remove slack and a series of half-hitches. Half-hitches, instead of square throws, are difficult to avoid and result when asymmetrical tension is applied to the suture strands. For this reason, the security of knots tied arthroscopically may not be equivalent to square knots and a greater rate of failure may occur. The most secure knot configurations were achieved by reversing the half-hitch throws and alternating the posts. These knots performed significantly better than all other knots tested ($P < 0.002$).

Thus, the surgeon can control the holding capacity and minimize suture loop displacement by properly alternating the tying strands and reversing the loop when placing the hitches.[159]

The importance of strength of fixation of suture anchor repair in the Bankart reconstruction is unknown because we do not stress these patients early on in the healing process. Because failure of Bankart repair is a complication of arthroscopic repair, however, strength of fixation may play a role in this repair failure.

Engaging Hill-Sachs Lesion

Impression fractures of the posterior humeral head are seen with both recurrent dislocation and acute dislocation. They are more common in the recurrent dislocator. Instability resulting from a Hill-Sachs lesion is dependant on size, orientation, and location of the defect. Lesions occupying 20% of the humeral head are rarely significant, while lesions of 40% or more are likely important contributors to instability.[59,256] Lesions between 20% to 40% have variable impact.

Lesions of clinical significance will engage the anterior glenoid rim in functional ranges of motion. The degree of involvement may be determined on axillary radiographs. Historically small Hill-Sachs lesions have been addressed by soft tissue procedures that limit external rotation. This prevents the Hill-Sachs lesion from engaging the anterior glenoid. Surgical options are those discussed in the section on failures of open Bankart repair.

Rehabilitation and Early Mobilization

Because the fixation strength is probably less after arthroscopic than after open repair, the period of postoperative immobilization likely plays an important role in preventing recurrence of instability. Johnson determined that he could cut his recurrence rate (of about 20%) after arthroscopic staple capsulorrhaphy by one-half if he increased the period of postoperative immobilization from less than 3 weeks to 4 weeks.[123] Three to 4 weeks of strict immobilization has also been recommended by other investigators.[6,40,189] Limiting early trauma is also thought to be extremely important. If the patient desires to return to contact sports, a 6-month delay has been reported to lower the rate of instability recurrence.[244] Recently Kim et al.[131] has investigated an accelerated rehabilitation program in nonathletes who underwent arthroscopic Bankart repair. Sixty-two patients were followed for an average of 31 months. One group underwent a "traditional" rehabilitation program consisting of 3 weeks of immobilization in an abduction sling and the other group was managed with accelerated rehabilitation. Motion and strength exercises were begun from the first postoperative day in this group. Recurrence of instability was the same between the two groups, with two patients from each group experiencing postoperative apprehension. Patients in the accelerated group had earlier return of functional range of motion and had earlier return of functional activity. Accelerated rehabilitation also resulted in decreased levels of pain and increased patient satisfaction.

Loss of Motion

It has become increasingly evident that shoulder motion, especially external rotation, need not be sacrificed for stability. In theory, by not incising the subscapularis or violating the humeroscapular motion interface, arthroscopic repair offers the potential for better preservation of glenohumeral motion than does open repair. Despite the potential advantages over open repairs, the incidence of postoperative stiffness following arthroscopic stabilization ranges from 2% to 15%.[47,191]

After arthroscopic Bankart repair, most patients can anticipate a reliable return of motion that is nearly symmetrical with that of the contralateral shoulder.[255] Arciero et al. treated 25 patients by arthroscopic Bankart repair using a cannulated bioabsorbable fixation device. Nineteen patients with an average follow-up of 19 months had an average loss of external rotation of 3 degrees.[11] Warner and Warren reported on 26 patients with a minimum of 24-month follow-up, treated with the Suretac device, who had an overall recurrence rate of 8%. They noted an average loss of external rotation of 7 degrees, with five of eight throwing athletes and four of four swimming athletes being able to return to their premorbid level of athletic participation.[271] Finally, Uribe and Hechtman reported on 11 young athletes with acute anterior Bankart lesions treated with the transglenoid suture repair. When using the Rowe scale, there were nine excellent and two good results. All eventually achieved full external rotation in adduction and 90 degrees of abduction compared with the opposite side.[265] Fabbriciani et al.[54] compared open and arthroscopic repairs in equivalent patient populations. They found that outcome measurements such as pain, recurrence, function, and strength were equivalent. Postoperative motion for the arthroscopic group was better than that seen after open repairs.

Wall and colleagues reported that their postoperative protocols commonly expect normal motion by 10 to 12 weeks in nonthrowing athletes and 6 to 8 weeks in throwing athletes. If after these time periods there is restricted motion, especially external rotation less than 10 degrees or abduction below 90 degrees, there is cause for concern. If aggressive physical therapy fails to regain motion, manipulation under anesthesia or arthroscopic release may be employed. The authors describe using an anterior release for limitations of external rotation and axillary pouch release for abduction contractures, sometimes using electrocautery or lasers as an adjunct. For lesser degrees of motion restriction, they will wait 6 months to treat arthroscopically.[267]

Neurovascular Complications

Vascular complications after arthroscopic shoulder surgery are thankfully rare. Burkhart[34] reported a case of deep venous thrombosis in a patient who subsequently was identified as having a hypercoagulable state. Pseudoaneurysm[38] and cephalic vein laceration[47] have also been reported.

The incidence of neurologic injury following shoulder arthroscopy has been reported as high as 30%.[178] Mechanisms of injury include traction from the lateral decubitus position, fluid extravasation, direct injury, tourniquet-like complications from wrapping of the upper extremity, and regional sympathetic dystrophy. Pittman et al.[209] noted that traction neurapraxia occurred in 10% of their study group and recommended somatosensory evoked potentials to monitor this complication. Segmuller et al.[2] noted direct nerve injury with 7% of their study group demonstrating transient sensory deficits. Permanent deficits requiring tendon transfers have been reported.[171]

Careful well-padded positioning, limited fluid extravasation, less than 7 kg of arm traction (for lateral decubitus position), and accurate portal placement are required to limit neurologic complications. Most neurologic injuries are transient, with resolution the rule and not the exception.

Conclusion

As with open repairs, treating anterior shoulder instability arthroscopically requires an accurate diagnosis, selection of correct operative procedure, appropriate equipment and training, and correct rehabilitation procedures. Patients with traumatic unidirectional anterior instability, confirmed Bankart lesion, and robust capsulolabral complex who do not participate in contact athletics and have no anterior glenoid bone loss or engaging Hill-Sachs lesion represent ideal candidates for arthroscopic stabilization. Without accurate identification and correction of the pathology, any arthroscopic procedure will have a high failure rate. Arthroscopic stabilization offers less morbidity, shorter surgical time, improved cosmesis, improved range of motion, and less postoperative pain. With improving techniques and by addressing all pathologies, results of arthroscopic repair rival that of open repair.

ARTHROSCOPIC REPAIRS FOR MULTIDIRECTIONAL INSTABILITY

Introduction

MDI was originally defined by Neer and Foster in 1980.[193] Their study group demonstrated instability anteriorly, posteriorly, and inferiorly. Capsular redundancy was demonstrated intraoperatively and was eliminated with an open humeral based capsular shift. Caspari first reported on arthroscopic techniques for MDI.[41,180] His technique utilized multiple sutures to shift the posterior–inferior glenohumeral ligaments, anterior–inferior glenohumeral ligaments, and anterior glenohumeral ligaments superiorly, thereby reducing capsular volume. Excessive capsular laxity is considered the primary pathogenesis of MDI. It can be congenital or acquired. Despite a general agreement that capsular laxity is the "essential lesion" of MDI, increased laxity alone cannot explain MDI, which often occurs in the midrange of motion when capsular ligaments are loose. Other contributing factors may include concavity–compression (muscle forces of the rotator cuff) and geometric conformity of the glenohumeral joint provided by labrum and articular cartilage.[180] Kim[132,133] identified labral pathology in all of his patients with either MDI or posterior–inferior instability. Additionally, there is evidence that the amount of translation in symptomatic shoulders with MDI is not significantly different from the laxity found in painless shoulders with MDI.[149,150,177] This implies that pathology other than capsular volume may contribute to painful MDI.[149,152,179]

Recurrence of Instability

Incidence

Reports of recurrent instability after arthroscopic treatment of MDI are scarce. Gartsman et al.[68] reported on 47 patients treated with multiple suture anchors for MDI with just 1 of 47 experiencing recurrent instability. McIntyre[181] reported a 5% recurrence rate using multiple sutures.

Arthroscopic transglenoid treatment was first described by Duncan and Savoie[52] and later by McIntyre et al.[180] McIntyre utilized a transglenoid approach in 19 patients over 34 months with anterior sutures brought out posteriorly and tied over the infraspinatus fascia, and the posterior sutures brought out anteriorly through a supraclavicular portal and down through a drill hole in the scapular spine and tied down over bone. Only 1 of 19 patients experienced recurrent instability. Treacy et al.[263] reviewed 25 patients treated with arthroscopic shift via the transglenoid technique. At 60 months of follow-up 22 of 25 had stable shoulders.

Causes of Recurrent Instability

Incorrect Diagnosis

As with open repair, incorrect diagnosis may lead to recurrence. The physician should note the primary direction of instability during provocative testing. Volitional dislocators should be identified and those with psychiatric problems must be identified. Connective tissue disorders such as Marfan's or Ehlers-Danlos syndrome must be elicited. True scapular winging must be diagnosed if present.

Surgical Errors

The pathology of MDI that can be addressed arthroscopically includes increased capsular volume, labral deficiencies, and a deficient rotator interval. Failure to address these variables may result in a technical failure. Suture "pinch-tuck" imbrication and pinch-tuck plication of the capsule to the labrum combined with rotator interval closure is an effective method of achieving these surgical goals. Kim[133] reported good or excellent outcomes in 30 of 31 patients studied over 51 months using this technique. Gartsman et al.[68] was 94% successful with 47 patients studied over 35 months utilizing these techniques. Treacie et al.[263] reported on 25 patients with MDI after arthroscopic stabilization followed for 5 years with 88% satisfaction and 12% resubluxation but no dislocation.

Conclusion

Treatment of MDI first requires patience. A trial of nonoperative rehabilitation emphasizing scapular stabilizers and rotator cuff strengthening should be employed with anticipated success rates of 80%. If instability persists after 6 months, arthroscopic stabilization may be undertaken. Preoperative gadolinium MRI may confirm labral lesions or loss of the chondrolabral contour, suggesting tears. This is especially true posterior–inferior. Examination under anesthesia should confirm laxity in all three directions and be compared to the contralateral extremity. Arthroscopic labral repair and capsular shift (posterior and anterior) and rotator interval closure is then completed. Rehabilitation for a period of 4 to 9 months is conducted before contact sports allowed. With advanced arthroscopic skills, success rates over the short and intermediate term of 80% to 95% can be anticipated. Perhaps the greatest advantage of arthroscopic evaluation and treatment of MDI is that it allows treatment of a spectrum of pathoanatomy. With MDI, variation of pathoanatomy is common.

ARTHROSCOPIC REPAIRS FOR POSTERIOR INSTABILITY

Introduction

Posterior shoulder instability is present in approximately 2% to 4% of all instability cases. It represents a continuum of problems that remains poorly understood.[280] No single lesion is thought responsible for the development of posterior instability. Most researchers distinguish between acute traumatic dislocations and chronic atraumatic posterior instability. The former is typically caused by a single traumatic event while the latter develop insidious laxity and instability over time. Pain and instability are caused from abnormal posterior translation of the humeral head relative to the glenoid. Rehabilitation of the rotator cuff, especially the infraspinatus, remains the first line of treatment for posterior instability. If rehabilitation fails and symptoms persist, operative intervention including posterior Bankart repair and/or capsulorrhaphy may be indicated.

Recurrence of Instability

Recurrence of instability for arthroscopic repair techniques is extremely variable. Rates of 0% to 50% have been reported.[10,81,128,280] These high rates of recurrence may reflect the technical difficulty associated with addressing all potential sources of posterior instability. It may also reflect study groups with different types of posterior instability. In general, recurrence after arthroscopic shoulder repair is secondary to incorrect diagnosis/patient selection, surgical errors, or inadequate rehabilitation.

Etiology of Instability Recurrence

Incorrect Diagnosis

Failure to recognize MDI instead of unidirectional posterior instability may lead to surgical failure. Patients with posterior instability typically complain of mild shoulder pain, mechanical symptoms (catching, clicking, clunking), and laxity in positions of flexion, adduction, and internal rotation. Trauma with the arm in this position is usually described. They usually present in their teens or 20s. They may or may not have pain. Hawkins[111] reported that 40% of patients with posterior subluxation had no complaints of pain. This was true even in the face of ongoing subluxation. Activity-related pain or discomfort is common and ranges from dislocation to repeated subluxations. Physical examination should confirm unidirectional posterior instability.

Failure to recognize glenoid hypoplasia, retroversion, or posterior erosion may lead to surgical failure if one utilizes a soft tissue–only procedure. Standard radiographs, including anteroposterior, lateral, and axillary views should be obtained to rule out a retroverted, posteriorly eroded, or dysplastic glenoid. In the setting of failed arthroscopic repair for posterior instability, a CT scan should be considered to assess glenoid version and posterior glenoid erosion. If greater than 20 degrees of retroversion is present, a soft tissue–only procedure may lead to high recurrence. MRI with gadolinium enhancement is useful to evaluate the posterior–inferior capsulolabral complex. Kim et al.[129] identified four types of posterior labral pathology identified on MR arthrogram.

Kim et al.[129] also found that retroversion of the chondrolabral glenoid was present in posterior instability (5 to 8 degrees) when compared to controls (1.7 to 2.9 degrees). Failure to correct for labral pathology and/or chondrolabral retroversion may lead to increased recurrence.

Failure of Operative Technique

Surgical intervention begins with an examination under anesthesia. Cordasco et al.[45] studied patients with isolated

posterior or inferior labral tears but no clinical instability. On examination under anesthesia, all had posterior instability. MDI, if present, should be confirmed and the direction of maximal translation recorded and correlated to the patient's symptoms. Failure to identify MDI may lead to recurrence. Arthroscopically, attention is paid to defects in glenoid concavity or other bony pathology. The posterior–inferior labrum is thoroughly assessed. Lesions in this location may vary from labral tears to more subtle deep lesions on the chondrolabral junction (Kim's lesion). Kim's lesion may be evident after liberating the superficial chondrolabral surface and thus exposing the loose underlying labrum. Failure to rectify all potential pathology may lead to recurrence.

Suture anchor capsulorrhaphy restores the posterior–inferior labral height and capsular tension. In the revision situation, the repair may be performed arthroscopically or open. The repair may be augmented with suture capsulorrhaphy. Kim et al.[1] recommended augmenting the repair with a posterior capsular shift, anterior capsular shift, and rotator interval (RI) closure if anterior translation and/or inferior translation coexist. They studied 31 patients over 51 months. Thirty of 31 had good or excellent results by the Rowe grading system with 3% recurrent instability. Williams et al.[280] studied 27 shoulders over 5 years treated with posterior suture anchor capsulorrhaphy and found 8% recurrence of instability. Mair et al.[164] studied nine patients treated in similar fashion with no recurrence at a minimum of 2 years of follow-up. Goubier et al.[81] treated 11 patients for posterior instability with suture anchor capsulorrhaphy and had no recurrence of instability at 34 months.

Although there are several techniques available for arthroscopic suture capsulorrhaphy, either with or in the absence of a labral lesion, we prefer a "pinch-tuck" technique. Through the posterior portal a Linvatec Spectrum (Linvatec, Largo, FL) device is introduced and pierces the capsule approximately 1 cm lateral to the labrum. The hook then reenters the capsulolabral junction and sutures are tied arthroscopically. Care is taken to incorporate the posterior IGHL and to achieve a shift of the capsule superiorly. If the posterior capsule is tenuous or torn, the infraspinatus tendon may be incorporated into the repair. Wolf and Eakin[286] reported on 14 patients treated with either suture anchor capsulorrhaphy or suture capsulorrhaphy with recurrence in one patient over 33 months. Posterior capsular redundancy was found in all patients and labral pathology was present in 86%. Provencher et al.[211] reported on 35 patients treated likewise at follow-up after 25 months and found good or excellent results in 31 of 35. There were five recurrences.

Role of the Rotator Interval

Cadaveric models have been used to study the role of the rotator interval on stability. Harryman et al.[92] found that sectioning the rotator interval capsule allowed increased amounts of laxity in all three directions. They also found that imbrication of the interval decreased inferior and posterior translation. Field et al.[57] reported on 15 patients with MDI whose intraoperative findings were only a hole or defect in the rotator interval capsule. Closure of this defect led to good stability at average follow-up of 3.3 years. Rowe and Zarins[226] identified holes in the rotator interval in 20 of 37 patients undergoing open procedures for instability. Kim et al.[129] recommended RI closure on patients with large sulcus and predominantly inferior instability. Failure to address a rotator interval defect may be associated with instability recurrence.

Conclusion

Unlike anterior instability, there likely is no agreed upon "essential" lesion of posterior instability. Several lesions may contribute to posterior–inferior instability and including capsular laxity, stripping of the capsule and synovial tissue, erosion of the cartilage, labral tears or bony defects, and bony or chondrolabral retroversion.[10] This variation in pathology makes treatment decisions difficult and failure to address the pathology can be associated with instability recurrence. Preoperative CT scans and MR arthrograms should be considered for patients who have failed prior attempts at soft tissue repair to evaluate glenoid version, hypoplasia, posterior glenoid defects, labral pathology, and chondrolabral retroversion. For glenoid retroversion of greater than 20 degrees, glenoid osteotomy with a soft tissue procedure should be considered. Lesser degrees of retroversion may be treated with suture anchor capsulorrhaphy plus or minus capsular shift to restore labral height and chondrolabral version. Arthroscopic management of posterior instability should address all potential causes of instability including posterior labral defects, capsular redundancy, and the rotator interval defects. Success using arthroscopic techniques for posterior instability range from 75% to 94% at short and intermediate follow-up.

Neurovascular Injury

Neurovascular injury has been an infrequent but potentially devastating complication of arthroscopic instability repair. These complications can be separated into those resulting from positioning and setup, portal placement, or suturing techniques.

Positioning

Shoulder arthroscopy has classically been performed in either the lateral decubitus or beach-chair position. The lateral decubitus position has a potential disadvantage in that the necessary use of a traction device may endanger the brachial plexus.[272] Andrews and others have recommended that longitudinal traction of 15 lb (6.8 kg) be

applied with the patient in the lateral decubitus position and with the arm in 15 degrees of forward flexion and 70 degrees of abduction.[9] Traction setup used for shoulder arthroscopy in the lateral position should not exceed 15 lb, with 10 lb usually being sufficient. Careful attention to excessive shoulder abduction and extension should be paid to prevent potential neuropraxia.[22,267] It is recommended that traction be maintained at less than 20 lb (9.1 kg) to minimize the risk of neurapraxias.[222] The beach-chair position has been used by many surgeons, and no episodes of brachial plexus nerve palsy have been reported. There is one case report of a hypoglossal nerve palsy, thought to result from change in position of the head and subsequent compression beneath the angle of the mandible.[192] The reported rate of neurapraxia after shoulder arthroscopy has ranged from 0% to 30%.[9,135,203,209,222,242,243] Small reported on a prevalence of less than 0.1% of neurologic complications. These included three brachial plexus traction injuries (two subsequent to arthroscopic anterior staple capsulorrhaphy) and one axillary nerve injury (during an arthroscopic anterior acromioplasty).[243] Ogilvie-Harris and Wiley reported one transient musculocutaneous nerve palsy after a total of 439 shoulder arthroscopies, a prevalence of 0.2%.[203] Andrews and Carson reported on three neurologic injuries, one musculocutaneous neurapraxia and two involving the ulnar nerve, after 120 arthroscopies, a prevalence of 3%.[8] Klein et al. cited a prevalence of transient paresthesias of about 10%; however, no specific numbers were given.[135] Pitman reported on two transient neurapraxias in 20 patients, one involving the lateral antebrachial cutaneous nerve and the other with diffuse hypoesthesia of the lateral aspect of the arm and forearm with paresis of the wrist extensors.[209] Weber and Jain[276] studied interscalene blocks for routine arthroscopic shoulder surgery and found a 13% failure rate in 218 patients. They further reported high use of postoperative narcotics despite use of the block and included reports of cardiovascular collapse, pneumothorax, and permanent nerve injury when using this technique. They concluded that complications of interscalene blocks necessitate a detailed discussion of risks and benefits with the patient prior to attempting this technique.

Klein and colleagues studied the strain on the brachial plexus that results from traction loads applied at various arm positions and correlated this with visibility through the arthroscope. They mounted strain gauges to the upper trunk, lateral cord, median nerve, and radial nerve of five fresh human cadavers. Each cadaver was placed in a lateral decubitus position, with the head fixed in a neutral position, as for shoulder arthroscopy, and the strain on the plexus was measured as a function of arm position and traction load. A final cadaver was used to determine the accessibility and visibility in the arthroscope at each of the arm positions. At a given flexion angle, increasing abduction was associated with decreasing strain. At a given abduction angle, increasing flexion resulted in decreased strain. The minimum overall strain was noted at 90 degrees of flexion and 0 degrees of abduction. Visibility at this position was limited. Klein et al. concluded that the ideal arthroscopic position is a combination of two positions that would maximize visibility while minimizing strain to the nerves. These two positions are 45 degrees of forward flexion and 90 degrees of abduction in combination with 45 degrees of forward flexion and 0 degrees of abduction.[135]

Somatosensory evoked potentials (SEPs) were recorded by Pitman during shoulder arthroscopy in 20 patients to monitor the musculocutaneous nerve, ulnar nerve, and either the median or radial nerve. In all 20 cases, abnormal SEPs of the musculocutaneous nerve were demonstrated. In 16 patients, this was produced on initial joint distention; in 15 patients, by traction; in 11, by longitudinal traction of greater than or equal to 121 lb; and in 6, by perpendicular traction of greater than or equal to 7 lb. In 10 patients, there were varying combinations of median, ulnar, and radial nerve involvement. There were two cases of clinical neurapraxia in this series. One resolved in 24 hours and one in 48 hours. Pitman concluded that there is a real potential for neurologic damage during shoulder arthroscopy and that the musculocutaneous nerve is the most vulnerable. Factors responsible include joint distention, excessive traction, extravasation of fluid, and tension on the musculocutaneous nerve as it was stretched over the humeral head with the shoulder in extension, abduction, and external rotation.[209]

Because most neurologic insults are neurapraxic-type injuries, the initial approach should be observation. If no nerve recovery is seen by 3 weeks, one should consider an EMG, and then serial EMGs based on the clinical examination. In neurapraxic injury, the surgeon should expect neurologic function to be fully recovered by approximately 3 months.

Portal Placement

Knowledge of the local anatomy is essential in portal placement. Matthews and others reported on one proximal median nerve palsy in 47 shoulder arthroscopies, a prevalence of 2%. The presumed cause was injury during anterior portal placement.[173] For anterior portals, staying superior and lateral to the coracoid within the "safe triangle" avoids injury to the musculocutaneous nerve.[173,202] Wolf has done cadaveric studies describing an anteroinferior portal that lies 1.5 to 4 cm from the musculocutaneous nerve, and provides a more useful working angle for stabilizing the anteroinferior labral ligamentous complex. This distance increases with adduction; thus, he recommended placing this portal with the arm in less than 30 degrees of abduction.[172,289]

Laceration of the cephalic vein may also occur during anterior portal placement. Ligation is indicated, and

meticulous hemostasis intraarticularly is essential to prevent hemarthrosis. The use of epinephrine in the irrigating fluid, proper joint distention, and hypotensive anesthesia may also aid in controlling bleeding.[22]

Suturing Techniques

Arthroscopic stabilization procedures can be complicated by neurovascular injury. The most notorious of these complications is injury to the suprascapular nerve during transglenoid suturing. Mologne et al. reported the clinical outcome of arthroscopic labral reconstruction using a transglenoid suture technique in 49 shoulders.[187] Suprascapular nerve palsy occurred in three cases (6%).

Bigliani and colleagues studied the course of the suprascapular nerve and its distance from fixed scapular landmarks in 90 cadaveric shoulders. In an additional 15 cadavers, three pins were passed at various angles in an anterior-to-posterior direction through the middle of the glenoid neck just inferior and lateral to the base of the coracoid process. The distance between the exit site on the posterior glenoid neck and the suprascapular nerve at the base of the scapular spine was recorded for each pin. Inferiorly directed pins were the furthest from the suprascapular nerve and averaged 16 mm. On the basis of these data, a relative safe zone was described in the posterior glenoid neck.[24] Morgan recommended that the anterior starting point for transglenoid drilling should be at the 2 o'clock position (right shoulder) and directed 30 degrees inferior to the transverse plane and 15 degrees medial to the plane of the glenoid to minimize injury to the suprascapular nerve.[189]

In patients undergoing transglenoid drilling, the presence of suprascapular nerve injury postoperatively should signal a prompt workup. An EMG may be indicated in the first 24 hours to rule out axonotmesis. If no recovery is seen in 24 to 48 hours, early exploration should be considered.

Hardware Complications

Mechanical Complications

Numerous hardware problems have been seen after arthroscopic staple capsulorrhaphy. The earliest staples were made malleable so that they would not break. This proved to be a problem in that they were easily bent by forceful direction change during insertion. Subsequent generations were less malleable and thus the complication of staple loosening (usually as a result of reinjury or dislocation), impingement, breakage, and eventual migration was seen.[123]

Another staple complication involved placement of the staple in an incorrect perpendicular orientation to the anterior glenoid. Even if oriented correctly, the staple may be placed only partly (one tine only) in bone or be placed too deep into the bone, thus amputating the glenohumeral ligaments or cutting into the labrum. If placed too superior, the staple may end up in the coracoid process. The staple head may also cause irritation to the subscapularis tendon. Placement of the metallic staple at the anterior glenoid margin may cause erosion of the humeral head cartilage (Fig. 16-35).

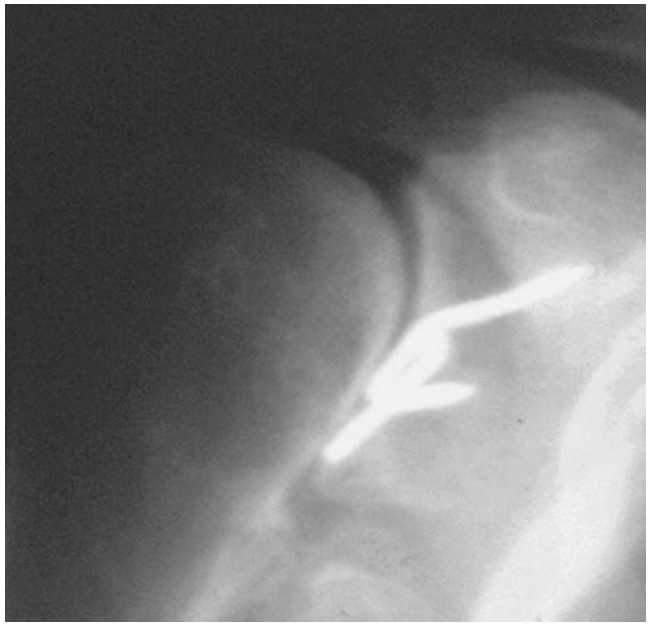

Figure 16-35 Radiograph demonstrating a painful intraarticular staple that had been placed arthroscopically.

Clearly, any implant can loosen and cause pain or chondral injury. This complication is not specific to staple capsulorrhaphy. If a patient who has undergone instability repair with a metallic implant develops pain, crepitation, or locking sensation, radiographs and possibly a CT scan are mandatory to ensure the implants are secure in bone (Fig. 16-36). For a patient that has had prior placement of absorbable anchors

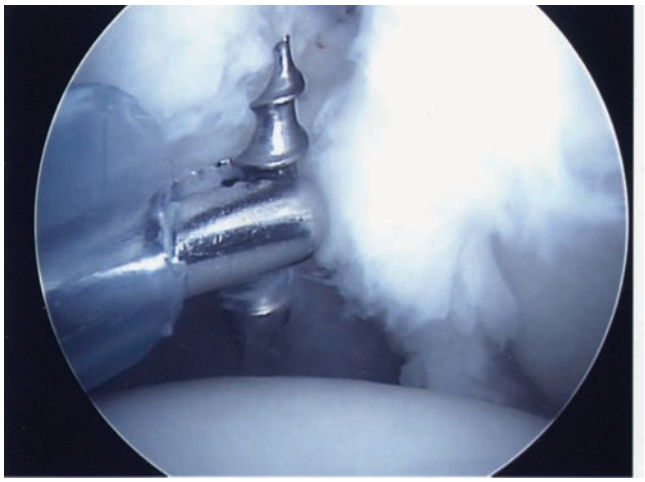

Figure 16-36 Loose metallic anchor after prior arthroscopic Bankart repair. There was significant chondral wear found during arthroscopic anchor removal.

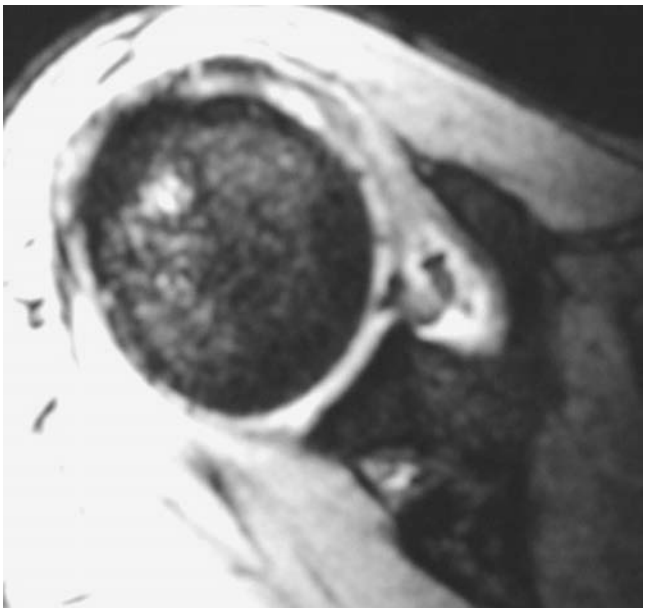

Figure 16-37 Magnetic resonance imaging scan demonstrating a loose absorbable anchor in the subcoracoid recess.

or tacks, an MRI scan should be obtained to investigate any mechanical complaints (Fig. 16-37).

Biologic Response to Absorbable Implants

In recent years, bioabsorbable implants have been used in arthroscopic stabilization procedures with good success. Bioabsorbable anchors have some potential advantages over metallic implants. Gradual load transfer to healing tissues, radiolucency, and reduced need for hardware removal are reported advantages. Both polyglycolic acid (PGA) and polylactic acid (PLA) tissue anchors are currently available for use. PLA has a slower biodegradation rate than PGA. They are not without potential complications. By definition, absorbable devices erode from a biologic response. If this response becomes exaggerated, clinical implications may result. Although clinically significant reactions to polyglyconate and polylactic acid anchors have been infrequent, they clearly can occur (Fig. 16-38). As seen with other biomaterials, the extent of the biologic reaction is usually material-specific.

Warner et al. reported two cases of biopsy of the repair site on "second-look" arthroscopy 6 to 8 months after labral repair using the Suretac device, a polyglyconate absorbable suture anchor. The authors found residual polyglyconate polymer debris surrounded by a histiocytic infiltrate.[268] Tetik et al. reported on humeral head damage after Suretac placement for a SLAP lesion 2 years after index procedure. Arthroscopy revealed prominent indentation at the implant site, hypertrophied labral tissue, and humeral head wear.[257] Edwards and colleagues have reported on adverse reactions to the Suretac absorbable anchor in five of their patients. All patients complained of increasing pain and loss of shoulder motion. They all required arthroscopic lavage and débridement of the intracapsular synovitis. Nonspecific granulomatous reactions were identified histologically in all cases and no organisms were grown from the operative specimens. One specimen had a dense inflammatory response surrounding refractile material (presumably particles of the Suretac).[33]

Poly-L-lactic acid has also been implicated in hardware failure. Wilkerson et al.[279] reported on four patients treated with polylactic acid tacks. Three had polylactic acid anchors placed for arthroscopic SLAP stabilization and one for a rotator cuff tear. All four had evidence of hardware failure and tack debris 3 to 10 months postoperatively at second-look arthroscopy. The authors theorized that increased demand across the shoulder joint and increased absorption rate of the tacks in soft tissue led to implant fracture and failure.

Any patient who develops symptoms of worsening pain, decreasing motion, increasing warmth, or erythema within 4 to 6 weeks of surgery should have an infection workup. In addition, one should consider an early arthroscopic exploration with removal of residual anchor debris and synovectomy.

Miscellaneous Complications

Fluid extravasation into the adjacent tissues is a very common complication of shoulder arthroscopy. This fluid may

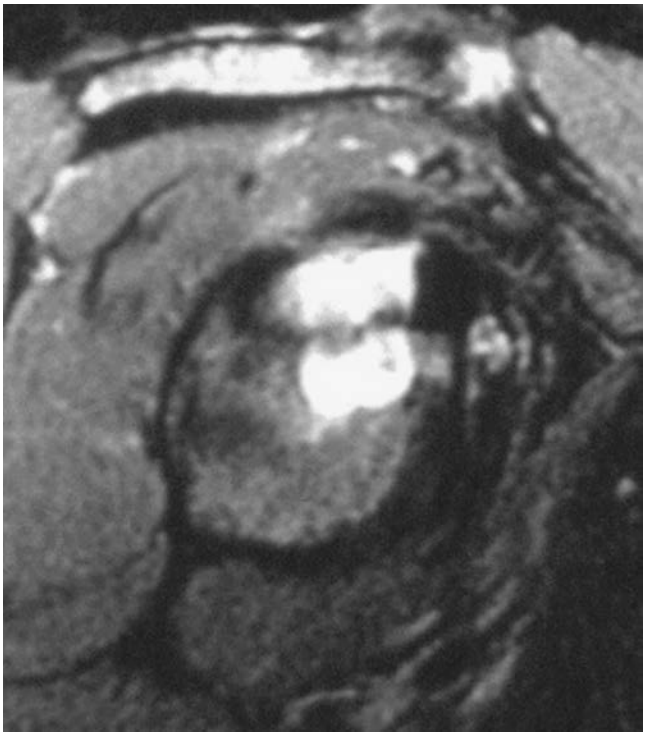

Figure 16-38 Magnetic resonance imaging scan demonstrating fluid collection and osteolysis around two absorbable Suretac devices.

increase compartment pressures in the arm, potentially leading to compartment syndrome. Lee and colleagues measured the intramuscular deltoid pressure using a slit catheter. Although they found that the intramuscular pressure became transiently elevated during the arthroscopic procedures, in every case the pressures promptly returned to normal levels within 30 minutes postoperatively. The clinical swelling and tenseness remained for a longer period. Despite a sometimes alarming amount of swelling during shoulder arthroscopy, intramuscular pressure elevations were only sustained for a short time and returned to normal levels very quickly after the cessation of fluid infusion.[145] There are several ways to control this extravasation, including making small, tight portals around cannulas, decreasing operative time, and carefully monitoring fluid inflow pressures.[123] Case reports of compartment syndrome have been presented.[208] Injury to the rotator cuff is also a possible consequence of incorrect portal placement. These punctures may produce permanent rotator cuff defects. Creating a cuff defect is best avoided by making the superior portal with the arm adducted and the posterior portal with the arm adducted and internally rotated.[201]

COMPLICATIONS OF THERMAL CAPSULORRHAPHY

Recurrence of Instability

Introduction

The use of thermal energy to shrink lax or redundant connective tissues is not new. Hippocrates treated recurrent shoulder instability with a red-hot iron inserted into the axilla.[167] Thermal shrinkage of capsular tissue has been proposed as a means to reduce capsular redundancy associated with shoulder instability. The goal of such a procedure, either primary or adjunctive, is to restore stability to the shoulder with similar success of more traditional open or arthroscopic means.

Initial reports suggested highly successful clinical application of this technique based on short-term follow-up. Thabit et al.[258] used a holmium:YAG laser to achieve 90% good or excellent results at 6 months. Lyons et al.[161] reported 89% satisfactory results at an average follow-up of 27 months. Fanton and Khan[55] were the first to use a radiofrequency (RF) probe and reported 90% excellent results in 54 patients at a 2-year follow-up. Patients with MDI had a 90% success rate at 1 year, which dropped to 75% by year 2. Their best results were in unidirectional instability of "mild to moderate" degree. Their success in these patients was high, with a 5% reoperation rate.

Although early reports were optimistic, concern over failures, especially in MDI, have been observed with longer follow-up. D'Alessandro et al.[48] studied 84 patients with traumatic anterior instability, recurrent anterior–inferior subluxation, and multidirectional instability for an average of 46 months. Overall unsatisfactory American Shoulder and Elbow Surgeons (ASES) scores at 3 years were found in 37% of the patients and 12% underwent revision surgery for recurrent instability. The failures in the MDI group reached 41% and repeat surgery in this group resulted in only a 50% satisfaction rating.

Levy et al.[148] reported a 36% failure rate for laser-assisted treatment of MDI and 24% failures in those treated for MDI with an RF probe. Frostick et al.[64] reported a 16% failure rate at 26 months for patients with MDI, and Miniaci and McBirnie[184] reported recurrence of instability in 9 of 19 patients treated for MDI at 9 months.

Postoperatively it may take as long as 12 weeks for thermally modified tissues to regain normal strength.[234] Connective tissues show an immediate loss of stiffness after thermal shrinkage. More shrinkage results in a greater loss of stiffness. Thus, tissues treated in this fashion become weaker and more compliant in the postoperative period. When thermally treated tissues are subjected to physiologic loads postoperatively, within 4 weeks they can stretch to preoperative length.[234] One study has demonstrated increased reducible cross-links in pathologic tissue. This would suggest that pathologic tissue is more susceptible to thermal shrinkage and may be weaker in the immediate postoperative period.[103]

Results of thermal shrinkage as an adjunctive procedure have been more encouraging. Mazzocca et al.[174] treated 18 collision athletes with unilateral anterior instability with arthroscopic stabilization and adjunctive thermal treatment of the anterior–inferior glenohumeral ligament. Two of 18 experienced redislocation over a 2-year period. Savoie and Field[233] studied MDI in 30 patients treated with thermal shrinkage and arthroscopic suture plication of the rotator interval. At 28 months 93% of the patients were satisfied with the results. Gartsman et al.[69,70] reported on the use of thermal or laser energy for both MDI and bidirectional instability as an adjunct to arthroscopic suture techniques. Limited use of a holmium:YAG laser to augment capsular tightening resulted in 91% good or excellent results at 34 months of follow-up. In another study by Gartsman et al.,[69] 94% of patients had good or excellent results at 35 months when rotator interval closure was combined with RF thermal treatment.

Capsular Necrosis

Case reports of capsular necrosis and chondrolysis following thermal capsulorrhaphy have been reported[13] (Fig. 16-39). This devastating complication makes revision surgery difficult. For mild cases, areas of capsular necrosis can simply be closed, either arthroscopically or open. For more advanced cases of capsular necrosis, capsular reconstruction with auto or allograft tendon is necessary.

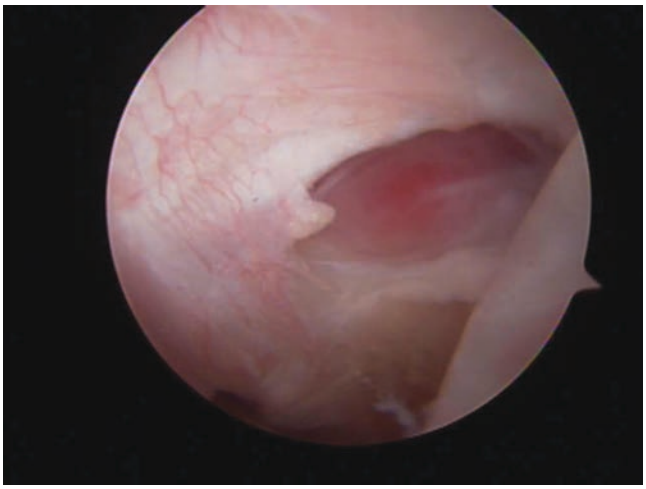

Figure 16-39 Capsular necrosis in a patient who had undergone a thermal capsulorrhaphy for multidirectional instability.

Chondrolysis

The relationship between chondrolysis and use of a thermal device is controversial. The temperature of arthroscopic fluid during thermal capsulorrhaphy can influence chondrocyte survival. Lu et al.[160] studied the temperature of the lavage solution while utilizing monopolar radiofrequency probes to smooth grade II chondromalacia changes in human knees. A higher lavage temperature (37°) resulted in less chondrocyte death than a lower lavage temperature (22°). The authors concluded that a higher lavage temperature allowed the monopolar probe to reach the preset temperature in a shorter time, thereby allowing less energy delivery to the tissues. Both temperatures, however, caused chondrocyte death.

Two patients in a study by Levine et al.[147] experienced glenohumeral joint destruction within 12 months of thermal capsulorrhaphy. Both patients went on to glenoid resurfacing with lateral meniscal allograft and humeral resurfacing with Copeland resurfacing humeral arthroplasty. This complication, while rare, is likely much more common than the few case reports would suggest. Unfortunately, given that thermal capsulorrhaphy is usually performed in the young, chondrolysis is devastating (Fig. 16-40).

Nerve Injury

Axillary nerve injury after thermal capsulorrhaphy was studied by Williams et al.[291] and found to be 1.4%. Most cases involved the sensory portion of the axillary nerve with resolution within 2 to 3 months. Anatomic studies by Bryan et al.[30] demonstrated that the axillary nerve is on average only 3 mm from the inferior aspect of the capsule. The amount of radiofrequency energy needed to impose irreversible axillary nerve injury is not known. Caution should be exercised if shrinking this portion of the capsule. Most authors recommend either "striping" this area or using the probe only on the posterior portion of the inferior glenohumeral ligament.

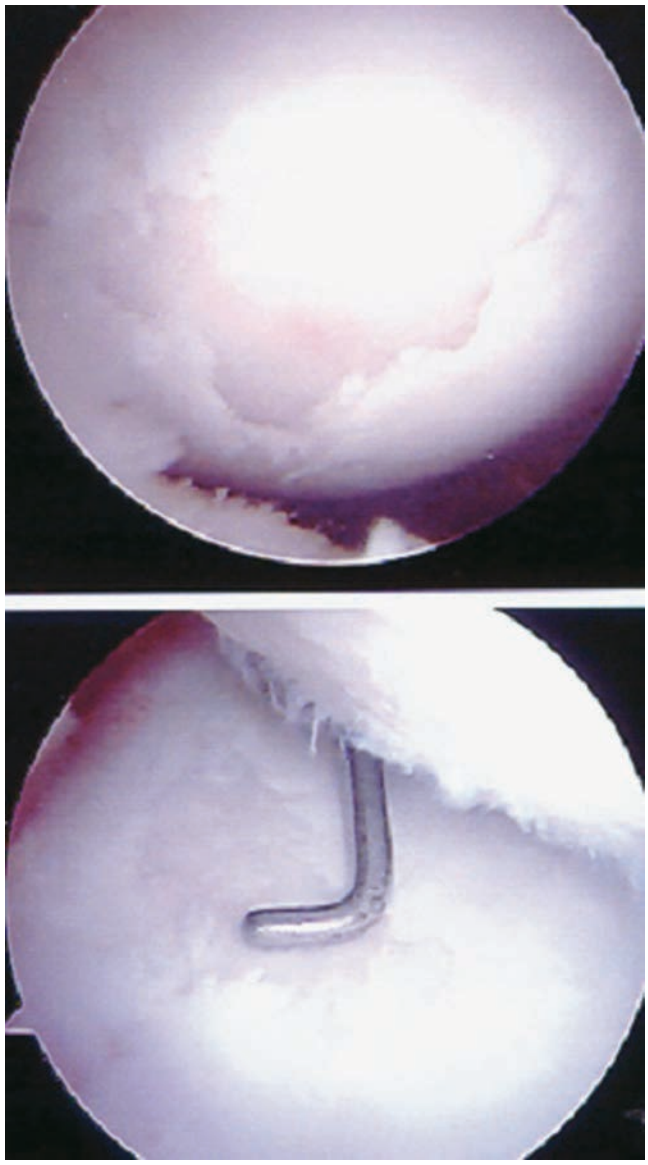

Figure 16-40 A 16-year-old baseball pitcher with potential for a scholarship developed worsened pain after a thermal capsulorrhaphy. Arthroscopic evaluation revealed significant chondrolysis.

Conclusion

Use of thermal energy as a primary method for arthroscopic treatment of instability is no longer recommended. Its use to augment more traditional means of restoring stability is a matter of ongoing debate and the surgeon should proceed with caution. It should be remembered that thermal shrinkage occurs at 65°C but significant cell death can occur at temperatures as low as 45°C. Thus, even when "striping" the capsule and ligaments, a significant amount

of cell death may occur even in untreated tissues. Treated tissues lose stiffness postoperatively and are subject to creep, which limits rehabilitation for up to 3 months. Complications including recurrence (16% to 37%), axillary nerve injury, or chondrolysis have been described. Revision surgery, if needed, is generally more difficult, with attenuated or ablated capsule reported in 18% to 33% of patients requiring revision surgery after failed thermal capsulorrhaphy.[291]

PATIENT EVALUATION AFTER FAILED INSTABILITY REPAIR

It is clear that "failure of instability surgery" encompasses multiple diagnoses. A careful and thorough approach is necessary to elucidate the problem and arrive at the appropriate treatment plan, particularly if the problem is recurrent instability or motion loss (Figs. 16-41 and 16-42).

History

The most important aspect of the patient history is questioning about the initial instability event. It is clear from the literature that failure of diagnosis is the most common cause of error. The patient, therefore, should be questioned about the activity that caused initial dislocation, the position of the arm at the time of dislocation, and the severity of the initial trauma. The physician should question about subsequent instability events, again determining arm position during these incidents. A history of contralateral shoulder instability may indicate multidirectional instability.

The patient should be questioned in detail about current symptoms. Once the unique feeling of instability is defined for the patient as such, it is a feeling that is never forgotten, that sensation forming the basis for the apprehension test. The patient should be questioned as to whether he or she still feels unstable. Was there a period after the operation when he or she was improved? Was there a significant

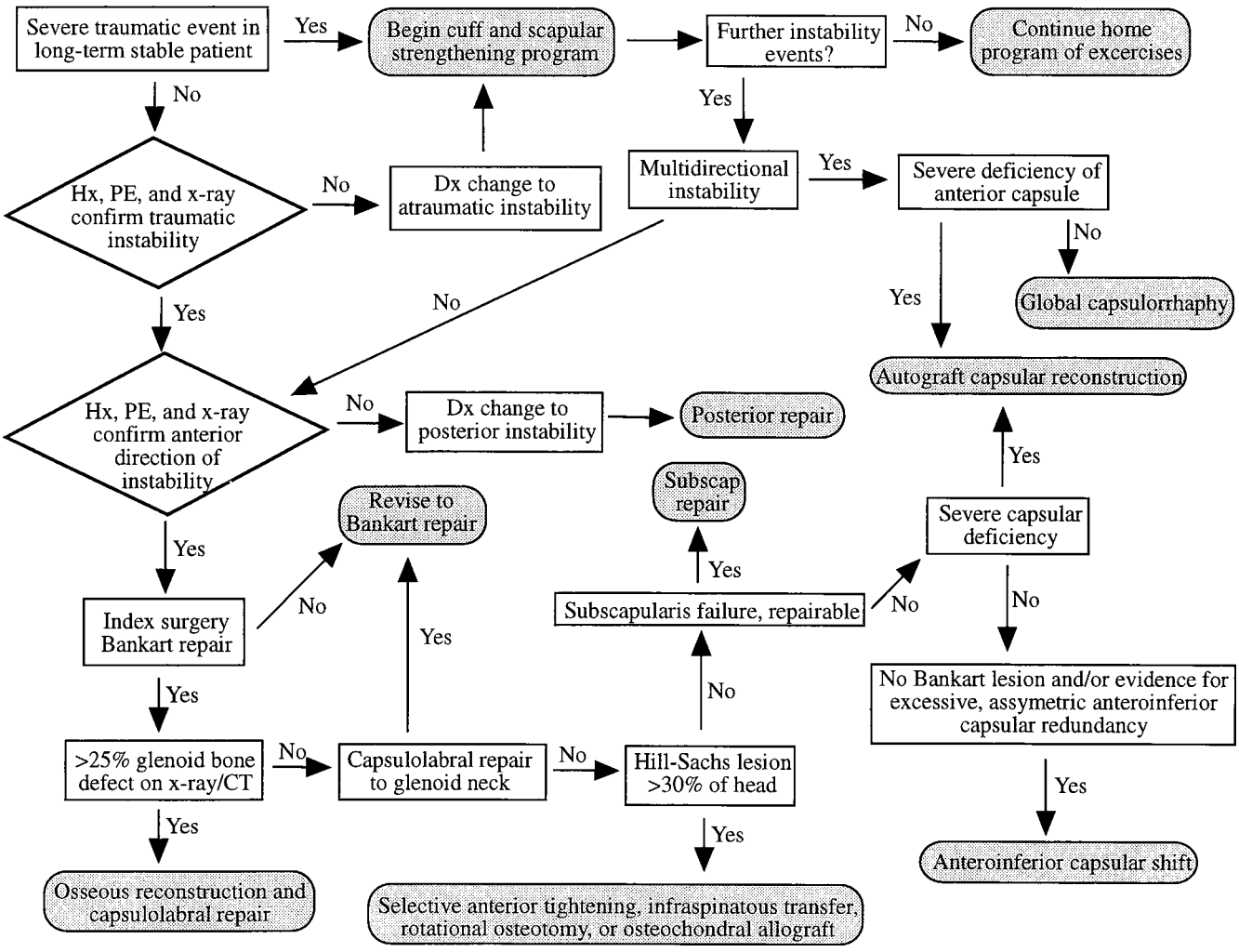

Figure 16-41 Algorithm for the management of recurrent instability after previous surgical repair.

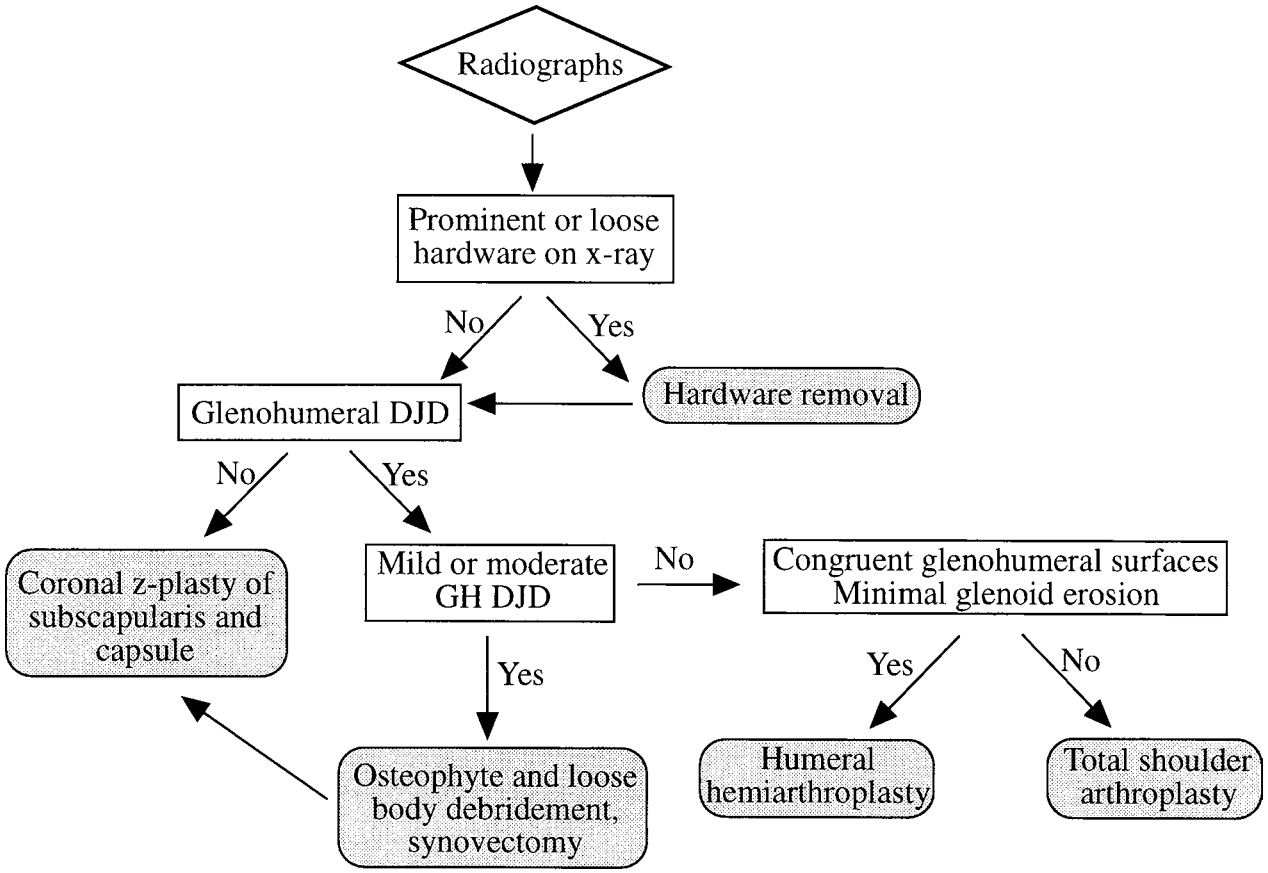

Figure 16-42 Algorithm for the management of motion loss (external rotation less than neutral) after previous instability repair.

postoperative traumatic event? Which humerothoracic positions are associated with instability events?

In cases of stiffness, the degree of functional impairment should be assessed. The patient should be questioned on the difficulty with activities of daily living, sporting activities, and hobbies. One should attempt to ascertain the degree of patient compliance with his or her index postoperative rehabilitation.

Operative notes from prior procedures should be obtained. Those notes should be reviewed to determine the exact approach used and intervals incised as, in cases of postsurgical contracture, these locations are likely to contain the most adhesions. Knowing the technique of prior repair can help determine the cause of failure in situations of postoperative instability.

Physical Examination

The shoulder should be observed for prior incisions. A thorough examination for range of motion should be performed. Severe limitation of external rotation indicates a "too-tight" repair. Increased passive external rotation in the position of humerothoracic adduction suggests subscapularis failure. A complete examination for instability is necessary, concentrating on laxity tests and directions of apprehension. The patient should be asked to voluntarily dislocate the shoulder. If he or she is able to perform this maneuver with muscular contraction and not by arm positioning, the patient should be questioned further to elucidate a true history of voluntary instability. Tests of rotator cuff function are necessary, specifically the lumbar lift-off test for subscapularis integrity. A complete neurologic examination is indicated, concentrating on axillary and musculocutaneous function.

Diagnostic Tests

Every attempt should be made to obtain radiographs taken from before the index operation, for these studies may assist in either confirming or correcting the diagnosis. If hardware was used at the time of index operation, initial postoperative radiographs should be reviewed and compared with recent studies. In our practice, new radiographs including a true scapular anteroposterior view in both internal and external humeral rotation, an axillary lateral, a West Point lateral, and a Garth apical oblique view are

obtained. If the plain radiographic studies indicate an osseous glenoid deformity or a Hill-Sachs lesion, a CT scan is used to further define these abnormalities. An MRI scan is usually not needed in cases of failed instability, although it may be helpful in defining subscapularis failure in the face of an equivocal examination. To differentiate a recurrent labral tear, capsular laxity, or HAGL lesion, an MR arthrogram or even diagnostic arthroscopy may be necessary. If there is any examination evidence for neurologic dysfunction, EMG studies should be performed.

SUMMARY

Obtaining stability while retaining mobility is the goal of both arthroscopic and open instability procedures. The optimal method for accomplishing this is evolving. Lesions of the anterior–inferior capsulolabral complex can be effectively or ineffectively treated with open or arthroscopic procedures. Evaluating and treating the patient who has had a failed instability repair can be a daunting task. Broadly, surgical failures can be categorized into failure of diagnosis or patient selection, failure of surgical procedure, and failure of rehabilitation. A complete evaluation is needed to define the problem and determine appropriate solutions. The most important technique that will result in an excellent result from revision surgery is assuring the exact diagnosis. Once that diagnosis is confirmed, the appropriate surgical intervention can be applied. At revision, the surgeon should be prepared for a complete range of pathology, from soft tissue contractures and adhesions to osseous and soft tissue deficiencies. The goal in treatment is to restore stability and motion with minimal alteration to normal anatomy.

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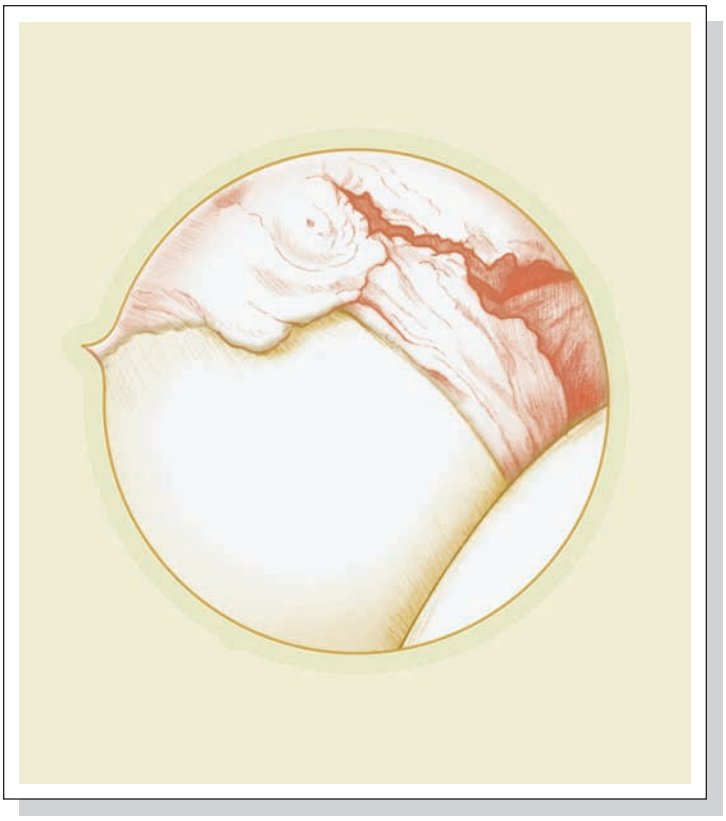
Frozen Shoulder

III



17 Diagnosis and Management of the Stiff Shoulder

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DEFINITION

Frozen shoulder or adhesive capsulitis is one of the most common, yet one of the most poorly understood disorders of the glenohumeral joint. This is primarily due to difficulty defining and differentiating it clearly from other conditions with similar symptoms and findings but with distinctly different causes. Codman described the disorder known as frozen shoulder as a "condition difficult to define, difficult to treat, and difficult to explain from the point of view of pathology."[26] Neviaser coined the term "adhesive capsulitis."[86] When he described the contracted, thickened joint capsule with chronic synovitus. The common teaching has been that the disease is a self-limiting condition with a natural history lasting 1 to 3 years.[103] Others have argued that 15% to 50% of patients have a persistent refractory course that is unresponsive to conservative treatment.[111]

Frozen shoulder is a condition of uncertain cause characterized by the spontaneous onset of pain with significant restriction of both active and passive range of motion of the shoulder.[103] In this chapter, frozen shoulder will be discussed in terms of classification, pathophysiology, clinical evaluation, treatment, and complications.

CLASSIFICATION

Classifying frozen shoulder into primary or secondary frozen shoulder can be difficult. Most cases of frozen shoulder have an idiopathic cause in an otherwise healthy individual, are characterized as primary, and can also be considered the classic presentation of adhesive capsulitis. Frozen shoulder is often divided into either primary frozen shoulder or secondary frozen shoulder[71] (Fig. 17-1). Secondary frozen shoulder is often defined with an association with a

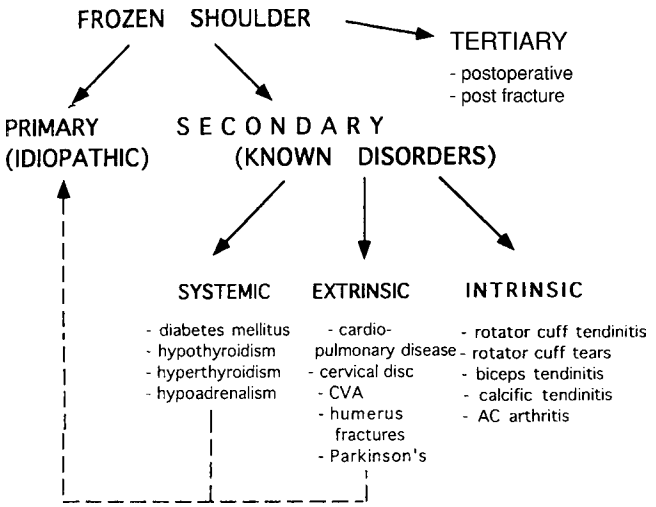


Figure 17-1 Proposed pathways for the development of frozen shoulder syndrome. AC = acromioclavicular; CVA = cerebrovascular accident.

known intrinsic, extrinsic, or systemic pathology. Secondary frozen shoulder could also be defined as a frozen shoulder that develops with an underlying intrinsic condition such as a rotator cuff tear, biceps tendon pathology, or calcific tendonitis. A third category has also been suggested for the stiff shoulder that develops after surgery or fracture (Fig. 17-1, schema). The postoperative frozen shoulder is often more resistant to both nonoperative and operative treatment. However, some patients do not fit clearly into one category or the other. Patients with a frozen shoulder and a small rotator cuff tear often are classified as having a secondary frozen shoulder but can often be treated as one would a primary frozen shoulder. Diabetic frozen shoulders often act differently than either a primary or secondary frozen shoulder. Future studies may need to address this to better characterize frozen shoulder so that cause and treatment can better be determined.

Reeves studied the natural history of 49 cases over 10 years and identified three phases of frozen shoulder.[103] Phase I consists of pain with progressive stiffness that lasted 2 to 9 months. Phase II is the stiff, contracted phase, which last 4–12 months. Phase III is the thawing phase where motion gradually improves over 12–42 months. Hannefin and Chiaia[44] identified four stages of adhesive capsulitis. Stage 1 occurs during the first 3 months of the disease. It is characterized by pain with range of motion with minimal or no loss of motion. Arthroscopy reveals diffuse synovitis of the anterosuperior capsule. Stage 2 is known as the freezing stage and occurs between 3 and 9 months after the symptoms begin. This stage is characterized by pain with decreased range of motion awake and under anesthesia with diffuse synovitis at arthroscopy. Stage 3 or "frozen stage" occurs at 9 to 15 months and is characterized by minimal pain except at the extremes of motion with loss of motion and a rigid end feel. Arthroscopy reveals a thickened, fibrotic capsule with no hypervascularity of the capsule. Stage 4 is the "thawing phase" with minimal pain and progressive improvement in range of motion. It occurs 15 to 24 months after the disease begins.[44]

PATHOLOGY

Shoulder motion is a combination of movements that occur at the glenohumeral and scapulothoracic joints at a ratio of 2:1. The normal shoulder is an inherently loose articulation that permits the large range of motion required for normal shoulder function. The capsule of the shoulder is normally a loose structure of which the surface area is almost twice that of the humeral head. The capsuloligamentous structures are the primary static stabilizers, especially at the extremes of rotation and translation. Thickenings of the joint capsule form the superior, middle, and inferior glenohumeral ligaments, while the tendons of the rotator cuff thicken the capsule anteriorly, superiorly, and posteriorly. At arthroscopy, several recesses may be identified in the normal shoulder. Anteriorly, a synovial recess is often present between the superior and middle glenohumeral ligaments, known as the rotator cleft or subscapular recess. There is a posterior outpouching of the capsule also, just deep to the infraspinatus muscle, known as the infraspinatus bursa. There is an inferior recess of redundant capsule forming a pouch-like fold when the arm is at the side.

In the pathologic state of frozen shoulder syndrome, the capsule becomes thickened and noncompliant with loss of the capsular recesses. Neer suggested that the tightened coracohumeral ligament restricts external rotation in frozen shoulder.[82] This contracted capsule does not allow the normal free movement of the shoulder which causes the scapula to move excessively in upward rotation to compensate for the loss of glenohumeral rotation as described by Nicholson.[90]

Numerous pathologic mechanisms have been proposed to explain the cause of frozen shoulder, but all remain largely theoretic. The syndrome of frozen shoulder was first described by Duplay in 1896. He introduced the term "scapulohumeral periarthritis," and felt that the initiating lesion was an obliteration of the subdeltoid bursa.[32]

Myer,[81] from a study of postmortem specimens, suggested that the initiating lesion was a breakdown on the intraarticular portion of the biceps tendon. His observations were supported subsequently by Pasteur[96] and Lippmann[65] and more recently by DePalma.[29] Codman, however, believed that the changes in the biceps tendon were of little etiologic significance.[26]

McLaughlin, one of the earliest investigators to describe the changes in the rotator cuff, stressed the importance of contracture of the subscapularis in the development of the syndrome.[76,77] Bateman reported his observations on the development of a hypertrophic inflammatory synovitis associated with intraarticular adhesions.[3] Several investigators

have proposed an autoimmune basis for frozen shoulder.[6,19-21,47] Although some clinicians have reported a high incidence of human leukocyte antigen B27 in patients with frozen shoulder,[19] others have not confirmed this association.[58,91,107,115] In later studies, serum immunoglobulin A (IgA) levels were significantly lower in patients with frozen shoulder, and the immune complex and C-reactive protein levels were increased.[6,20,21] In general, however, sufficient evidence to support immunologic therapy has been lacking.

A relation to myofascial pain syndrome has been proposed. A syndrome of active trigger points about the shoulder, specifically within the subscapularis muscle, has been suggested as a possible cause of frozen shoulder syndrome.[116] *Trigger points* are defined as locally tender, self-sustaining, hyperirritable foci located in the skeletal muscle or its associated fascia. The trigger points are also characteristically related to a zone of referred pain when the trigger is stimulated. Once activated, perpetuating factors may be responsible for the chronicity of pain. Another characteristic of the myofascial pain syndrome is palpable bands of muscle fibers that undergo a local twitch response when the trigger point is stimulated with a snapping palpation.[12,113,124]

Travell and others theorized that the subscapularis trigger points exert an influence on the sympathetic vasomotor activity, leading to hypoxia of the periarticular tissues. It is further theorized that the hypoxia leads to a local proliferation of fibrous tissue about the shoulder capsule, resulting in the clinical picture of frozen shoulder syndrome.[81,116] A biochemical basis for frozen shoulder has been proposed. Lundberg, in his analysis of the capsules from patients with frozen shoulder, found an increase in glycosaminoglycans and a decrease in glycoprotein content. These biochemical changes in the capsule, however, are consistent with the process of fibrosis, and they may represent the effect of frozen shoulder, rather than its cause.[70,71]

Neurologic dysfunction has been postulated to be a cause of frozen shoulder syndrome. In 1959, Kopell and Thompson[61] proposed suprascapular compression neuropathy as a possible cause of frozen shoulder, but electromyography (EMG) and nerve conduction studies have not supported this theory. Others have suggested that frozen shoulder is a result of autonomic dysfunction and represents a form a reflex sympathetic dystrophy.[107] Sufficient evidence to support these hypotheses has not been provided.

Bunker et al. prospectively studied 50 patients with the diagnosis of primary frozen shoulder. These authors were able to identify increased serum lipid levels in these patients compared with those of age- and sex-matched control subjects. The fasting serum triglyceride and cholesterol levels were significantly elevated in the frozen shoulder group. Increased serum triglyceride levels have also been found in patients with diabetes as well as Dupuytren's disease, suggesting that hyperlipidemia may be the common thread that links these three disorders.[22,23]

Various endocrine disorders are associated with frozen shoulder. In particular, patients with diabetes mellitus manifest a much greater incidence of frozen shoulder than their nondiabetic counterparts. Bridgman found that the incidence of frozen shoulder in 800 diabetic patients was 10.8%, compared with 2.3% in 600 nondiabetic controls.[15] A second study identified abnormal glucose tolerance test results for 28% of patients with frozen shoulder, compared with 12% in age- and sex-matched controls with other rheumatologic conditions.[64] Frozen shoulder has also been reported to occur with increased incidence among patients with thyroid disorders,[13,35] as well as those with hypoadrenalism[122] or corticotropin deficiency.[24]

Trivial trauma has been postulated to be an important factor, particularly when it is followed by a prolonged period of immobilization.[29,100] This does seem to be the sequence of events in some patients who develop frozen shoulder. The association of frozen shoulder with major trauma to the shoulder or other parts of the upper extremity is recognized. The association with minor trauma that may be forgotten is difficult to document and may be overlooked.[26,77] Most patients who sustain minimal trauma, even when combined with a period of immobilization, do not develop frozen shoulder. This has led some investigators to conclude that there are some patients who possess a "constitutional" predisposition to develop a frozen shoulder. Support for this theory is provided by the significant incidence of bilateral frozen shoulders.[19,25,92,103,125]

The role of psychologic factors has been considered in the development of frozen shoulder. Some investigators have suggested that a certain personality structure, coupled with untoward life events and inappropriate responses to stress, may serve as a predisposing or precipitating factor for its development.[27,35,68,107] Coventry chose the term "periarthritic personality" to describe one component of a three-part theory on the pathogenesis of frozen shoulder in a group of patients with painful stiff shoulders. He observed that most patients had "a peculiar emotional constitution in which they were unable to tolerate pain, expected others to get them well, and refused to take any personal initiative in their recovery."[27] Other studies, however, have found no evidence for a characteristic personality disorder.[92,125] It would appear, therefore, that a specific periarthritic personality type is difficult to identify. The role of psychologic factors should be considered, at best, a secondary factor in the management of these patients.

Fibromatosis has also been implicated in the causation of frozen shoulder syndrome. The pathomechanics are believed to be found in fibrous tissue contracture formed in response to cytokine, lymphocyte, or monocyte products. Platelet-derived growth factor is a potent mytogenic polypeptide for mesenchymal cells. Immunocytochemistry was performed with monoclonal antibodies on the rotator ligaments excised from 12 patients with resistant frozen

shoulder. Bunker and Anthony report that the pathologic process is active fibroblastic proliferation accompanied by some transformation to a smooth-muscle phenotype (myofibroblasts). The fibroblasts lay down collagen that appears as a thick nodular band or fleshy mass. These appearances are reportedly very similar to those seen in Dupuytren's disease of the hand, with no inflammation and no synovial involvement. The contracture acts as a checkrein against external rotation, causing a loss of both active and passive movement.[22]

Frozen shoulder associated with a known underlying disorder is considered to be secondary, and this group includes intrinsic, extrinsic, or systemic disorders. Intrinsic shoulder abnormalities include rotator cuff tendinitis, rotator cuff tears, tendinitis of the long head of the biceps tendon, calcific tendinitis, and acromioclavicular arthritis. Extrinsic disorders, which represent pathologic conditions remote from the shoulder region, include ischemic heart disease and myocardial infarction,[79,125] pulmonary disorders including tuberculosis,[55] chronic bronchitis, emphysema,[111] tumor,[28] cervical disc disease and radiculopathy,[2,56,125] cerebral vascular hemorrhage,[14,16] previous coronary artery bypass graft surgery,[113] previous breast surgery, lesions of the middle humerus,[114] and central nervous system disorders, such as Parkinson's disease.[106] Systemic disorders represent generalized medical conditions that are known to occur in association with frozen shoulder. Such conditions and poor prognostic indicators include diabetes mellitus, hypothyroidism, hyperthyroidism, and hypoadrenalism.

Epidemiologically, the exact prevalence and incidence of frozen shoulder are not known, but the cumulative risk of at least one episode of frozen shoulder has been estimated to be a minimum of 2%.[25] It is most frequently found in patients between the fourth and sixth decades of life, and it is more common in women than men.[2] The nondominant extremity appears to be more commonly involved, with most reported cases being described as affecting the left side.[25,29,65] Bilateral involvement occurs in 6% to 50% of cases, although only 14% of these bilateral cases manifest simultaneously.[2,6,21,25,107] When a history of bilateral involvement is identified, the possibility of a constitutional predisposition should be explored.[3,25,107] The same shoulder is rarely involved again with adhesive capsulitis.[6,7]

There is significant controversy over the natural history of frozen shoulder relative to both objective and subjective outcomes. Historically, frozen shoulder has been touted as a condition for which "recovery is always sure and may be confidently expected."[26] Several investigators using a variety of treatment methods have reported that a high percentage of affected patients achieve full range of motion.[26,39,65] In addition, they have found complete or near complete symptomatic relief.[39,47] More recent investigators have questioned the early optimistic reports, finding measurable restriction at follow-up in 39% to 76% of patients[18,25,79,80,103] and persistent symptoms in up to 45%.[6,100]

The time course of adhesive capsulitis has been described as classically lasting 18 to 24 months.[6] Recent studies have challenged this commonly held belief. Reeves noted that the mean duration of symptoms was 30 months.[103] Patients describing themselves as functionally recovered tend to underestimate their loss of motion.[19] Reeves described some restriction in shoulder motion in more than 50% of patients in a 5- to 10-year follow-up, but functional impairment was identified in only 7%.[103] Clark et al. found that 42% of patients had persisting limitations of motion after 6 years of follow-up.[25] Binder et al., in a prospective study, noted that 90% of patients did not regain the minimum range of motion when matched for age and sex with a controlled group 6 months after diagnosis.[6] They also reported that 40% of patients failed to regain a minimum range of motion when matched for age and sex with a controlled group when followed for a minimum of 3 years. In a retrospective study, performed by Schaffer et al., of a carefully selected group of patients with frozen shoulder, almost half remained symptomatic many years after the onset of symptoms, and up to 56% had residual restriction in one or more planes.[111]

Despite the subjective and objective outcome of this disorder, there seems to be widespread agreement on the seeming lack of significant or frequent functional disability documented at cessation of treatment. Regardless of objective restriction or the presence of symptoms, few patients are reportedly functionally restricted to any significant degree.[6,25,66,103] The lack of correlation between subjective and objective findings has been noted consistently.[6,8,18,25,47,80] Symptomatic patients frequently have no measurable restricted range of motion in any plane. Conversely, those patients with the most significant motion restriction were often pain-free. Whether this is due to adaptation to such restriction or whether restriction in motion is unimportant for daily living activities is an unresolved issue. According to Neer, however, the presence of such restriction depends on the functional demands of the patient. Even in the active patient, the presence of 150 degrees of active elevation, 50 degrees of external rotation, and internal rotation to the eighth thoracic vertebra is probably sufficient for normal function.[82] In Schaffer et al.'s report of an older population whose functional demands were surely less than the aforementioned, the degree of restriction tolerated in any plane was certainly even greater. They stated that the preeminent importance of forward flexion and elevation in daily activities superseded the findings of restriction predominantly in the abducted and externally rotated positions, which resulted in little functional impairment.[111]

In general, the natural history of frozen shoulder is uncertain, and additional randomized, prospective studies are needed. Difficulty exists in performing these studies owing to the ethical dilemma of assigning patients to an untreated group.

CLINICAL EVALUATION

Frozen shoulder represents a disease process that is diagnosed with careful history and physical examination rather than with diagnostic tests. Patients often report a gradual onset of pain that often was initiated by a minor event. Common initiating events include reaching to put on a coat, reaching into the backseat of the car, or a minor tug on the arm. Pain can be anywhere from minor to severe in intensity and often exists for many months prior to presentation. Pain is usually present over the deltoid muscle with occasional pain that radiates into the forearm. Many patients experience a fullness or swelling in the upper arm with an occasional subjective tingling sensation. Pain that wakes them at night is often their primary complaint. Patients with this condition often report a gradual loss of shoulder function that is exacerbated with use. Overhead and behind the back activities become increasingly difficult to perform as motion is diminished with progressive scarring. The symptoms of frozen shoulder closely resemble those found in rotator cuff pathology. Careful physical examination needs to be performed to identify a frozen shoulder, especially in those patients who present with a magnetic resonance imaging (MRI) diagnosis of partial-thickness rotator cuff tear.

The physician also needs to take a careful history of related risk factors, especially diabetes mellitus, thyroid disease, cardiovascular disease, or cervical disease. The rate of frozen shoulder among diabetic patients is much higher than in the normal population, with bilateral presentation in as many as 77% of cases.[96] Patients who have taken insulin for more than 10 years have a significantly higher incidence of ongoing disability than other patients, despite treatment of the diabetes.

Often patients have undergone treatment for rotator cuff tendonitis with cortisone shots in the subacromial space and physical therapy with rotator cuff strengthening.

HISTORY

An integral component to the evaluation of a patient with a frozen shoulder is a careful clinical history. Patients typically report a vague discomfort in the upper arm and deltoid region that may be present for a protracted period of time before presentation. Often the symptoms start in an insidious fashion with minimal or no trauma. Other patients report a specific incident that they report as the inciting event. Common presenting complaints include pain reaching to put on a coat, reaching in the back seat of the car, or reaching out the car window at a drive-through. These events most likely represent the first recognition of the condition rather than the inciting event. Pain also may radiate down the arm into the forearm with a number of patients complaining of a vague tingling sensation.

Pain is a critical component to the frozen shoulder condition with the pain at night, pain with dressing and daily activities, and pain exacerbated with use. Often the intensity of the pain varies during the course of the disease. Early in the inflammatory phase, patients often complain about an intense burning pain compared to a dull fullness during the contracted stage. Functional deficits often become prominent as loss of motion occurs. Gradual loss of motion occurs as the condition progresses into this contracted phase. Activity overhead or behind the back becomes especially difficult to perform. Early in the course of the disease, the symptoms closely resemble those found in rotator cuff pathology. As the shoulder stiffens, there is a progressive loss of glenohumeral motion. The most significant loss of motion is usually with external rotation with a smaller loss of abduction and internal rotation. Sharp pain at the endpoint of restricted shoulder motion is the hallmark of frozen shoulder. Most patients complain about reaching, putting on a coat, or fastening a bra. Pain about the scapula is probably secondary to increased scapulothoracic motion attempted to compensate for the decreased glenohumeral motion.

The physician also needs to inquire about risk factors such as diabetes, thyroid disease, or cervical disease. Among diabetic patients, the risk of frozen shoulder is much higher than in the normal population, with bilateral presentation in as many as 77% of cases.[110] Frozen shoulder has been reported to affect 20% of patients afflicted with diabetes versus an incidence of approximately 5% in the general population. Diabetic patients have also been found to have poorer outcomes with treatment.[40]

Certain medications also have been associated with frozen shoulder. Protease inhibitors used to treat HIV have been reported to cause frozen shoulder. Barbiturates and antituberculosis agents have also been associated with frozen shoulder.[37]

PHYSICAL EXAMINATION

Physical examination is critical in the diagnosis of frozen shoulder, especially early in the disease when it is commonly confused with rotator cuff pathology. The physical examination should begin with a careful clinical inspection. The patient needs to be in a gown that allows exposure of both shoulders both in the front and the back. Swelling or erythema about the shoulder could represent infection as a cause of a painful, stiff shoulder. Inspection for atrophy, especially about the deltoid, could represent an axillary neuropathy or cervical radiculopathy as a cause of pain and loss of active motion. Scapular winging should always be noted, although compensatory winging can sometimes be difficult to differentiate from long thoracic nerve palsy. Masses about the shoulder could indicate a musculoskeletal tumor as the cause of a stiff shoulder.

Finally, careful palpation of the bones about the shoulder may localize an area of pain or crepitation from a fracture about the shoulder girdle.

A loss of active or passive range of motion of the glenohumeral joint is the clinical hallmark of frozen shoulder (Fig. 17-2). Careful assessment and recording of range of motion in all planes during each office visit are important to follow the progress achieved by the treatment employed. The examiner must be careful to identify and control compensatory motions to measure only pure glenohumeral motion. Patients with glenohumeral stiffness often exhibit relatively good motion secondary to increased scapulothoracic motion or trunk lean. Also, the limits of motion caused by firm endpoints need to be measured rather than muscular guarding caused by pain. Pain is usually absent within its free range and only present at the extremes of motion.

Active shoulder elevation is measured in the plane of the scapula, with the patient seated, and is referenced to the patient's thorax, not to a line vertical to the floor. Internal rotation is measured behind the back along the lumbar or thoracic vertebrae. Active external rotation is measured with the arm at the side and in 90 degrees of abduction. Passive motion should be evaluated with the scapula fixed by placing the patient supine on the examination table. This restricts excessive scapulothoracic movement and trunk tilt, thereby providing a more accurate assessment of glenohumeral motion. Passive forward elevation, external rotation with the arm at the side, and internal and external rotation at 90 degrees of abduction are measured. A firm endpoint is often appreciated with pain at the extremes of motion. There is often no single capsular pattern seen in patients with frozen shoulder.[109]

A complete shoulder examination should include palpation about the acromioclavicular joint, sternoclavicular joint, and anterolateral acromion over the rotator cuff insertion. Identifying concomitant rotator cuff pathology can be a difficult diagnostic dilemma. Impingement sign, Hawkins' sign, and Jobe's test are often unreliable secondary to the capsular irritation caused by the synovitis associated with a frozen shoulder. The abdominal compression test and lift-off test to identify subscapularis pathology are often limited by the loss of active internal rotation. Any shoulder examination would not be complete without an evaluation of the cervical spine to identify loss of motion,

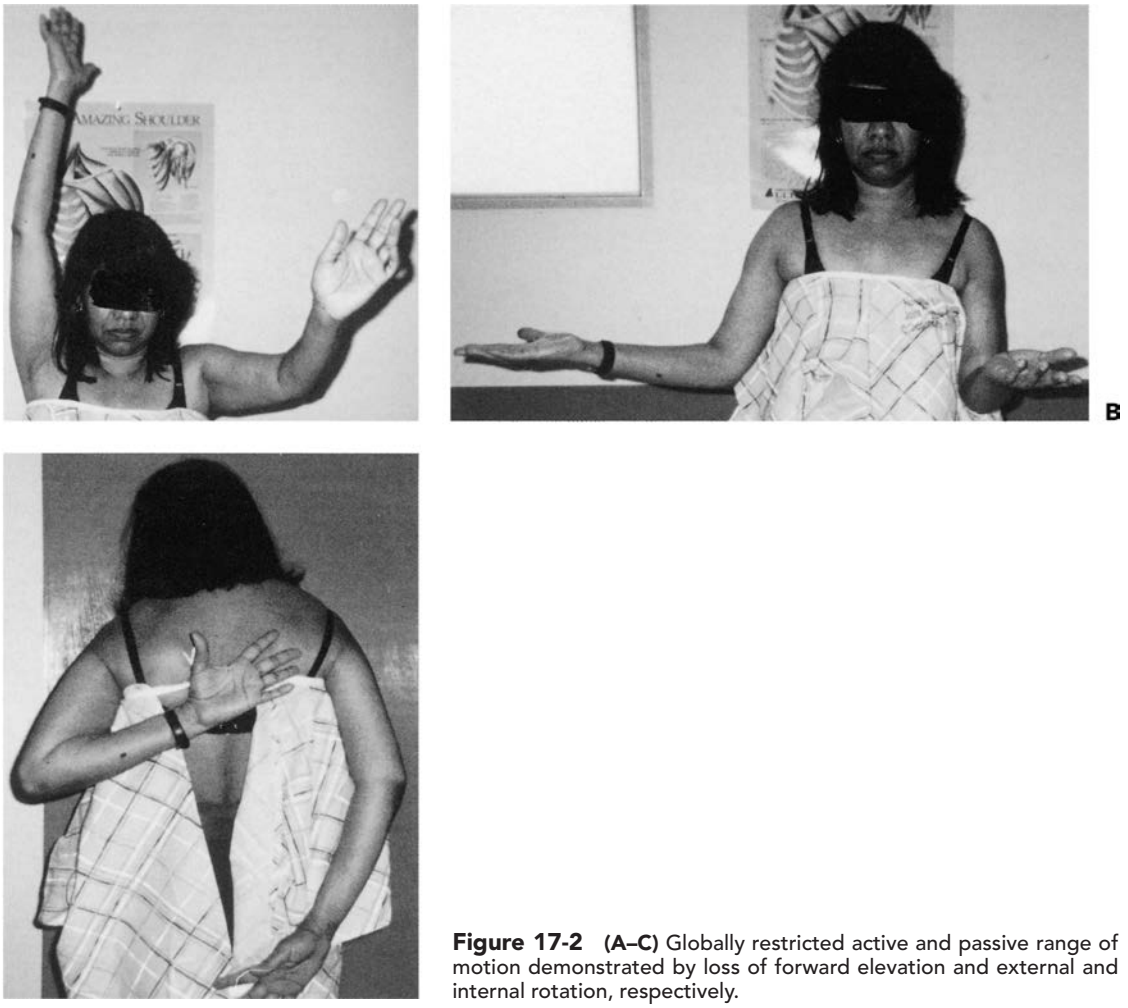


Figure 17-2 **(A–C)** Globally restricted active and passive range of motion demonstrated by loss of forward elevation and external and internal rotation, respectively.

pain, or radicular symptoms that may identify concomitant cervical disease. A neurologic examination may help to identify a brachial plexus palsy or reflex sympathetic dystrophy.

Recognition of the different patterns of motion loss is important in determining the cause as well as planning treatment strategy. Motion loss often correlates with the location of the capsular contracture. Limitation of external rotation with the arm abducted is usually associated with scarring of the anteroinferior region of the capsule. Limitation of external rotation with the arm in adduction is associated with contracture of anterior and superior capsule from the rotator interval to the middle glenohumeral ligament. Loss of the subscapularis recess is associated with this contracture. Limitation of internal rotation in adduction and abduction is associated with scarring in the posterior capsule. Extraarticular contracture in the subdeltoid space can cause global motion loss in cases of postsurgical contracture.

A helpful adjuvant to the history and physical examination is the lidocaine injection test. If loss of motion is felt to be secondary to pain from a rotator cuff tear, an injection of 10 mL of 1% lidocaine into the subacromial space can help eliminate pain-inhibited motion. Loss of motion secondary to true capsular contracture will not be improved with a lidocaine injection.

IMAGING STUDIES

A complete set of radiographs should be obtained in all cases of frozen shoulder. Anteroposterior films in internal and external rotation and supraspinatus outlet and axillary views should be obtained to exclude other disorders causing loss of motion. The axillary view will help exclude a locked dislocation or fracture about the glenohumeral articulation. A locked posterior dislocation can present as a loss of external rotation without an adequate axillary radiograph. Radiographs are also important to rule out destructive changes within the bone as seen with primary or metastatic bone tumors or infectious processes. A recent report described five case reports of malignant shoulder girdle tumors misdiagnosed as a frozen shoulder. The cases cited included Ewing's sarcoma in a 60-year-old, malignant fibrous histiocytoma in a 42-year-old, chondrosarcoma of the glenoid in a 50-year-old, and metastatic squamous cell to the supraspinatus muscle belly.[99] Most tumors about the shoulder have a soft tissue mass or characteristic radiographic changes; however, approximately 10% have normal radiographs. Primary chest wall tumors can also present as a painful stiff shoulder. In a high-risk patient, the lung should be considered.[28]

An evaluation of the glenohumeral articulation looking for inferior osteophytes or joint space narrowing will help exclude degenerative arthritis as a cause of decreased range of motion. Primary frozen shoulder usually has unremarkable radiographs, although disuse osteopenia is possible.

Technetium bone scanning often exhibits increased uptake about the proximal humerus. Findings of increased uptake, although nonspecific, are probably secondary to hypervascularity. Wright and Haq noted a favorable association between an increased pertechnetate uptake about the shoulder and rapid response to corticosteroid injection.[125] Binder and associates observed that over 90% of their patients had an increased uptake on diphosphonate scans, with 29% having more than a 50% increase in uptake compared with the opposite shoulder. These authors, however, could not find any association between the bone scan finding and the duration of symptoms, the initial severity of the disease, the arthroscopic findings, or the eventual recovery.[8]

Shoulder arthrography for the diagnosis of frozen shoulder was described by Neviaser, which gave a better understanding of the underlying joint pathology. He described the combination of decreased joint volume, an irregular joint outline, and variable filling in the bicipital tendon sheath.[89] The reduction in the shoulder joint capsule capacity to less than 10 to 12 mL and the variable lack of filling of the axillary fold and subscapular bursa are the current accepted characteristic findings (Fig. 17-3).[1,48,69,87,89] Binder and coworkers noted that, although arthrography is useful in the diagnosis of frozen shoulder, arthrographic findings do not indicate the type of onset (i.e., primary or secondary) or the rate or extent of recovery.[8] Other authors confirm that such findings also have not had any productive value in terms of disease severity or prognosis.[53,69]

Arthrography has also been used to document tearing of the joint capsule during manipulation under anesthesia. Lundberg found that tears of the capsule allowed dye to escape into the extracapsular space but found no tears of the rotator cuff following manipulation under anesthesia.[71]

MRI reports are often read as normal; however, radiologists continue to explore their ability to diagnose a frozen

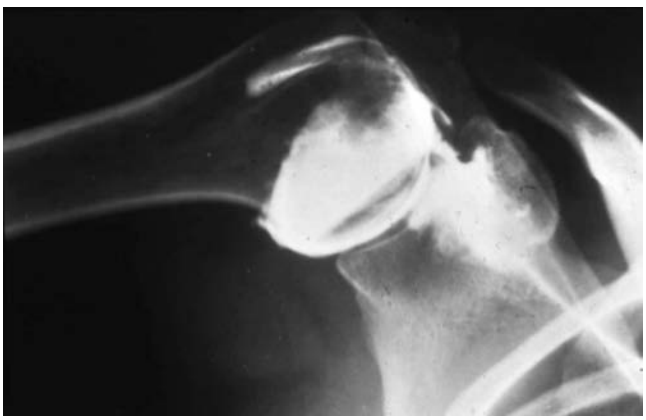


Figure 17-3 Arthrogram in a patient with a frozen shoulder showing absence of the inferior capsular pouch and marked decrease in the capsular volume.

shoulder with this modality. Significant thickening of the coracohumeral ligament and rotator interval can be seen on MRI, which is correlated with what is seen at arthroscopy. The inferior capsule has not been found to be thickened despite the decreased volume of the recess. Complete obliteration of the subcoracoid triangle sign between the coracohumeral ligament and coracoid process was found to be 100% specific for frozen shoulder but only 32% sensitive.[79] Others have recommended using MR arthrography to help in identifying frozen shoulder. Lee et al. found significant increase in the thickness of the inferior capsule and a decreased filling ratio of the fluid-distended axillary recess when comparing controls to patients with arthroscopically proven adhesive capsulitis.[62] The practical use may be limited by the fact that the procedure depends on distending the joint fully without rupturing the capsule. MRI is currently not useful to diagnose frozen shoulder but is useful in identifying other causes of frozen shoulder, such as infection or tumor, which may mimic a stiff shoulder. It is also useful to identify concomitant pathology such as rotator cuff tears.

LABORATORY STUDIES

Routine laboratory studies are usually normal in a patient with frozen shoulder. The erythrocyte sedimentation rate may be elevated in as many as 20% of patients, which can make this test difficult to use as a way to differentiate from a shoulder infection. A complete blood cell count can be helpful in these situations, although it may be normal early in the infection or in an immunocompromised patient. A chemistry profile can be useful to identify diabetes mellitus, which is a common cause of frozen shoulder. Rheumatoid factor and antinuclear antibody may be helpful to identify an underlying inflammatory disease.

There remains ongoing controversy about the significance of the reported increased levels of immune complexes in patients with frozen shoulder.[19,20,57] Bulgen and associates reported 40 patients with clinical signs of frozen shoulder who had shown an increase in immune complex levels including C-reactive protein and impaired cell-mediated immunity.[20]

TREATMENT

Options

The overall goal in the treatment of patients with frozen shoulder is to relieve pain and restore motion and function. Ideally, efforts should be directed toward prevention of this syndrome by identifying the patient at risk and initiating early intervention. Early in the course of the disease, patients may only present with vague pain characterized by pain with terminal stretch. The most important point to consider is to avoid the misdiagnosis of other shoulder disorders that may require surgery. Surgery addressing other pathology may dramatically worsen the pain and stiffness of frozen shoulder.

Most patients present with many months of pain that has progressed to significant loss of motion. The clinician must design a treatment plan that is individualized and based on the severity and chronicity of the patient's symptoms, as well as previous therapeutic efforts. Treatment may have to be modified based on the patient's clinical response and perceived disability. Some patients tolerate a protracted conservative treatment plan with range-of-motion exercises, while others necessitate a more aggressive approach.

As in other medical conditions for which the pathophysiology is poorly understood, many different forms of treatment are used. The first objective in the treatment of any patient with frozen shoulder is to relieve pain so as to allow the patient to perform the appropriate exercise program to improve motion and function. Pain-relieving methods include nonsteroidal antiinflammatory medications, corticosteroid injections, or transcutaneous electrical nerve stimulation (TENS). The routine use of narcotics should be avoided because of the protracted course of the disease and risk for dependency. Nonsteroidal antiinflammatory medications can be effective, but no controlled studies to document their efficacy have been performed.[31,52,104] Oral corticosteroids have been recommended, but their exists little evidence to support their routine use.[7,10,11,33,85] One randomized, double blind placebo-controlled trial of 50 patients treated for 3 weeks with 30 mg of oral prednisolone demonstrated a short-term benefit for 3 weeks in pain relief and range of motion, which was equal to placebo at 12 weeks.[17]

Intraarticular injections are a helpful adjuvant to relieve pain in the inflammatory or painful phase of frozen shoulder. This may allow patients to participate in their rehabilitation protocol. Hollingworth reported 50% improvement in range of motion in 26% of cases.[49a] The ability of the physician to adequately inject the joint may limit the effectiveness of the injections.[105] In one study, a technically satisfactory arthrogram could not be achieved in 12% of patients.[121] The use of intraarticular injections has theoretical benefits to decrease the synovitis associated with a frozen shoulder, but, in general, the evidence is equivocal. Patients with significant pain and capsular irritation with terminal stretch may benefit from an intraarticular injection. However, steroid injections have not been shown to improve the rate of return of shoulder motion.[71,101]

Nonoperative Treatment

Patients with frozen shoulder should be placed on an exercise program with the aim of maintaining and regaining range of motion (Fig. 17-4). Indications for treatment of

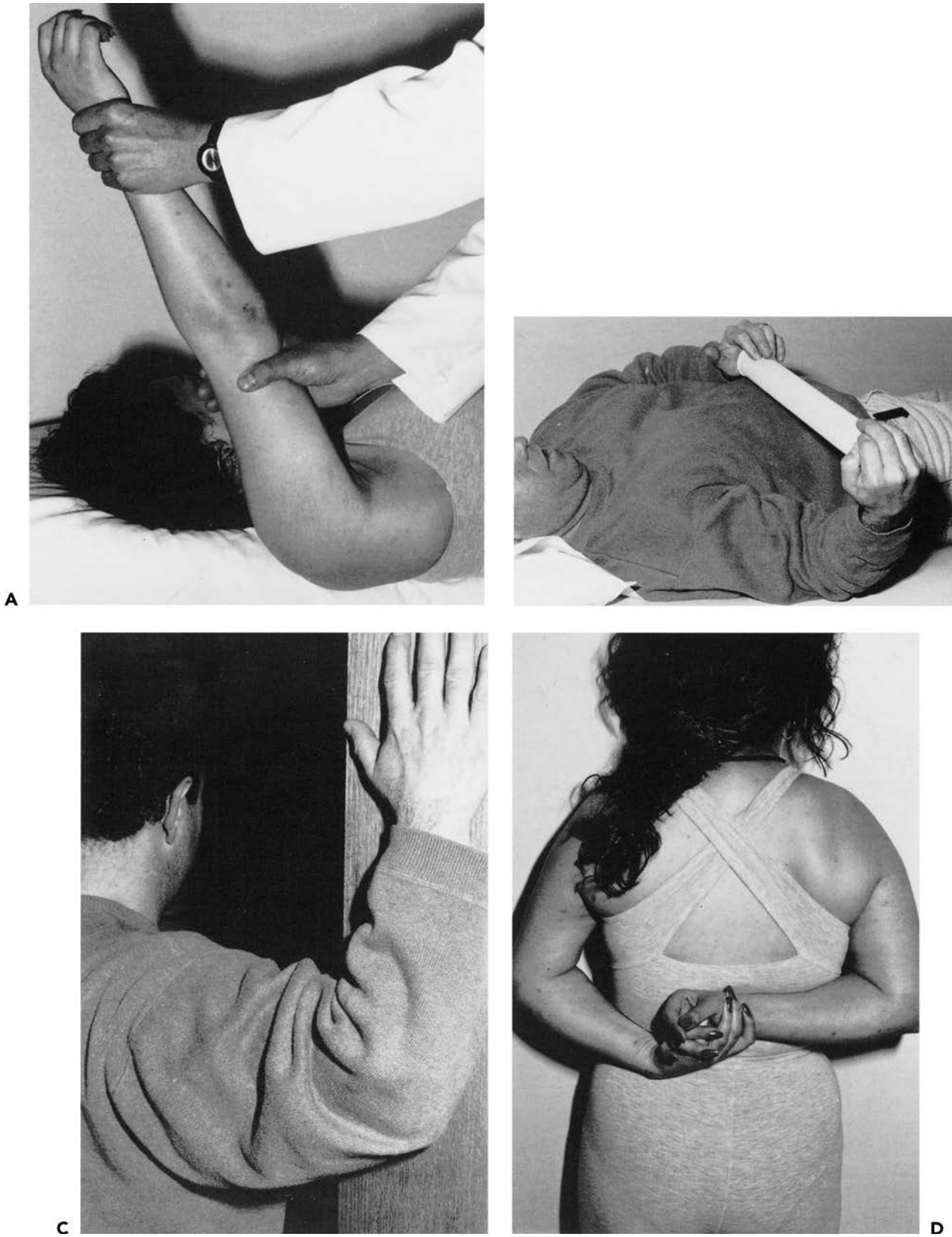


Figure 17-4 **(A–D)** Passive stretching exercises performed for overhead elevation, external rotation at side, 90 degrees of abduction, and internal rotation behind back.

the frozen shoulder in general are based on the chronicity and cause of the stiffness. Nonoperative treatment is indicated for those primary or secondary frozen shoulders with stiffness less for than 6 months and or no previous treatment. Each patient should begin an active assisted range-of-motion exercise program complying with gentle, passive, stretching exercises. These exercises should be performed four to five times per day, including forward elevation, internal and external rotation, and cross-body adduction. They can be performed standing or sitting, but are most readily performed in the supine position. It is most important to perform four or five short sessions per day, lasting 5 to 10 minutes, rather than one long session, because the shoulder will become stiff again in between each session. It is important to perform these exercises gently, but it needs to be stressed that at each session the arm should be pushed slightly past the point of pain; otherwise, no progress in the range of motion would be expected. Forward elevation may be assisted by using the opposite arm or a pulley to pull the arm up over the head. External rotation is aided with a cane while the patient is lying supine and holding the arm at the side, resting the elbow on a pillow. Internal rotation is performed by pulling the arm up behind the back with the assistance of the opposite arm or a towel. Using the opposite extremity, the affected limb is stretched to its limit and slightly past it, held in place for a count of 5 to 10 seconds, and then brought to a resting position. Periods of rest in between each session are necessary to relieve muscle tension and pain.

To encourage continued exercises, daily bar charts are helpful to document progress because small improvements in range of motion might otherwise be unnoticed by the patient, especially those who are easily discouraged. Local modalities consisting of heat at the initiation of the exercise session and ice at its conclusion may be helpful to increase flexibility and reduce inflammation, respectively. These modalities are not curative, but can aid in decreasing discomfort, which will allow greater ease in performing the exercises.

Forward elevation of the shoulder is performed with the extremity in the plane of the scapula as it is grasped either at the wrist or behind the elbow and pushed upward gradually. This is best performed supine to keep compensatory factors such as trunk tilt to a minimum. A pulley can also be used to accomplish this motion, which is best performed seated with the back of the patient to the pulley. Similarly, cross-body adduction is performed as the affected extremity is pulled across the chest toward the contralateral shoulder. This maneuver assists in stretching the posterior portion of the capsule, which is of utmost importance in obtaining internal rotation. External rotation is performed supine with the elbow close to the body. A stick is held in the hand with the elbow flexed to 90 degrees and is used to rotate the affected extremity away from the body. Internal rotation is performed by pulling the wrist of the affected extremity into extension first behind the back, then bringing the hands up between the shoulder blades. It may also be assisted with a towel or by grasping a door handle behind the back and performing a deep knee bend. In the early stages of the exercise program, one should start with simple stretching, such as Codman's pendulum exercise, to gently loosen and relax the shoulder. The patient bends at the waist while balancing with the good arm on a firm surface. This allows the affected arm to swing with gravity in a circular motion with the hand turned inward and outward. Overly forceful stretching exercises are contraindicated in the early phases of the frozen shoulder syndrome and may exacerbate symptoms. Constant reassurance from the physician is necessary to promote continued compliance.

The physical therapist plays a major role as a teacher, explaining to the patient that it will take time to resolve symptoms and that pain will decrease as motion improves. Whenever possible, the therapy program should be performed under the supervision or in addition to a physical therapist on a weekly basis. The patient should be instructed that the success or failure of the therapy largely depends on his or her compliance in performing the exercises as directed, not only with the therapist but four or five times daily on his or her own. In a loosely supervised physical therapy program, most patients will improve with time, although it may take months. Thus, in most cases, more invasive treatment is generally not required. Griggs et al. found 90% satisfaction with nonoperative treatment, with only 7% requiring manipulation or capsular release. However, despite significant improvements in patient satisfaction, pain, and range of motion, there was still a significant difference when compared to the unaffected shoulder.[40]

Capsular Distention or Brisement

The method of capsular distention, referred to as distention arthrography or brisement, has been advocated as a means of expanding the contracted capsule.[1,34,36,51,69,94] This procedure involves injecting fluid into the glenohumeral joint in sufficient enough volume to generate pressures high enough to cause capsular disruption that is evidenced by a significant decrease in the pressure necessary to continue injection. This procedure has been performed in a variety of ways: with injection of contrast as part of an arthrogram (Fig. 17-5)[36,69,94,103]; with injection of saline and local anesthetic, such as hydraulic distention[34,42]; and arthroscopically.[51] As with all other methods of treatment for frozen shoulder syndrome, the reported results have been variable and difficult to interpret because the procedure often is combined with other procedures such as manipulation or corticosteroid injections, and the experience has been limited. The addition of these variables makes it difficult, if not impossible, to compare reports. Some have found that capsular distention was not

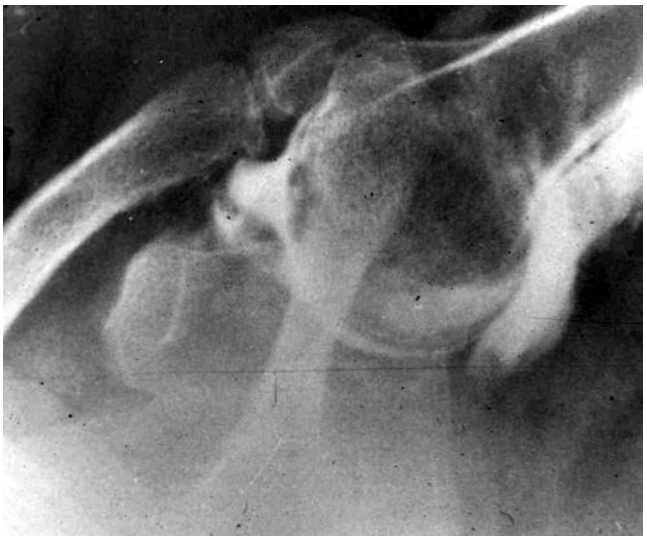


Figure 17-5 Distention arthrography with disruption of the inferior pouch and leakage of contrast from the capsule.

as effective in the face of advanced disease with significant stiffness.[1,117] Vad et al. recommended performing capsular distention for those patients who had failed physical therapy and were in Hannafin stage II disease, with early loss of motion before the capsule becomes thickened.[117]

Rizk and Pinals examined 16 patients with adhesive capsulitis of the shoulder treated in an open trial of capsular distention with intraarticular injection of 30 mL of fluid containing 8 mL of 1% lidocaine, 2 mL of corticosteroid, and 20 mL of radiocontrast material. A capsular tear during arthrography occurred in all cases, usually at the subscapular bursa or subacromial bursa, as documented by extravasations in either of these areas. Rupture at the distal bicipital sheath occurred in two patients and was not associated with pain relief. Thirteen of 16 patients experienced immediate pain relief and increased shoulder mobility. This improvement was maintained over a follow-up interval of 6 months. Given these results, the authors felt that disruption of the contracted capsule by hydraulic distention seemed to be the mechanism for achieving symptomatic relief in adhesive capsulitis.[108]

Manipulation under Anesthesia

Manipulation under anesthesia has been the mainstay of operative treatment of frozen shoulder.[47,48,49] It results in a rapid return of shoulder motion, although some authors disagree about whether it shortens the disease course.[71,80] The timing of manipulation remains controversial, but lack of improvement in motion after 3 to 6 months of physical therapy is often cited. It is important to emphasize that operative treatment of primary adhesive capsulitis should not be considered while the patient is experiencing the severe pain seen in the inflammatory phase of frozen shoulder. Despite the initial improvement, patients may experience quick deterioration of motion secondary to capsular injury during the painful inflammatory phase.[88] It is important to wait until pain is present only at the extremes of range of motion.

Manipulation can be performed either under general anesthesia, regional anesthesia, or both. The addition of regional anesthesia (i.e., scalene block) allows the patient to awaken from the procedure without pain and to observe the recovered motion. The block may be administered as a single long-acting block or by placement of an indwelling interscalene catheter.[97] If a single injection is used, a long-acting agent such as bupivacaine should be used, which will provide approximately 12 hours of anesthesia. The interscalene block has the added risk of complications associated with the block, which can include pneumothorax, peripheral paresthesias, brachial plexus palsies or neurapraxias, and hoarseness. There is also the risk of the block being ineffective for intraoperative or postoperative anesthesia. Therefore, intraarticular pain catheters using bupivacaine are used by some with reported excellent pain relief without motor blockade.[126]

Manipulation should be performed in a gentle, controlled fashion with the patient in a supine position to control for scapular mobility. A gentle forward flexion maneuver is performed by grasping the humerus close to the axilla to avoid injury by decreasing the lever arm of the humerus. This maneuver usually results in rupture of the inferior portion of the joint capsule[87,89] (Fig. 17-6). External rotation is increased by applying firm external rotation force with the arm close to the body and in abduction. Great care must be employed because of the risk of torquing the humerus and causing a spiral fracture. If

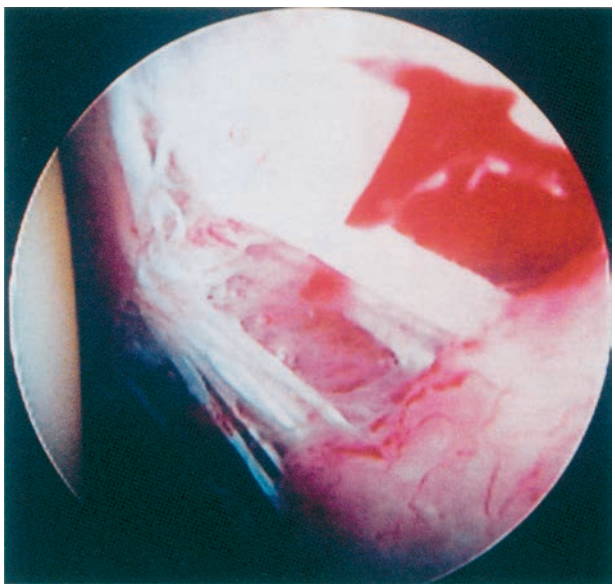


Figure 17-6 Tom labrum and capsule in inferior axillary pouch postmanipulation.

release does not occur, an alternative method to obtain external rotation should be employed. Internal rotation is increased by stretching the arm in internal rotation with the arm in abduction.

Attention must be placed on gentle manipulation. If a firm endpoint is felt, excessive pressure should not be applied because of the risk of fracture. If manipulation under anesthesia fails to restore symmetrical motion equal to the opposite side, converting to an arthroscopic release should be considered.

Complications of manipulation are best avoided by gentle, controlled pressure. Humeral fractures, glenohumeral dislocation, rotator cuff tears, and radial nerve injuries have been reported. Manipulation should be avoided in the elderly, osteoporotic patient with high risk of fracture as well as those with reflex sympathetic dystrophy. Manipulation is also ill advised in the patient with stiffness secondary to previous surgery or fracture with risk of disruption of the soft tissue repair. Loew et al. evaluated 30 patients with an arthroscope after a manipulation under anesthesia to evaluate for intraarticular lesions. They identified four superior labral anterior and posterior (SLAP) tears, three partial-thickness subscapularis tears, three anterior labral detachments, and one small osteochondral defect in the anterior inferior glenoid.[67]

Postmanipulation rehabilitation is of paramount importance to maintain the motion gained. In the outpatient setting, physical therapy is begun before discharge or the following day. Encouragement of removing the sling after the block has resolved and performing passive range of motion and active assisted range of motion with a home pulley is recommended three to four times per day. Continuous passive motion machines have also been used after manipulation, but there has been no long-term, controlled studies to verify their efficacy.

The use of corticosteroids with manipulation under anesthesia has also been debated. Many feel injection after manipulation allows less pain after surgery with ease of performing range-of-motion exercises. However, efficiency of corticosteroids is debated. A randomized study comparing manipulation with and without corticosteroid injection showed no significant difference.[60] A prospective clinical study was performed by Bulgen and coworkers examining the evaluation of three treatment regimens for frozen shoulder, including intraarticular steroids, mobilization, and ice therapy as compared with no treatment at all. In this prospective study of 42 patients followed for 8 months, the authors stated that there was little long-term advantage in any of the treatment regimens, but that the steroid injections may have been of benefit for pain and range of movement in the early stages of the condition.[18]

Manipulation of the shoulder has been advocated as a safe and effective means of enabling these patients to return to functional motion about the shoulder area.[30] Haggart and coworkers looked at 97 patients treated with manipulation of the shoulder.[41] They found that 95% had excellent or good results with 4 to 9 years of follow-up. Hamdan and Al-Essa evaluated 100 patients with frozen shoulder treated with manipulation alone or with methylprednisolone or a large volume of saline followed by physiotherapy. Their findings showed improved results with manipulation under anesthesia combined with a large volume of saline.[43] Harmon reported 400 manipulated shoulders on three separate follow-up studies.[45] He found that there was full painless motion in 64% to 94% of the patients. In a study by Hill and Bogumill, 9 of 12 (75%) of the patients with frozen shoulder syndrome had painless full range of motion of the shoulder at the time of follow-up postmanipulation, whereas 3 of 12 (25%) continued to complain of minimal aching about the shoulder joint after vigorous activity, but did not feel that this was impairing their ability to function.[49] All of the patients felt they were able to perform satisfactorily at their job, during activities of daily living, and during recreational activities. These authors stated that manipulation was found to be a safe means of treating adhesive capsulitis and significantly shortened the course of the disease. A higher incidence of failure was found among diabetic patients with frozen shoulder.[43]

Arthroscopic Capsular Release

If manipulation under anesthesia fails to restore symmetrical motion or if manipulation is felt to be deleterious, an arthroscopic capsular release should be considered. A controlled release of contracted tissue without the risk of fracture or injury to normal structures is a significant advantage. Arthroscopy also provides diagnostic information on concomitant disorders such as labral tears, chondromalacia, biceps pathology, rotator cuff tears, large anterolateral acromial spurs, or calcium deposits. Arthroscopic release is also beneficial for postsurgical contracture because it allows precise, selective capsular release of intraarticular contracture as well as subacromial and subdeltoid scar tissue. It also allows more immediate, aggressive range of motion than after open release.

The operative technique involves three basic components: **anesthesia;** preferably interscalene block with or without general anesthesia; and **manipulation** of the shoulder performed either before, during, or after the **arthroscopic release**. The procedure can be performed in the beach-chair position, which allows exposure to both anterior and posterior portals and allows for the free manipulation of the arm. A lateral decubitus position is preferred by some surgeons. In a cadaver study, the axillary nerve was found to be the furthest from the glenoid with the arm in abduction and external rotation. The nerve is at greatest risk the closer one gets to the humeral head at the 5 to 7 o'clock position.[54] A careful assessment of range of motion in all planes before and after treatment is documented. A blunt trocar is used to enter the joint through a

standard posterior viewing portal and carefully sliding over the humeral head owing to joint contracture. Care should be taken to avoid cartilage injury or inadvertent entrance into the humeral head in the contracted shoulder. To increase joint volume, a gentle manipulation can be performed prior to insertion of the trocar. A blood-filled joint is encountered after manipulation and needs to be lavaged prior to inspection of the joint. An anterior portal is made with a spinal needle localization between the biceps and subscapularis tendons. An outflow cannula is useful to allow fluid flow and to prevent fluid extravasation onto the operating room floor. A systematic inspection should be undertaken to identify synovitis and contracted tissue as well as to identify ruptured structures after manipulation. A complete evaluation of the joint may not be possible until after release owing to the contracted volume of the joint. The biceps tendon and superior border of the subscapularis tendon should be identified first. A hooked electrode is introduced through the anterior portal and release of the rotator interval is performed from the upper edge of the subscapularis tendon to the biceps tendon. The thickened interval is released full thickness until the overlying conjoined tendon fibers are seen running longitudinally from superior to inferior (Fig. 17-7). The rotator interval division releases the coracohumeral ligament and anterosuperior capsule, which often inhibits external rotation with the arm at the side.

The anterior capsule over the subscapularis tendon is released next to reopen the subscapularis recess. Care must be taken to divide the capsule but preserve the underlying tendon and muscle belly. Significant contracture of the middle glenohumeral ligament is often encountered with obliteration of the recess and significant loss of external rotation. The release proceeds from superior to inferior with the depth marked by the rolled border of the subscapularis tendon and muscle fibers. This release is carried down to the anterior band of the glenohumeral ligament. The axillary nerve is certainly a concern inferiorly. The teres minor branch of the axillary nerve is found on average to be only 12.4 mm from the glenoid rim at the 6 o'clock position.[98] Performing the release close to the glenoid rim (~1 cm) decreases the likelihood of encountering the axillary nerve. The subscapularis muscle also acts as a buffer in the adducted position, which is one reason the beach-chair position is advantageous. Capsular scissors to separate the capsule from the underlying tissue is performed by some surgeons, while others use electrocautery using a hooked probe. Avoidance of muscle relaxation during use of electrocautery can be helpful in identifying when the nerve is near. Use of a posterior–inferior portal for the cutting instrument while viewing from either the standard posterior portal or anterior portal will allow greater ease of cutting the inferior capsule. Berghs et al. reviewed 154 patients with primary frozen shoulder, of which 25 underwent arthroscopic capsular release. They demonstrated dramatic improvements in pain and range of motion within 2 weeks of the procedure in 88% of the patients, which did not deteriorate over a 1-year follow-up. The patients who did poorly early in the postoperative rehabilitation also did poorly long term. In this study, the authors did not release the inferior capsule arthroscopically, but with a manipulation to avoid injuring the axillary nerve.[5]

The superior capsule is released above the glenoid rim until the underlying supraspinatus muscle belly can be seen. The release is carried from the rotator interval release to the posterior portal. A restriction in cross-body adduction and internal rotation generally indicates further constriction in the posterior capsule, warranting posterior capsular release. In these cases the viewing and working portals are switched and a posterior capsular release is performed near the rim until the infraspinatus muscle belly is seen. The arthroscopic release of the posterior capsule must be performed about 1 cm from the glenoid rim to prevent dividing the infraspinatus tendon.

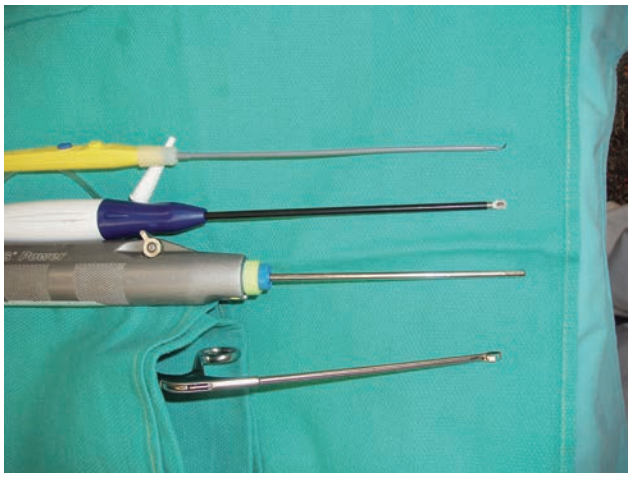
A small subset of patients may have an isolated posterior capsular contracture characterized by motion loss primarily limited to internal rotation, cross-chest, or horizontal adduction and flexion, with relative preservation of external rotation. This type of posterior capsular contracture has been implicated in impingement-type pain and may result in non–outlet-type impingement caused by increased anterosuperior translation during shoulder elevation and internal rotation. This condition is treated with posterior capsular release to restore lost motion and normal kinematics.[120]

Postoperative rehabilitation begins with immediate continuation of the range-of-motion exercises. Demonstrating the postoperative range of motion to the patient in the recovery room with the block in place is helpful to the patient's psyche. The patient is encouraged to perform the range-of-motion exercises three to four times per day. The sling is removed on the first postoperative day and the arm is used for daily activities. Any lifting, pulling, or pushing is avoided.

Arthroscopic capsular release has also been shown to increase range of motion in patients with secondary frozen shoulder seen after a previous operation or fractured shoulder.[50] Patients with postoperative frozen shoulder often have an extensive subacromial scar that can be resected arthroscopically. The scar is best excised with the scope brought in from a lateral portal and the shaver brought in posteriorly. Great care needs to be taken to protect the previous rotator cuff repair by keeping the open portion of the shaver at an angle to the tissue.

Open Release

Open surgical release is reserved for those patients for whom manipulation or arthroscopic release does not allow them to regain range of motion or for patients in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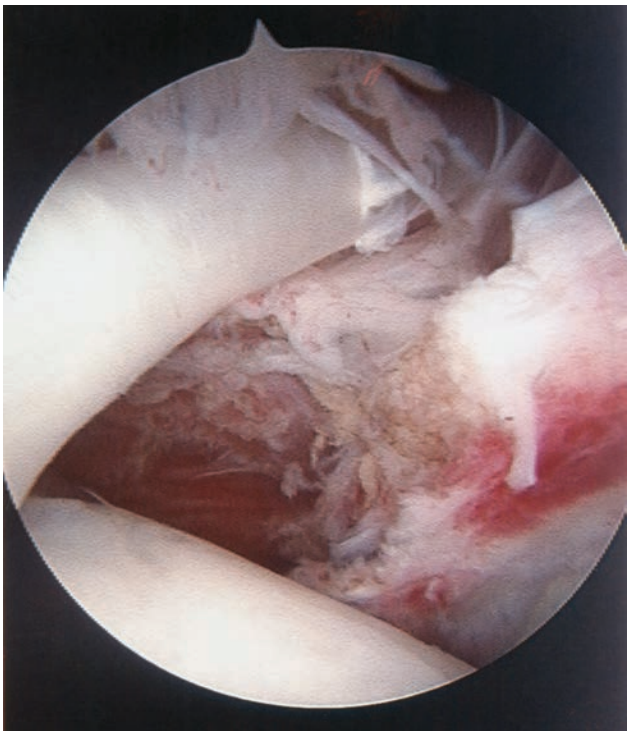
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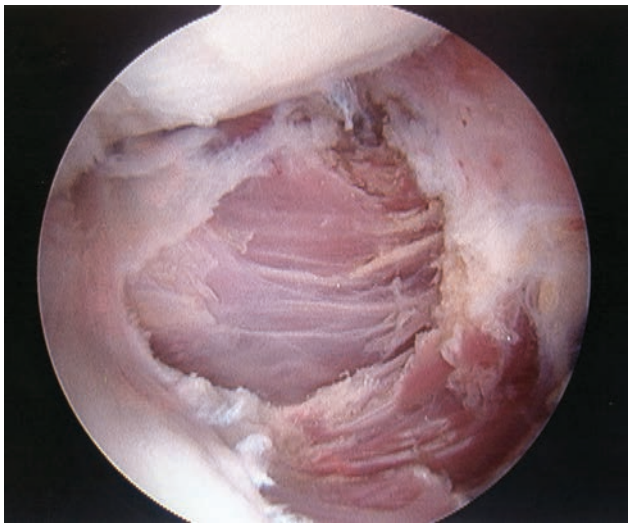
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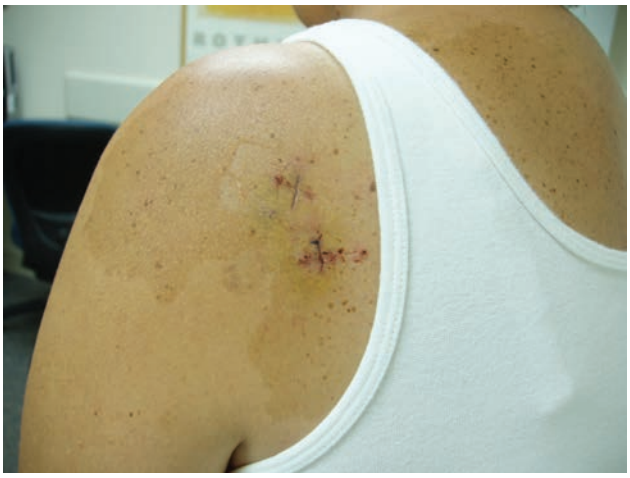
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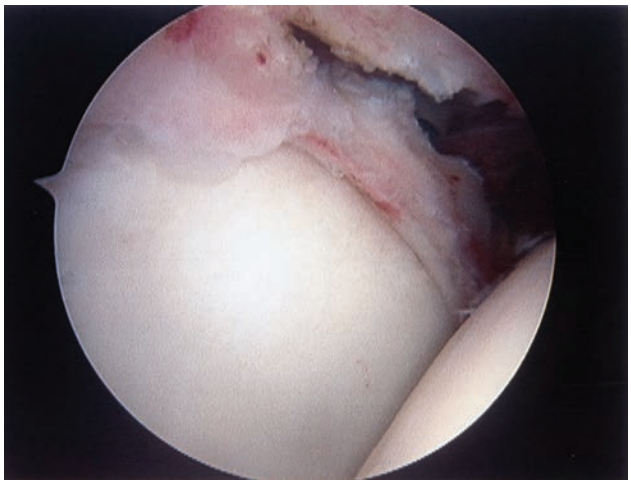
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Figure 17-7 **(A)** Instruments used for arthroscopic capsular release. **(B)** Intraarticular view of the anterior superior release. **(C)** Release of the inferior capsular pouch, which is often performed using the posterior inferior portal **(D)** and the posterior release **(E).**

whom the adhesions are primarily extraarticular. Frozen shoulder in the setting of prosthetic arthroplasty is best done by open surgery. In most cases of severe stiffness after arthroplasty, prosthetic malposition may also be a factor and open release alone may not be sufficient. Previous open repairs, especially involving the subscapularis tendon, may have scar tissue that may be difficult to release from a purely arthroscopic technique. In general, the goal of this procedure is to release contracted structures so that the range of motion can be increased while maintaining

glenohumeral stability. The advantage of open surgical release is that it can free up both sides of the joint with less risk to soft tissue or bony injury.

Open surgical releases generally have been successful, but experience and reports of large series have been limited.[59,73,74,84,86] The major advantage of this procedure is that it offers direct visualization of the structures undergoing release or lengthening. However, disadvantages include increased postoperative pain affecting ability to perform postoperative exercises as well as the risk of disruption of any lengthened tissues (i.e., subscapularis tendon). The structures released include subacromial and subdeltoid adhesions,[59,96] the coracohumeral ligament and rotator interval,[59,63,84,85,96] circumferential perilabral capsular release, and subscapularis release and lengthening.[7,94]

After induction of general and/or interscalene block anesthesia, a deltopectoral approach is employed for an open release of adhesions between the deltoid, acromion, coracohumeral ligament, coracoid, and strap muscles from the underlying rotator cuff. First, adhesions between the deltoid and the humerus are released either sharply or bluntly. Great care must be undertaken to protect the axillary nerve on the back side of deltoid approximately 3 cm distal to the acromion. The dissection is made easier with the shoulder in the abducted position, which allows the deltoid to become lax and more easily retracted. Dissection is started lateral to the coracoid and conjoined tendon to protect the axillary nerve. Internal rotation of the arm while gently retracting the deltoid muscle will allow release of the subdeltoid adhesions in an anterior to posterior manner until the deltoid can move freely over the proximal humerus when the arm is rotated. Within the subacromial space, the coracoacromial ligament should be incised with electrocautery and the extensive scar in the subacromial space excised. Care must be taken to identify the underlying rotator cuff and protect it.

Dissection deep to the conjoined tendon is performed next, with the surgeon being careful to use a combination of blunt and sharp dissection to protect the neurovascular structures. A plane can usually be developed above the subscapularis and beneath the conjoined tendon and a retractor placed to protect the neurovascular structures.

The coracohumeral ligament and rotator interval are next released by dissecting the supraspinatus and subscapularis from the coracoid process. The interval between the tendons is released sharply from the humerus to the coracoid.[84,86,96] A gentle manipulation can be performed to see if full motion can be achieved. If external rotation is still limited, the subscapularis tendon and anterior capsule need to be addressed. This can be achieved by splitting the subscapularis or by performing a tenotomy from the lesser tuberosity and sharply developing the plane between the muscle and the capsule, followed by excision of the capsule while protecting the subscapularis. Another technique involves a coronal Z-plane lengthening, where a vertical incision is made in the superficial fibers of the tendon at its insertion on the lesser tuberosity. The subscapularis is then separated from the underlying remaining tendon and capsule and reflected medially. The superficial half of the tendon remains attached to the muscle, with the remaining deep half divided at the glenoid and remaining attached to the lesser tuberosity. The deepened or remaining tendon and capsule are then incised from the labrum medially. This technique is usually reserved when the prior surgery involved shortening the subscapularis. The orientation of the coronal dissection can be guided by determining the thickness of the scarred tendon and capsular tissue once the rotator interval region has been opened. The subscapularis is usually entrapped in scar tissue. To achieve full mobility, it may be necessary to visualize and dissect the axillary nerve. With the nerve adequately visualized and protected, the subscapularis can be released circumferentially on its superior, inferior, deep, and superficial surfaces.

If abduction and internal rotation are still limited, the inferior and posterior capsule can be released. With the axillary nerve identified and protected, the inferior capsule is released just lateral to the labrum and extended into the posterior capsule. To aid in visualization of the posterior capsule, a humeral head retractor may be placed to displace the humeral head posteriorly. At the end of the release, the Z-plasty is closed with nonabsorbable suture by stitching the lateral end of the superficial subscapularis to the deep end of the tendon and capsule. Each centimeter of length gained from the lengthening increases external rotation by approximately 20 degrees (Fig. 17-8). Range of motion is assessed again to determine where there is tension on the soft tissue repair and thus define a safe zone for early passive range of motion and rehabilitation.

The results of open release of recalcitrant chronic adhesive capsulitis of the shoulder and the role of contracture of the coracohumeral ligament and rotator interval were evaluated in a study by Ozaki et al. Seventeen patients who failed to improve with standard nonoperative measures underwent open release through an anterolateral incision. Release concentrated on the hypertrophied coracohumeral ligament and contracted tissues within the rotator interval at operation. The major cause of restricted glenohumeral movement was contracture of these structures. Histologic study revealed fibrosis, hyalinization, and fibrinoid degeneration in the contracted connective tissues, as well as fibrosis of the subsynovial tissue and absence of the synovial cell layer on the joint side of the rotator interval. Once release had been performed within the rotator interval, the glenohumeral joint was then gently manipulated through a full range of motion, also mobilizing the biceps tendon within its groove.

Immediately postoperatively, active mobilization of the shoulder was begun. At an average follow-up of 6.8 years, 16 patients were reported to have full range of motion, with one presenting with a slight limitation of motion. The

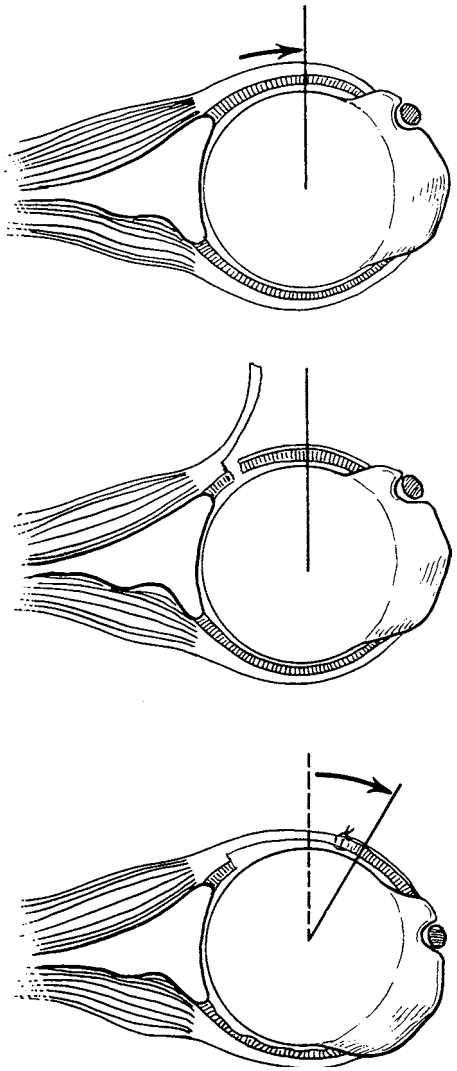

Figure 17-8 Contracted subscapularis and anterior capsule (*top*) limit external rotation. This condition is treated by incising the subscapularis from the lesser tuberosity laterally (*middle*) and suturing it to the medial end of the capsule, which is transected close to the glenoid (*bottom*). This release results in substantial lengthening of these structures. Each centimeter of length gained increases external rotation by approximately 20 degrees.

authors stated that they felt the essential changes in the tissue at the time of surgery could have been related to an intrinsic disorder of collagen and therefore recommended open release of contracted tissue.[96]

COMPLICATIONS

In most patients, frozen shoulder runs a self-limited course and will improve over time. Complications associated with frozen shoulder are usually iatrogenic and are the result of the treatment used. Potential complications of manipulation include fractures about the proximal humerus and shaft, fractures about the glenoid rim, dislocation of the glenohumeral joint, rotator cuff tears, labral detachment, or brachial plexus stretch injury. Additional complications include recurrent stiffness or axillary nerve injuries secondary to arthroscopic or open release.

Proximal humeral or shaft fractures represent a significant risk with manipulation, especially in the osteoporotic elderly population. Another risk factor is the osteopenia that occurs with regional pain syndromes and frozen shoulder. Treatment of the fracture is directed at early mobilization by performing an open reduction, internal fixation of the fracture. The best treatment consists of avoidance of the complication with gentle manipulation and avoidance of torquing the humerus, especially in external and internal rotation. The advent of arthroscopic capsular release allows direct lysis of adhesions in a controlled fashion without the risk of fracture.

Glenoid rim fractures or labral detachments may occur secondary to the pull of the ligaments during manipulation. These complications would go unrecognized except for the advent of the arthroscopic evaluation after manipulation. Instability is a rare complication and no repair is warranted. In fact, repair of these lesions would lead to further stiffening and loss of motion.

Rotator cuff tears rarely occur as a result of manipulation. Most tears were probably present prior to the advent of the stiff shoulder and its treatment. Repairing a rotator cuff tear in the presence of a frozen shoulder markedly increases the risk of pain and stiffness. Acute large retracted tears can be repaired with a concomitant capsular release and early postoperative rehabilitation. Leaving these large tears unfixed for a long duration may make them difficult to fix on a delayed basis, and the pain and dysfunction from the tear may make treatment of the stiffness difficult. Repair of small chronic tears, especially partial-thickness tears, should be reserved until after the stiffness resolves. Repair of these lesions may lead to severe postoperative pain and stiffness.

Instability is a rare complication after manipulation despite the extensive release of the capsule. Overhead suspension of the arm should be avoided in the regional blocked extremity. Inadvertent subluxation or dislocation may go unrecognized with risk to the axillary nerve. Overhead suspension can also be a source of a neurologic stretch injury.[89] Traction injuries of the brachial plexus and peripheral nerves have been reported after closed manipulation of the shoulder.[9] These injuries are usually neurapraxias that should recover spontaneously over time. It is critical to maintain passive motion while the nerves are recovering to avoid recurrent stiffness. Although a sling should be used to prevent excessive traction on the plexus while it is recovering, it should be removed for physical therapy and home exercise sessions. The patient and the physical therapist need to be made aware of the problem so that extremes of range of motion, particularly forward elevation and

abduction, are avoided to prevent further nerve injury. The diagnosis of middle and lower plexus injuries can be made almost immediately with somatosensory-evoked potentials. Diminished sensory nerve action potentials at 5 days after the injury may also be helpful in diagnosing an early plexopathy. Definitive electrodiagnostic studies can be performed 3 weeks after the injury to evaluate the extent of nerve damage and the potential for recovery.

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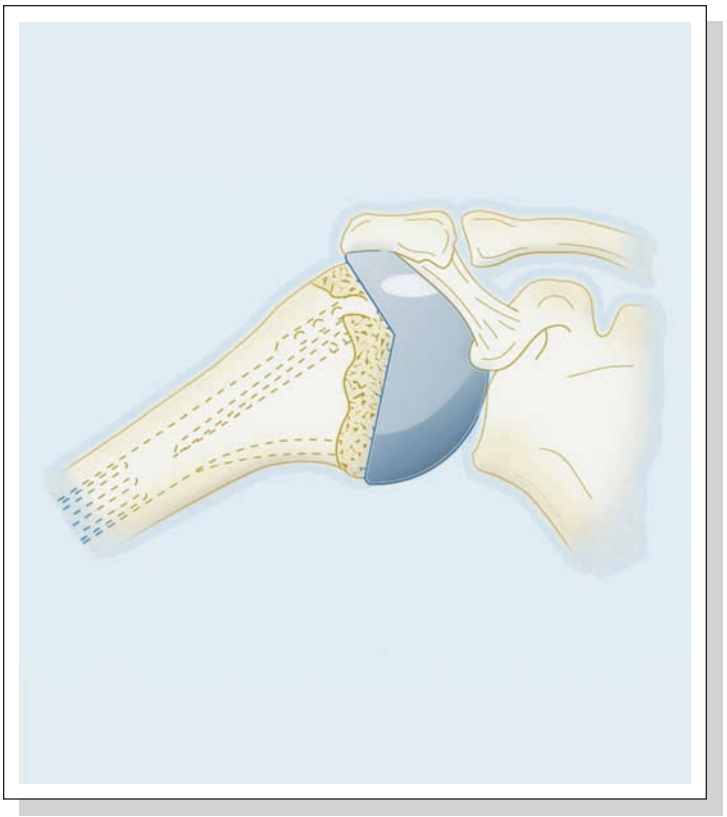
Glenohumeral Joint Arthritis and Related Disorders

IV



18 Pathophysiology, Classification, and Pathoanatomy of Glenohumeral Arthritis and Related Disorders

David N. Collins

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INTRODUCTION

Regardless of the cause or the location of the joint, the pathologic changes of arthritis have the greatest effect on the articular cartilage and underlying bone. Initially, the soft tissue involvement includes only the synovium, but later it may involve, both directly and indirectly, the joint capsule, ligaments, and adjacent tendons. The progression of arthritis is not only influenced by the existing disease processes, but also by the extent and rate of joint loading by mechanical forces.

The predominant clinical feature of arthritis is pain. For most major joints, this is on the basis of the intraarticular changes of arthritis. Unlike other joints, however, the shoulder is more often affected by periarticular soft tissue disorders, which may coexist with glenohumeral arthritis and influence its clinical course. The functionally important musculotendinous cuff, along with the capsule and supporting ligaments, may undergo secondary changes as a result of glenohumeral joint arthritis. These include muscular weakness, pathologic musculotendinous shortening, and attenuation of ligamentous tissue. With articular disease progression and the associated direct or indirect involvement of the unique soft tissue envelope of the glenohumeral joint, the clinical effect will be profound.

The glenohumeral arthritides and associated disorders can be among the most challenging diagnoses. The clinician is required to use the acquired skills of careful history taking, thorough physical examination, and prudent use of cost-effective testing. Glenohumeral arthritis and allied conditions include osteoarthritis, posttraumatic arthritis, arthritis of dislocation, osteonecrosis, rheumatoid arthritis, crystalline arthritis, rotator cuff tear arthropathy, noninfectious inflammatory arthritis, and miscellaneous arthropathies.

Shoulder pain is not only a very common symptom that clinicians are asked to evaluate, but also the chief complaint of patients with glenohumeral arthritis. Acute traumatic shoulder pain is reviewed elsewhere in this textbook. This chapter will highlight the major causes of glenohumeral arthritis and profile the characteristics of each disorder.

Clinical Presentation

Although the shoulder is no less susceptible than other diarthrodial joints to arthritic conditions, arthritis is not a typical cause of shoulder pain. Probably as few as 5% to 10% of patients with shoulder pain have arthritis as a part of a polyarticular systemic disease, or a monarticular disease process. Painful dysfunction is more often a disorder of one or more of the soft tissue elements of the shoulder.

There exist more than 100 different types of arthritis. They may be defined as inflammatory or degenerative, polyarticular or monarticular, and acute or chronic. Distinction is made from historical factors, physical findings, radiographic analysis, and tissue sampling, including synovial fluid analysis and serum studies.

The predominant symptom of patients with arthritis is pain, usually intensifying with use and interfering with sleep. Shoulder motion is restricted by synovial membrane inflammation and increased fluid production. The synovium and capsule are richly innervated structures easily irritated by distention from the accumulation of synovial fluid. Exacerbation of discomfort with rotary movement with the elbow at the side is a characteristic physical finding of an inflammatory condition of the glenohumeral joint. Degenerative conditions are more tolerant of passive range of motion. Tenderness over the posterior joint line, as noted by Neer, is notable in glenohumeral osteoarthritis.[361] Tenderness lateral and inferior to the coracoid process, the anterior joint line, is very suggestive of an inflammatory disease. It is commonly not elicited in rotator cuff disease or noninflammatory conditions.

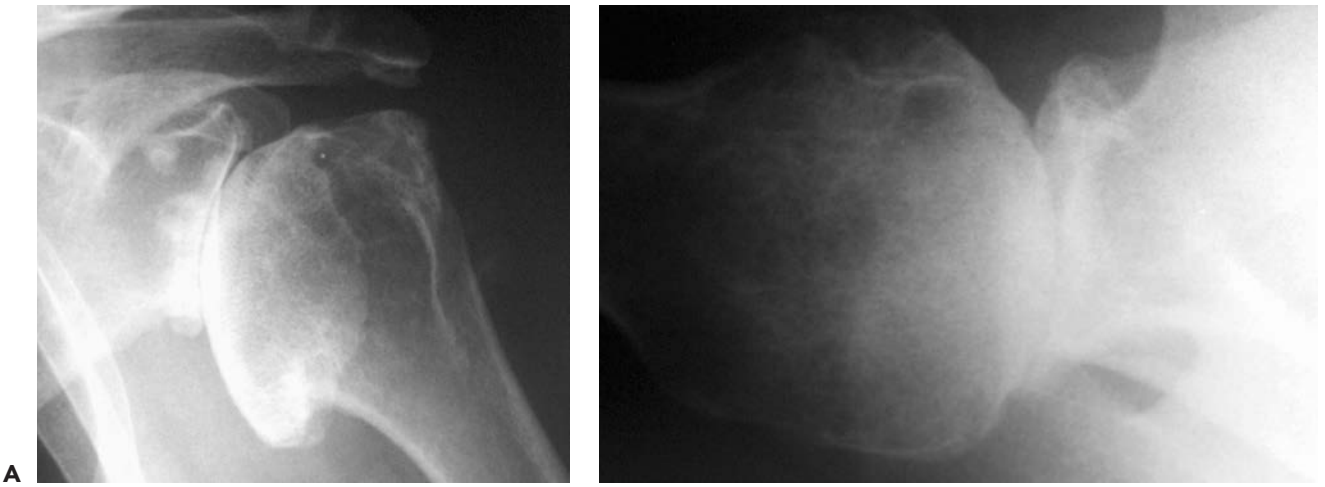


Figure 18-1 **(A)** True anteroposterior view of the glenohumeral joint and **(B)** transaxillary lateral view, adequately demonstrating moderately severe osteoarthritis.

Imaging

Green and Norris have provided an excellent review of imaging strategies to be employed for patients with glenohumeral arthritis.[163] Initial evaluation of the painful shoulder will almost always include plain radiographs.[246] The most commonly employed views for the orthopedic surgeon are the true anteroposterior (AP) of the glenohumeral joint orthogonal to the plane of the scapula and the transaxillary lateral. Glenohumeral arthritis is easily distinguished from other soft tissue conditions and is classified based on features identified on plain films (Fig. 18-1).[430] These include alignment and relation of the humerus to the glenoid and acromion process, the width of the articular cartilage, osseous erosions, productive changes including osteophytes, and the presence of soft tissue swelling and calcification.

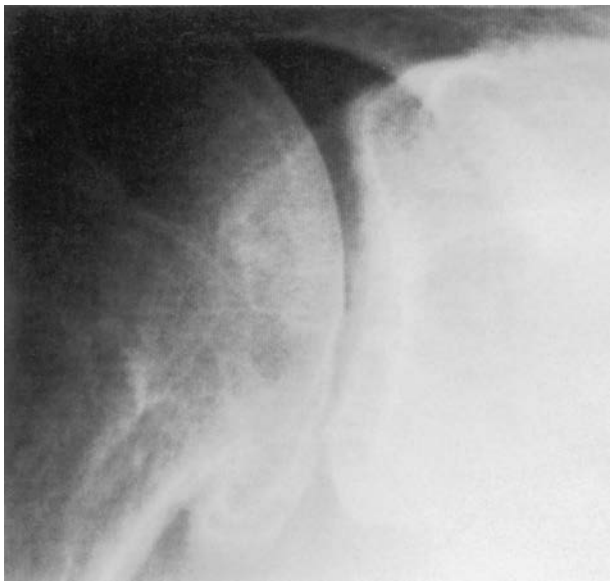


Figure 18-2 Humerus in 35 degrees of external rotation, profiling the articular surface in the true anteroposterior plane of the scapula.

Distinction of the articular cartilage space is best made when the x-ray beam is directed tangent to the joint surface. For the shoulder, this requires accurate patient, shoulder, and extremity positioning. Once the plane has been determined, the humerus can be externally rotated to approximately 35 degrees (Fig. 18-2). This will profile the load-bearing portion of the articular surface so that the earliest narrowing does not escape detection, and it is also the position favoring visualization of marginal humeral head osteophytes.[247] Apple et al. have suggested a weighted abduction Grashey shoulder method as a more sensitive means of detection of loss of articular cartilage.[13] When the disease is more advanced, positioning the humerus in external rotation is difficult and cannot be done exclusive of the position of the scapula (Fig. 18-3). For Nelson and colleagues, plain radiography, as opposed to magnetic resonance imaging (MRI), computed tomography (CT) arthrogram, and ultrasonography, was most useful for the diagnosis of osteoarthritis.[367]

Although shoulder arthrography is employed mostly for detection of rotator cuff tears, the articular surfaces and contours of the synovial lining of the joint can be easily visualized.[503] The arthrographic features may be enhanced by the use of poly- or computed tomography.[459] Filling defects may be observed in the cartilaginous surfaces and the thickness of the residual cartilage estimated. Magnetic resonance arthrography using gadopentetate dimeglumine may offer a more sensitive method for detection of intraarticular abnormalities.[265,384] These techniques, for practical

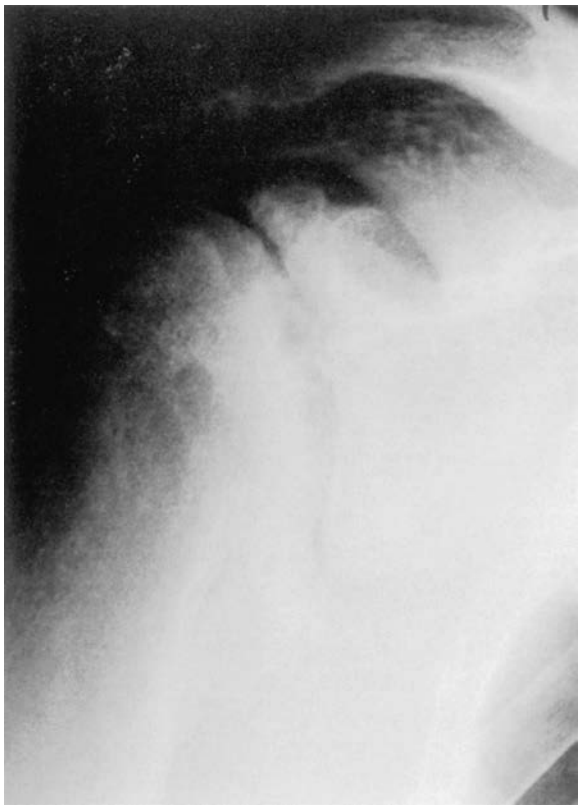


Figure 18-3 More advanced osteoarthritis, requiring total rotation of the humerus with the torso, a result of fixed internal rotation contracture of the glenohumeral joint.

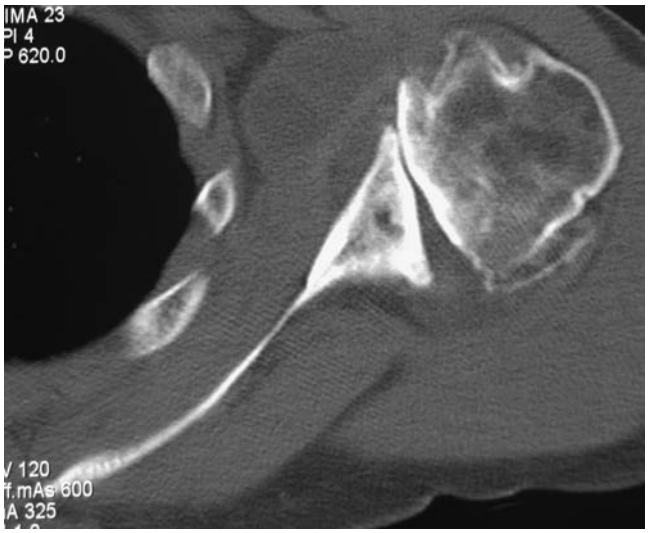


Figure 18-4 Computed tomography scan of osteoarthritis demonstrating a biconcave glenoid. There is posterior glenoid erosion that has a radius of curvature distinctly different than the anterior glenoid, where a small amount of articular cartilage persists. Peripheral humeral head osteophytes are apparent.

purposes, are rarely employed by the author for the routine assessment of glenohumeral arthritis.

Ultrasound study of the glenohumeral articulation has a very limited, yet undefined, role in the evaluation of the articular surfaces.

Although generally not required for the diagnosis of articular disorders, a CT scan can be quite helpful to delineate the extent of bone involvement and assess glenoid orientation (Fig. 18-4).[142,348] Glenohumeral morphology can be detailed, as can the quality and quantity of the glenoid. CT scanning will also help detect subtle cortical erosions, deposits of calcium, and the presence of newly formed bone. A more accurate assessment of glenohumeral relations may be realized by CT when extreme stiffness or deformity does not allow the shoulder to be positioned for optimal plain film study.

Perhaps the best imaging study for detecting early chondral injury or damage is MRI, even though assessment of the articular cartilage can be technically challenging.[156] Hayes et al. stated that an accurate MRI scan of articular cartilage requires good spatial resolution for the detection of small defects, good subject contrast and image contrast for the detection of signal intensity changes in articular cartilage, reliable distinctions for the detection of signal intensity changes in articular cartilage, and reliable distinction between articular cartilage and adjacent subchondral bone and joint fluid.[192] Broderick et al.'s study drew attention to the tendency for cartilage abnormalities to be underestimated with MRI when compared with arthroscopic observations.[43] The use of injectable contrast agents to enhance the accuracy of articular cartilage is not generally necessary.[62,265,324]

There is a consensus that normal articular cartilage can be imaged by MRI, perhaps to the extent of "zonal" analysis.[77,337,387,441] Three-dimensional imaging data can provide information about joint surface topography utilizing refined techniques.[17,61,390,391,418,501] Quantitative MRI is under investigation as a means of earlier detection and as a tool for monitoring the response of the articular cartilage to certain clinical conditions and to treatment.[176,517]

With few exceptions, scientific studies of MRI of articular cartilage involve the knee.[34,77,171,175,205,324,325,390,418] Huber et al., in one of the earliest published reports on MRI of the normal shoulder, cited the failure of MRI to effectively define articular cartilage.[212] The literature was replete with studies of MRI of the shoulder that had assessed features of the rotator cuff and capsulolabral morphology in impingement and instability disorders, respectively, and rarely mentioned the findings of the glenohumeral articular cartilage. Recent monographs and textbooks concerned with MRI of the shoulder and shoulder imaging inadequately address arthritis imaging by MRI, particularly the articular cartilage changes.[468,512,556] The preferred timing, sequences, and technical parameters have not been determined with uniform success and are evolving.[204] Therefore, MRI applications for glenohumeral

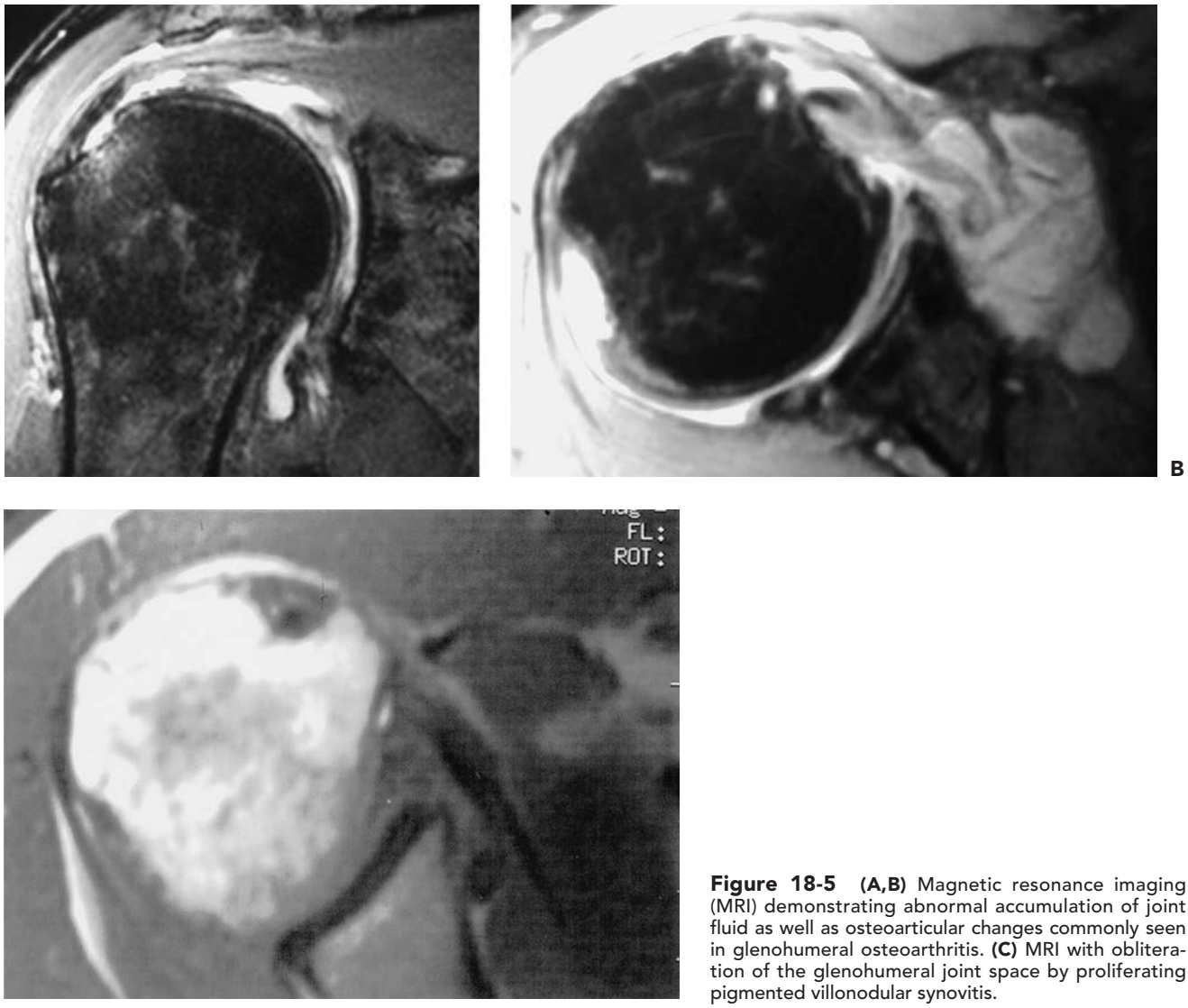


Figure 18-5 **(A,B)** Magnetic resonance imaging (MRI) demonstrating abnormal accumulation of joint fluid as well as osteoarticular changes commonly seen in glenohumeral osteoarthritis. **(C)** MRI with obliteration of the glenohumeral joint space by proliferating pigmented villonodular synovitis.

articular disorders are not yet as extensive as for the knee. Zlatkin has stated that MRI is rarely required to assess patients for glenohumeral osteoarthritis alone.[557] MRI arthrography utilizing gadolinium compounds may prove to be an effective technique to enhance the glenohumeral articular surfaces.[170,265]

A shortcoming of conventional radiographs is failure to detect soft tissue details, although in some cases swelling can be identified. Joint effusions may be suspected on plain films, but are best evaluated by ultrasound, CT, or MRI. The MRI is probably the most useful, accurate, reliable, and expensive.[512,541]

In normal shoulders, the MRI findings show only a thin rim of joint fluid around the biceps tendon sheath and narrow bands in the axillary recess and subscapularis bursa.[460] The volume has been observed to be increased with age.[460] Accumulation of glenohumeral joint fluid is abnormal; usually it is related to rotator cuff tears or osteoarthritis (Fig. 18-5 A,B).[460] The intravenous administration of gadopentetate dimeglumine will enhance the MRI of joint fluid, although the response is slower and less vigorous than synovium.[540]

Use of MRI allows direct visualization of inflammatory synovitis and synovial proliferation within the joint, as well as its penetration into the adjacent bone and periarticular soft tissues (Fig. 18-5 C).[422,542] Contrast enhancement with the use of gadolinium provides a more specific assessment of synovitis or pannus.[262,422] The rate and intensity of synovial enhancement may vary, depending on the activity of the synovium as a reflection of the activity of the disease process, as well as the image timing.[540]

Radionuclide isotope uptake in tissue reflects the rate and extent of blood flow through the tissue and, in bone, is a measure of bone metabolism, especially osteoblastic

activity. Scintigraphy, in particular triple-phase bone scan, may prove useful in distinguishing soft tissue inflammation from osteoarticular changes.[153,302,510] It is very sensitive, but it does not distinguish acute inflammatory disease from bone and joint sepsis.[509] Technetium scans have been used to confirm the presence of rheumatoid arthritis before the appearance of radiographic abnormalities.[110] Indium-111 chloride has been used for detection of rheumatoid arthritis as well as for following the course of disease activity.[450,467] Immune complex scintigraphy holds promise for monitoring disease activity and response to treatment.[96,251] MRI may have clinical application in the early detection of rheumatoid arthritis, its staging, and the assessment of treatment outcomes.[29,44,554] Uptake defects can exist in osteonecrosis and can be enhanced with single photon emission computed tomography (SPECT) images.[511,531] White cell–labeled scans are useful when an infectious disease process is being considered.[510,520]

Laboratory

Serum studies may be helpful, but only rarely can a diagnosis be obtained from this information alone. Complete blood count, autoantibodies (rheumatoid factor [RF] and antinuclear antibody [ANA]), uric acid and acute-phase reactant measurements, erythrocyte sedimentation rates (ESRs), and C-reactive protein (CRP) are most often performed as an initial screening battery and most helpful when the diagnosis is in question.[144,453] Acute-phase reactants are serum proteins formed in the liver and include coagulation, transport, complement, and miscellaneous reactive proteins. Their production is accelerated in the presence of inflammatory states and tissue necrosis.[145,272,454] The ESR is a rough, indirect quantification of their serum levels. The CRP level has been used more recently. Both are serum markers that reflect the extent or degree of inflammation and can be used for assessment of disease activity or response to treatment over time.[76,272] CRP levels (normally less than 1 mg/dL) may exhibit moderate elevation (1 to 10 mg/dL) in most connective tissue diseases; marked elevation (more than 10 mg/dL) may signify an acute bacterial infection such as septic arthritis.[342]

Clinically detectable effusions are uncommon in most arthritides. When present or suspected, however, a fluid sample should be obtained and submitted for analysis.[493] It should also be obtained any time the patient's history, physical examination, and radiographs support the diagnosis of arthritis but a diagnosis has yet to be determined.[147] The findings of joint fluid analysis have been shown to change a clinically suspected diagnosis, and often treatment, in 20% of samplings.[120] Particularly in acute arthritis, synovial fluid analysis is of major diagnostic value.[493]

Arthrocentesis of the shoulder can be performed from the anterior or posterior approach. Anteriorly, the tip of the coracoid process is palpated. After locally instilled anesthesia, an 18- or 21-gauge spinal needle is passed through the deltoid muscle at a point approximately 1.5 cm inferior and 1.5 cm lateral to the tip of the coracoid process. The cephalic vein is nearby and may be inadvertently punctured. Entry into the joint is gained with passage through the subscapularis muscle and the joint capsule. Posteriorly, the point of entry is two to three fingerbreadths inferior and one to two fingerbreadths medial to the posterolateral corner of the acromion process. Directed toward the coracoid process, the needle will penetrate the deltoid and infraspinatus muscles to enter through the capsule into the joint.

As much fluid as possible is withdrawn and its volume is determined. It is characterized as shown in Table 18-1.[80] The gross characteristics are noted. These include viscosity and color, which is normally clear and yellowish tinged. The fluid is typically not bloody, although blood tinging may suggest a traumatic tap or the existence of a pathologic lesion.

Fluid in normal joints is present in quantities that may preclude sampling by arthrocentesis. Recht et al. detected fluid in 14 of 20 shoulders by MRI in 12 asymptomatic young volunteers; in none was more than 2 mL evident.[419] If the joint fluid is obtained, the white count is less than 200 and predominantly monocytic as opposed to polymorphonuclear, and there are no red cells.[147]

Additional joint fluid types have been noted.[147] Noninflammatory fluid has a white cell count usually less than 2,000.[80] The fluid is transparent or nearly so. This type of fluid can be seen in trauma, osteoarthritis, systemic lupus erythematosus (SLE), sarcoid, and hypothyroidism.

Inflammatory fluid may show varying degrees of clarity and color. The white cell count is more than 2,000, but usually not more than 50,000.[80] This is commonly seen in rheumatoid arthritis, gout, and possibly some infectious disorders. It is also associated with Reiter's syndrome, ankylosing spondylitis, psoriatic arthritis, and juvenile rheumatoid arthritis.

Pyogenic fluid is opaque or grossly purulent. The white cell count exceeds 50,000, often higher, and is predominantly polymorphonuclear cells.[80] This is typical of infectious arthritis and, in some cases, gout or other very inflammatory arthritides.

Nonpyogenic fluid has variable characteristics. The white blood cell count is usually less than 20,000.[80] Additionally, joint fluid can be characterized as hemorrhagic. This will occur in cases of trauma, hemophilia, and other bleeding disorders.

Microscopic identification of crystals is a laboratory test with notable problems in sensitivity, specificity, and

TABLE 18-1
SYNOVIAL FLUID ANALYSIS

	Normal	Noninflammatory	Inflammatory	Pyogenic	Nonpyogenic
Gross analysis					
Volume	1–4 mL	Increased	Increased	Increased	Increased
Color	Clear, pale yellow	Yellow (xanthochromic)	Yellow-green, white	Yellow, white, gray	Variable
Clarity/turbidity	Transparent	Transparent	Transparent to opaque	Opaque, purulent	Variable
Viscosity	Very high	High	Low	Very low/variable	Decreased/variable
Mucin clot	Good (tight)	Good/fair	Fair/poor (friable)	Poor (friable)	Fair/poor
Spontaneous clot	None	Often	Often	Often	Variable
Microscopic analysis					
Leukocytes	<200	200–2,000	2,000–50,000	15,000–200,000	10,000–20,000
Neutrophils	<25%	<25%	25%–75%	>75%	50%–75%
Organism stains	Negative	Negative	Negative	Positive	Variable
Predominant cell types	Mononuclear	Mononuclear	Polymorphonuclear	Polymorphonuclear	Polymorphonuclear
Chemical analysis					
Fluid/serum glucose	1:1	0.8–1.0	0.5–0.8	<0.5	<0.5
Protein (g/dL)	<2.5	<2.5	2.5–8.0	2.5–8.0	2.5–8.0
Lactic acid	Plasma	Plasma	>Plasma	>Plasma	>Plasma
Culture	Negative	Negative	Negative	Positive	Positive

From ref. 38, with permission.

interobserver differences.[463] Despite this observation, the fluid should routinely be examined for calcium pyrophosphate and uric acid crystals that are further characterized by polarized light microscopy. Gout crystals are pointed and negatively birefringent.[148,396] Calcium pyrophosphate crystals are rhomboid and positively birefringent.[148,396]

Synovial fluid glucose levels are normally approximately 20 mg/dL less than the serum. Lower concentrations may be observed in joint sepsis. Synovial fluid protein is often increased in inflammatory disease. The utility of synovial fluid glucose and protein levels is questionable.[493] Appropriate organism stains and cultures should be obtained when there is even the most remote suspicion of an infectious process.

OSTEOARTHRITIS

Definition

Recognized as the most common of all arthritis, osteoarthritis is an irreversible, slowly progressive arthropathy characterized by the focal loss of articular cartilage, with hypertrophic reaction in the subchondral bone. Its cause is unknown. Although it does not seem to be a single disease, common features are found during the evolution of the disorder as it arises in a number of different conditions.

Incidence

The occurrence of osteoarthritis at the glenohumeral joint is much less common than at the hip or knee.[351] It is more likely to be found in women than men and in patients over 60 years of age.[351] Its incidence increases with age, and it is the site with the oldest average age of onset.[91,234] Difficulty in determining the early diagnosis, the timing of its onset, and the absence of longitudinal data make estimates of the prevalence of osteoarthritis and its incidence imprecise.

DePalma had an early interest in the aging shoulder and carefully studied its morphologic aspects.[100,102] On the glenoid side, from his study of cadavers ranging in age from 14 to 87, he determined that the degenerative process reached its maximum by the sixth decade. The most significant changes seemed to be located at the superior aspect of the glenoid. At the glenoid site of labral attachment, particularly in the anterior and anteroinferior regions, there was a marginal proliferation of bone and cartilage. This was believed to be secondary to traction forces applied to the soft tissues in these

areas, with a resultant functional response to stress loading. Over time, a generalized thickening of the synovial membrane was observed. This occurred secondary to proliferation of the fibrous stromal elements in the synovial areas, resulting in an increased number of villous projections. On the humeral side, articular surface changes were never profound. The changes were certainly never equal to those seen on the glenoid side and were felt to be attributable to the mismatch of the humeral to glenoid surface area (4:1).

Cadaver studies by others have confirmed the relative absence of humeral cartilage thinning with increasing age.[330,393] Petersson and Raedlulnd-Johnell radiographically reviewed the glenohumeral joint spaces in normal persons and concluded that, with age alone, the joint space does not decrease.[392]

Pathogenesis

Age is the greatest risk factor for the development of primary (idiopathic) osteoarthritis in all joints.[39,395] Additional systemic factors (gender, nutrition, race, ethnicity) and intrinsic joint vulnerabilities (previous damage, bridging muscle weakness, misshaped joint, malalignment, proprioceptive deficiencies) may increase one's susceptibility to osteoarthritis, and under the influence of loading conditions (obesity, trauma, physical activities), osteoarthritis may evolve or progress.[87–90] Bone density may play a role, as noted by the apparent inverse relation between osteoarthritis and osteoporosis.[257] There are no special associations in glenohumeral arthritis, except a coexistence of degenerative changes, including tearing, of the rotator cuff.[84,92–95] Feeney et al. identified a strong correlation between tears of the rotator cuff and degenerative changes of the articular cartilage that was independent of the factor of age.[130] However, the area of cartilage damage does not necessarily increase as the size of the tear increases.[211] Moreover, the incidence of full-thickness rotator cuff tears in osteoarthritis is low.

Pathophysiology

For details on the pathophysiology of osteoarthritis in general, the reader is referred to major textbooks of rheumatology, recent monographs, and reports discussing articular cartilage and osteoarthritis.[97–102] Most epidemiologic, clinical, and in vivo data come from studies of osteoarthritis of the large weight-bearing joints, the hip and the knee, as opposed to the glenohumeral joint. Suffice to say, the pathophysiology of osteoarthritis is poorly understood.

Osteoarthritis may not be so much a disorder of the articular cartilage as a response within the joint as an organ that ultimately fails.[412] Stresses applied to the joint exceed its capacity to repair what initially may be reversible changes. Radin and others have advocated the critical role of subchondral bone in the diseases process.[413] Day et al. suggest that subchondral stiffening is compatible with the process of normal bone adaptation.[95] Bone turnover may be increased severalfold, a process that ultimately may weaken the subchondral plate with the induction of altered properties of mechanical support for the articular cartilage.[21]

Normal cartilage is maintained in homeostasis by the repair capacity of chondrocytes within the cartilaginous matrix.[513] A general hypothesis for the initiation of osteoarthritis is that altered mechanical forces initiate metabolic activity change within the chondrocytes.[108–113] Loading characteristics, fluid mechanics, and joint congruity each contribute in special ways. Presuming that normally a feedback mechanism exists between the regulation of activity and mechanical stimulation, an imbalance of cartilage activity may result when the mechanical signal, or its transduction mechanism, is changed. The precise mechanism is unknown, but Mow and others suggest that the process is mediated by altered chondrocyte activity.[347] A cascade of events leads to the formation of a structurally impaired and weakened matrix that has irreversibly changed load–response characteristics.[347] Fragments of extracellular matrix proteins have been shown to bind to receptor sites on chondrocytes, inducing chondrolysis.[428] As a result, the capacity for cartilage to repair appears to fail, a dysfunction that ultimately leads to its loss.

Another hypothesis is that with increasing age, by the phenomenon of senescence, the chondrocytes lose their ability to replace their extracellular matrix, resulting in the deterioration in the structural and function properties of the cartilage and thereby increasing the risk of osteoarthritis.[306] Subject to the simplest biomechanical stress, the failing chondrocytes may produce proinflammatory cytokines (interleukin-1 and tissue necrosis factor-alpha) and chemokines that further contribute to the dysregulation of the chondrocytes, ultimately leading to progressive degradation of the cartilage matrix.[38,159] The local production of nitric oxide, prostaglandins, and hydrogen peroxide unfavorably alter the chondrocytes' response as well.[158] Anabolic mechanisms that normally support cartilage health are impeded.[507] It is compelling to meld aspects of both theories and envision a plausible scenario whereby altered mechanical forces are imposed upon chondrocytes with declining activity and responsiveness, the end result being osteoarthritis.

The major forces on articular cartilage result from contractions of the muscles that impart stability and motion to the joint.[421] Although the glenohumeral joint in normal persons is not truly weight bearing, the forces across the joint are not insignificant: 0.9 × body weight when the arm is held between 60 and 90 degrees of abduction.[401,402] Factors of load magnitude, its direction and duration, as well as the distance between the point of application and the

joint may influence the force applied to the articular cartilage. In a finite element analysis model, higher forces were expected on the posterior glenoid as the osteoarthritic shoulder, with its altered geometry, and normal shoulders are externally rotated.[47]

In addition to normal load, a depletion of articular cartilage may occur from joint trauma. Whereas chronic overloading of normal joints or subtle changes in joint mechanics may evolve slowly into osteoarthritis, significant joint injuries may precipitate significant osteoarthritis within a short time. An analogy for the shoulder would be the fate of the glenohumeral joint with glenoid dysplasia or hypoplasia, compared with one sustaining a comminuted displaced intraarticular glenoid fracture that resulted in an unfavorable malunion.[479,544]

Repetitive stresses may play a role, depending on the type of force transmitted to the joint. Articular cartilage can withstand significant shear forces without incurring irreversible damage.[291] Harryman et al. showed a wide variety of measurable translation in normal shoulders, implying an internal shear component during physiologic glenohumeral motion.[183]

With another in vitro model, the amount of glenohumeral translations was significantly influenced by the degree of humeral external rotation.[240] Higher than normal stresses on the articular cartilage leading to osteoarthritis may result from chronic posterior subluxation of the humeral head due to excessive glenoid retroversion.[525]

The thickness of articular cartilage is unfavorable for effective shock absorbency.[411] Impact loading forces are thereby delivered to the subchondral bone and the surrounding joint soft tissues.[197,411] The major bone load is transmitted to and attenuated by the cancellous subchondral trabeculae and the interposed fluid marrow elements.[372,413] This concept may help explain the observation concerning the inception of osteoarthritis predominantly on the glenoid side: less surface area, less cancellous bone volume.[101,416]

Acute ligamentous injuries alter the mechanics and biologic properties of the joint, especially its articular cartilage. The most extensively studied animal model for this condition has been the canine anterior cruciate ligament transection.[136–138] The mechanical and biomechanical components combine to progressively change the articular cartilage, not unlike that known to occur in osteoarthritis in the humans.[40]

Impact loading by mechanisms of trauma results in a more rapid or sudden increase in the force acting upon the articular cartilage. Experimental studies have demonstrated cellular changes with increased hydration and increased cellular activity, suggesting a repair or a remodeling capacity.[111,502] The overall effect is a weakening of the cartilage.

Just as there are conditions that alter joint load applied to normal cartilage, there are also those that infiltrate the cartilage or alter cartilage metabolism, rendering it vulnerable to physiologic loading alone. Primary disturbances in cartilage metabolism may occur in disorders such as hemochromatosis (iron), ochronosis (homogentisic acid), Wilson's disease (copper), gout (urate crystals), and calcium pyrophosphate dihydrate deposition disease, which result in the deposition of metabolic byproducts into the articular cartilage matrix.[142–147] Degeneration of the cartilage as a result of loss of its compliance, or chondrocyte injury, leads to osteoarthritis. Endocrine disorders, such as acromegaly, may also influence the quality of the cartilage.[32] Skeletal dysplasias may predispose the joint to increased susceptibility to mechanical forces (Fig. 18-6).[259,550]

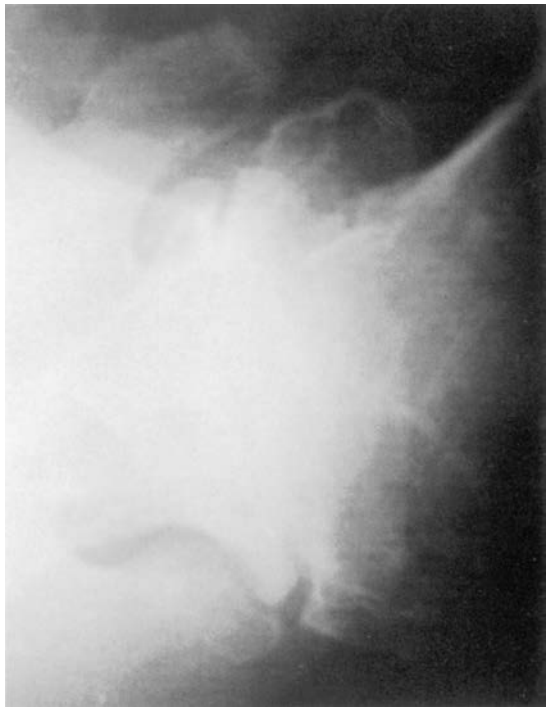


Figure 18-6 Osteoarthritis: Axillary lateral view with enlargement and flattening of the humeral head. There is concentric erosion of the glenoid.

At the same time certain catabolic activities within the joint are taking place in the cartilage, anabolic activities are under way, including angiogenesis.[35] Together these activities promote remodeling and hypertrophy of bone. Increased subchondral bony plate density or sclerosis is observed radiographically.[66] The tidemark advances with new bone formation in the basal layers of the calcified zone. Cartilage supported by this abnormally dense bone is exposed to more intense stresses, in itself contributing to the cartilage degradation. Degeneration of the cartilage decreases its capacity to distribute stress and causes higher peak stresses in the subchondral bone. Focal pressure necrosis of bone may occur.[333] In response to these changes in stress, additional bone is deposited.

Defects sometimes develop within the exposed subchondral bone plate. If there is excessive intraarticular pressure, fluid may be forced through these defects and into the subchondral marrow spaces.[277,424] Increasing accumulations of pressurized fluid, coupled with the secondary resorption of surrounding trabeculae, may result in the formation of cysts contiguous with the joint surface.[376] New bone formation may occur in areas that are loaded. The osteophytes enlarge the joint surface and may be covered by newly formed hyaline and fibrocartilaginous cartilage.[39,50] Intraarticular bodies, formed from fragmentation of surface cartilage or bone, may float in the joint or become sessile, maintaining potential for enlargement.[334]

Pathoanatomy

Damage to the joint and reaction to it may result in an early, subclinical stage of arthritis that may exist for years or decades. The degeneration takes the form of thinning and softening of the articular cartilage. Surface fibrillation and, in the deeper layers, fissures and vertical clefts develop as the cartilage succumbs, exposing subchondral bone. The classification of Outerbridge, initially proposed for chondromalacia of the knee, has been modified recently for the staging of articular cartilage lesions of the glenohumeral joint (viewed arthroscopically).[380,532] Stage I is softening or blistering of the cartilage. Stage II is fissuring and fibrillation. Stage III is deep ulceration, and stage IV is exposed subchondral bone. Its limitation is that stages II and III do not take into account accurately the depth of the lesion, although it is implied. There may be evidence of fibrocartilaginous repair.

The synovium thickens in response to the joint debris produced by the reactive changes in the articular cartilage and bone. Villous hypertrophy with random synovial cell hyperplasia may characterize the membrane. It may be filled with cartilage fragments and foreign body giant cells.[50] Loose or sequestered osteocartilaginous bodies may be seen.[50] The subsynovial region stroma may be filled with a mild chronic inflammatory reaction.[157]

The gross pathologic findings of glenohumeral osteoarthritis are fairly consistent and have been characterized by Neer.[356,357,361] Thinning or absence of the cartilage of the humeral head is most pronounced in a position corresponding to 60 to 90 degrees of abduction, the area of maximum joint reaction force.[217] The normally convex humeral head flattens. The exposed bone becomes eburneous and sclerotic, acquiring a marble-like appearance, often stippled with small reddish-brown vascular proliferations and fibrocartilaginous plugs (Fig. 18-7). Osteophytes appear circumferentially at the margin of the articular surface of the humeral head, resulting in its apparent enlargement (Fig. 18-6). The large inferomedial osteophytes, which have been termed "the goat's beard" by Matsen, may envelope and tension the adjacent capsule, contributing to limitations of external rotation (Fig. 18-7).[309] Subchondral cysts are often present on the glenoid as well as the humeral head.

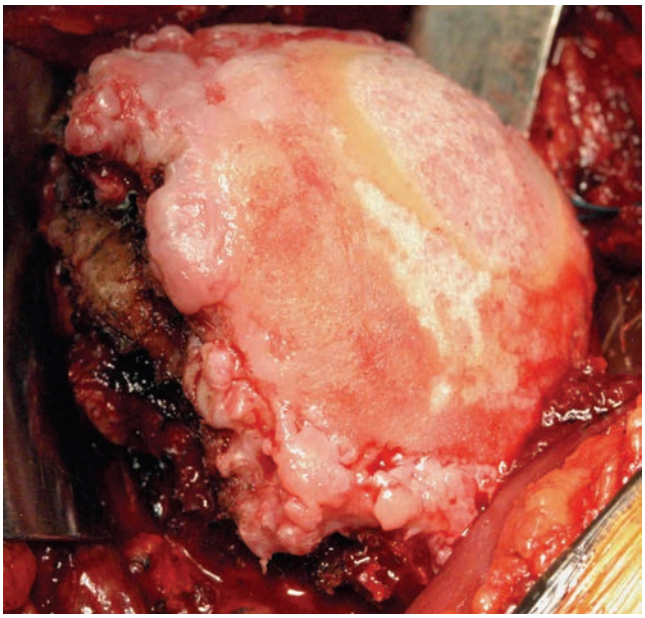


Figure 18-7 Osteoarthritis: Humeral head at the time of total shoulder arthroplasty. Flattening and enlargement of the humeral head is seen. Large proliferative osteophytes are noted circumferentially with predominance inferomedially, the "goat's beard." Punctate cystic lesions dot the eburneous bone.

The rotator cuff and the biceps tendon long head are intact in 87% to 95% of cases.[72,73,356,362,527] Neer believed that their integrity and capability to generate essential glenohumeral compressive forces are a prerequisite for the development of osteoarthritis.[362] He further believed that an enlarged osteoarthritic head helps prevent upward migration, resulting in fewer impingement-type rotator cuff tears.[362] Rupture of the biceps tendon long head can rarely occur, but the mechanism is in response to the presence of spurs in the intertubercular groove and not by the process of impingement against the coracoacromial arch.[362] The subacromial bursa has been noted to be thickened in the absence of acromial pathology in a high percentage of patients.[532] During arthroscopic evaluation and treatment of glenohumeral arthritis, the incidence of a concomitant lesion requiring treatment was 47%.[56]

Although primary glenohumeral osteoarthritis usually begins on the glenoid, the glenoid cartilage is typically spared anteriorly (Fig. 18-8).[416] The wear is more pronounced posteriorly, and a true crista (Fig.18-9) may be formed as a demarcation between intact articular cartilage and exposed subchondral bone. Disease progression favors excessive glenoid bone erosion posteriorly with resultant posterior humeral head subluxation. This stretches and attenuates the posterior capsule while the anterior capsule

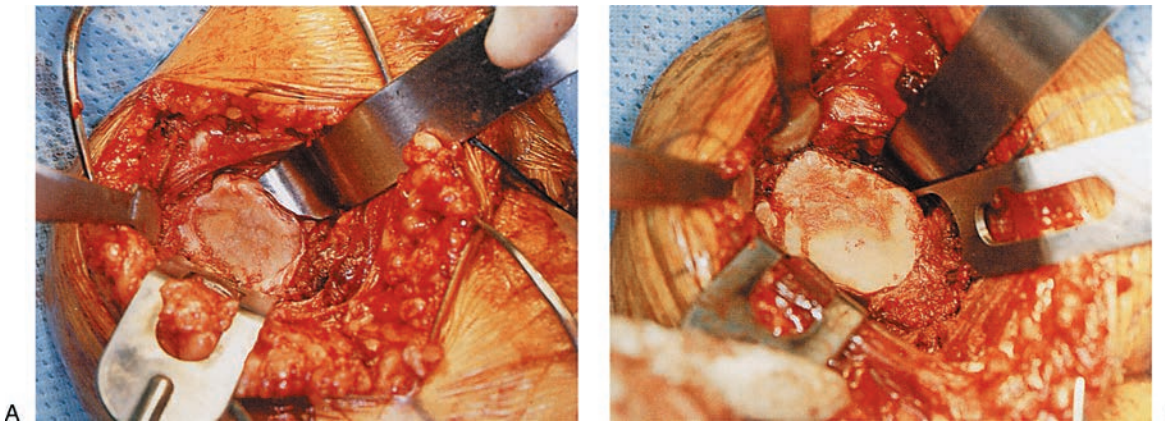


Figure 18-8 Osteoarthritis: Glenoids at the time of total shoulder arthroplasty demonstrating **(A)** demarcation between intact anterior cartilage and posterior eburneous subchondral bone. Peripheral osteophytes are seen. **(B)** Significant circumferential osteophyte formation.

significantly thickens and contracts. Peripheral osteophytes may "enlarge" the glenoid (Figs. 18-8B and 18-10). Osteocartilaginous bodies, either loose or attached, seek the recesses of the synovial cavity, especially the subscapularis bursa (Fig. 18-11). They may enlarge and become intimately attached to the adjacent bony structures, significantly distorting the normal anatomy. The soft tissue envelope tightens further as it drapes over the osteocartilaginous bodies and osteophytes. A morphologic study of the glenoid in osteoarthritis has enabled the classification of a well-centered humeral head, a posteriorly subluxed humeral head, and a retroverted, primarily dysplastic glenoid.[119,526,527]

The same findings are observed in secondary osteoarthritis. In addition, in cases of ochronosis, the characteristic blackening of the articular cartilage caused by homogentisic acid deposition is noted.

Clinical Evaluation

Patients may present for evaluation and treatment as early as age 40 to 50. Their lives are clearly altered by the disease process manifesting primary complaints of shoulder pain and loss of range of motion.[146,308] The source of pain may be local, peripheral, or central in origin.[107] Joints effusions, bone marrow lesions (edema), synovial hypertrophy, tendonitis, and bursitis are nocioreceptive structures shown to be involved in osteoarthritis.[132] More than 50% of patients are unable to sleep on the affected side and cannot perform common functional tasks.[314] Unremitting progression of the disease process will lead to more intense symptoms, failure of conservative management measures, and consideration for surgical treatment. The gender distribution is equal for this active and generally healthy subset of patients.[362] The dominant arm is involved to a greater extent than the nondominant arm.

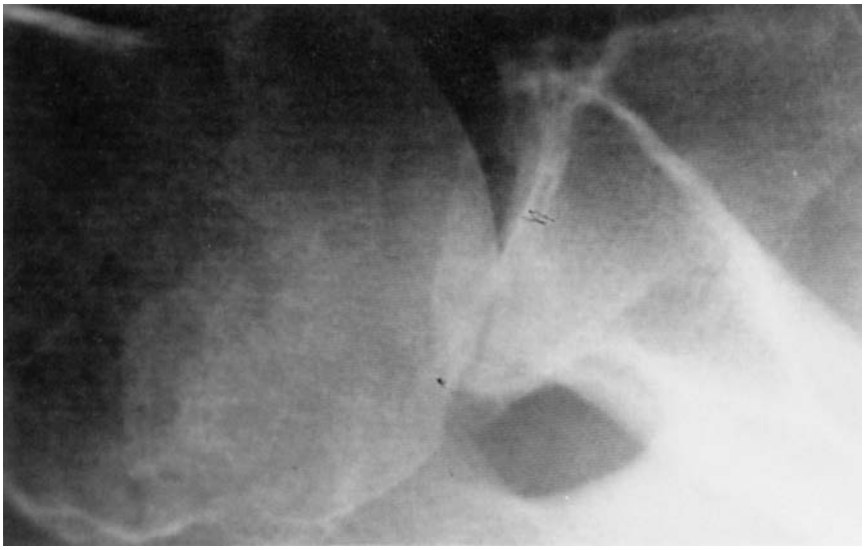


Figure 18-9 Osteoarthritis: Axillary lateral view demonstrating preservation of the anterior cartilage, posterior subluxation of the humeral head, and preferential wear of the posterior aspect of the glenoid.

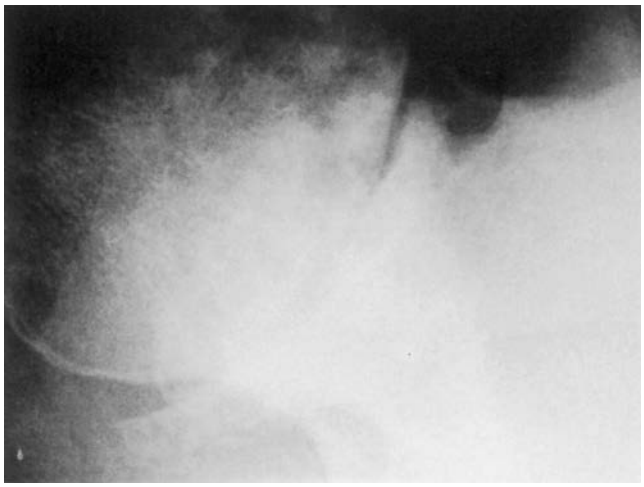


Figure 18-10 Osteoarthritis: Enlargement of the glenoid without evidence of significant preferential posterior wear.

Generalized atrophy of the shoulder is often notable, especially when the disorder is unilateral. Early in the disease, motion changes are minimal and difficult to detect, unless the most sensitive testing position—supine—is used. As the glenohumeral changes become more advanced, motion of the soft tissues is restricted, with a significant diminution of external rotation (Fig. 18-12). This is a more sensitive test of intraarticular activity than is the loss of elevation. A specific point of localized tenderness is over the posterior joint line, more easily elicited as the humeral head becomes subluxed with advancing disease. Attempts at active and passive motion are painful and may produce catching and squeaking sounds from the rough glenohumeral articulation. Synovial thickening and a large joint effusion may be palpable in thin individuals.

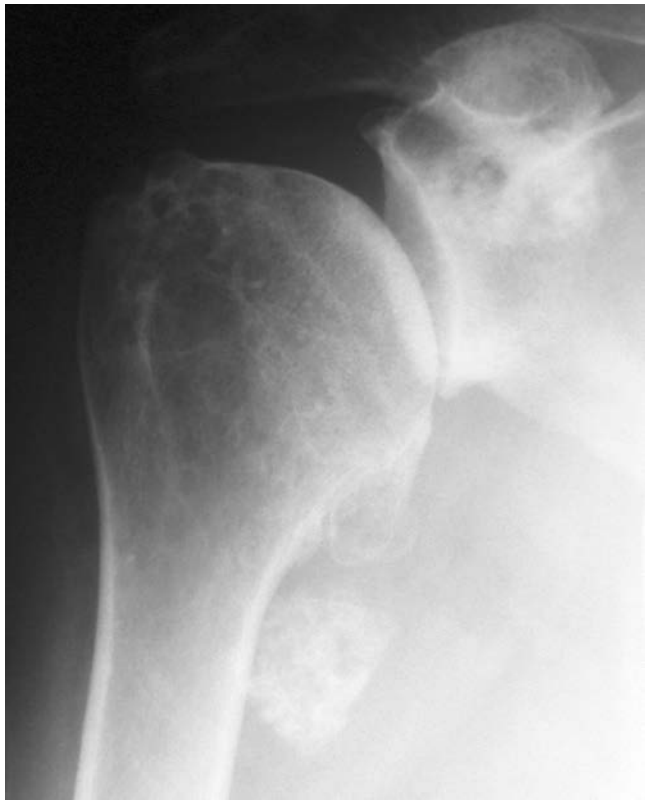


Figure 18-11 Osteoarthritis: Osteocartilaginous bodies are seen in the subscapularis bursa and in an unusually exaggerated inferior recess of the glenohumeral joint.

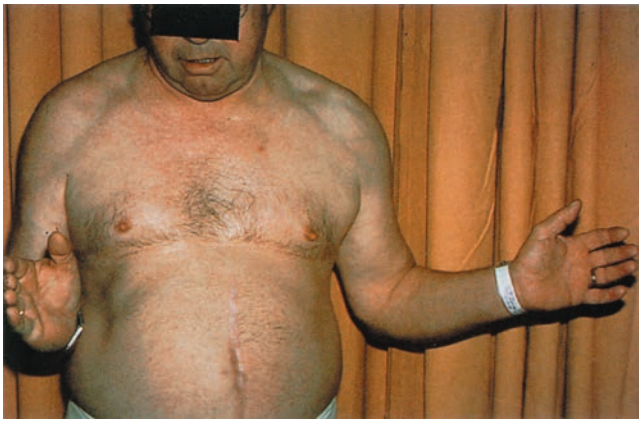


Figure 18-12 Osteoarthritis: Significant loss of external rotation is seen before total shoulder arthroplasty.

Imaging

Plain films are most helpful for making the diagnosis of osteoarthritis, but probably underestimate the extent of pathologic osteoarticular changes.[416] The AP view of the humeral articulation to the scapular plane and a high-quality axillary lateral view are all that is needed. They enable one to ascertain joint orientation, the amount of erosion of the glenoid, humeral head position, and the extent of disease activity. Weighted views may prove useful to help identify reduction of the glenohumeral cartilage space by as little as 1 mm.[514] Because of soft tissue contractures or pain, optimum films are sometimes not possible to obtain. In those instances, a CT scan will prove extremely useful in assessing glenoid morphology, glenohumeral relations, the presence of osteocartilaginous bodies, and the determination of the volume of the glenoid vault (Fig. 18-13).[20] Other findings of osteoarthritis are densification of subchondral bone, subchondral cysts, and peripheral osteophytes along the glenoid margins and adjacent to the articular surface of the humeral head, especially inferiorly. The humeral head may be flattened and enlarged. A triple-phase bone scan may prove beneficial when the plain films show early changes of glenohumeral arthritis (Fig. 18-14).

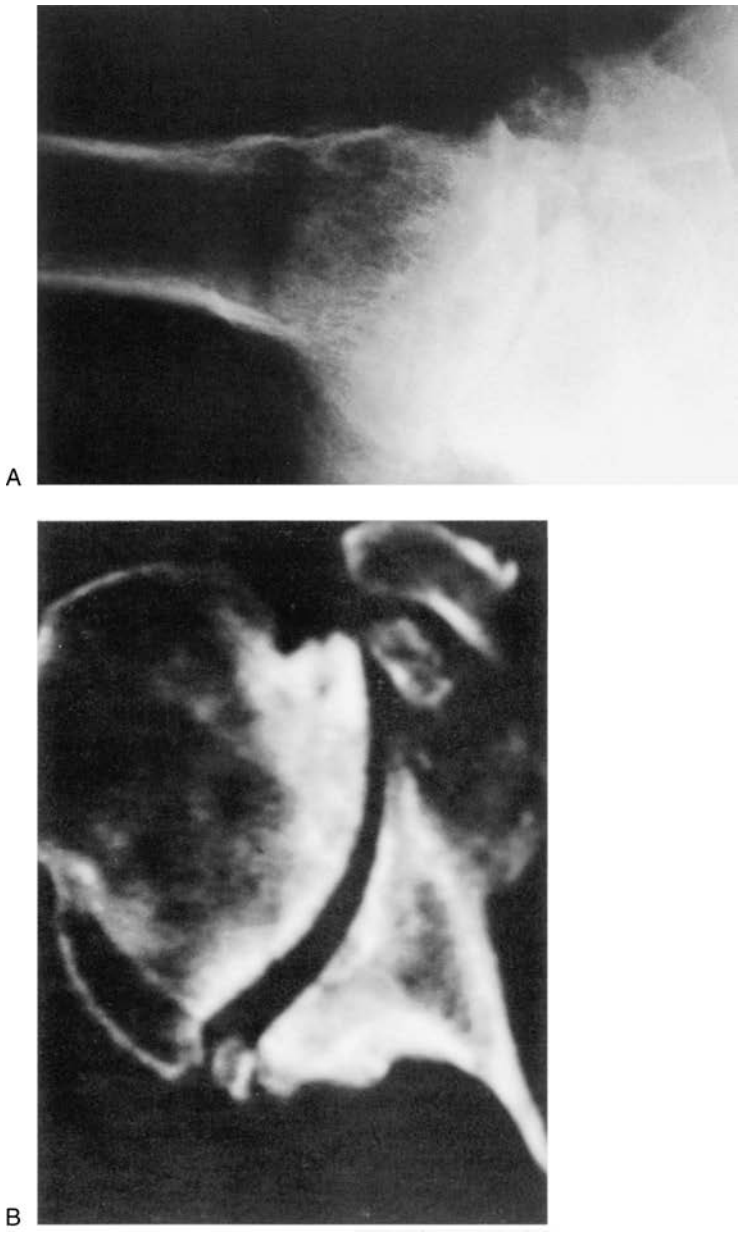


Figure 18-13 **(A)** Osteoarthritis: Findings on axillary lateral view may fail to define the pathoanatomy with accuracy. **(B)** Significant deformity noted on computed tomography scan. Loose bodies have attached to the anterior glenoid. External rotation limitation can be appreciated, due to contact of the posterior humeral head and osteophyte against the glenoid.

Laboratory

Laboratory studies are rarely helpful in the evaluation of primary osteoarthritis. However, investigators are in pursuit of promising identifiable disease markers such as COMP, antigenic keratin sulphate, hyaluronic acid, YKL-40, type III collagen N-propeptide, and urinary glucosyl-galactosyl pyridinoline.[409] Blood studies and synovial fluid analysis may help identify an underlying cause of secondary osteoarthritis. Synovial fluid levels of biochemical markers (aggrecan-aggregates) of catabolic activity within the articular cartilage enable the detection of all stages of glenohumeral osteoarthritis, with a high degree of accuracy.[416] The identification of the disorder before radiographic changes are present may prove extremely useful to expand treatment options and to monitor disease progression and response to treatment.[416]

Adjunctive Diagnostic Tests

Arthroscopy of the glenohumeral joint can improve the diagnostic accuracy when the clinical diagnosis is suspected and the radiographs do not show advanced stages.[121,416]

POSTTRAUMATIC ARTHRITIS

Injury and death of articular chondrocytes and the resiliency of the matrix macromolecular framework to withstand or recover from traumatic loading are fundamental

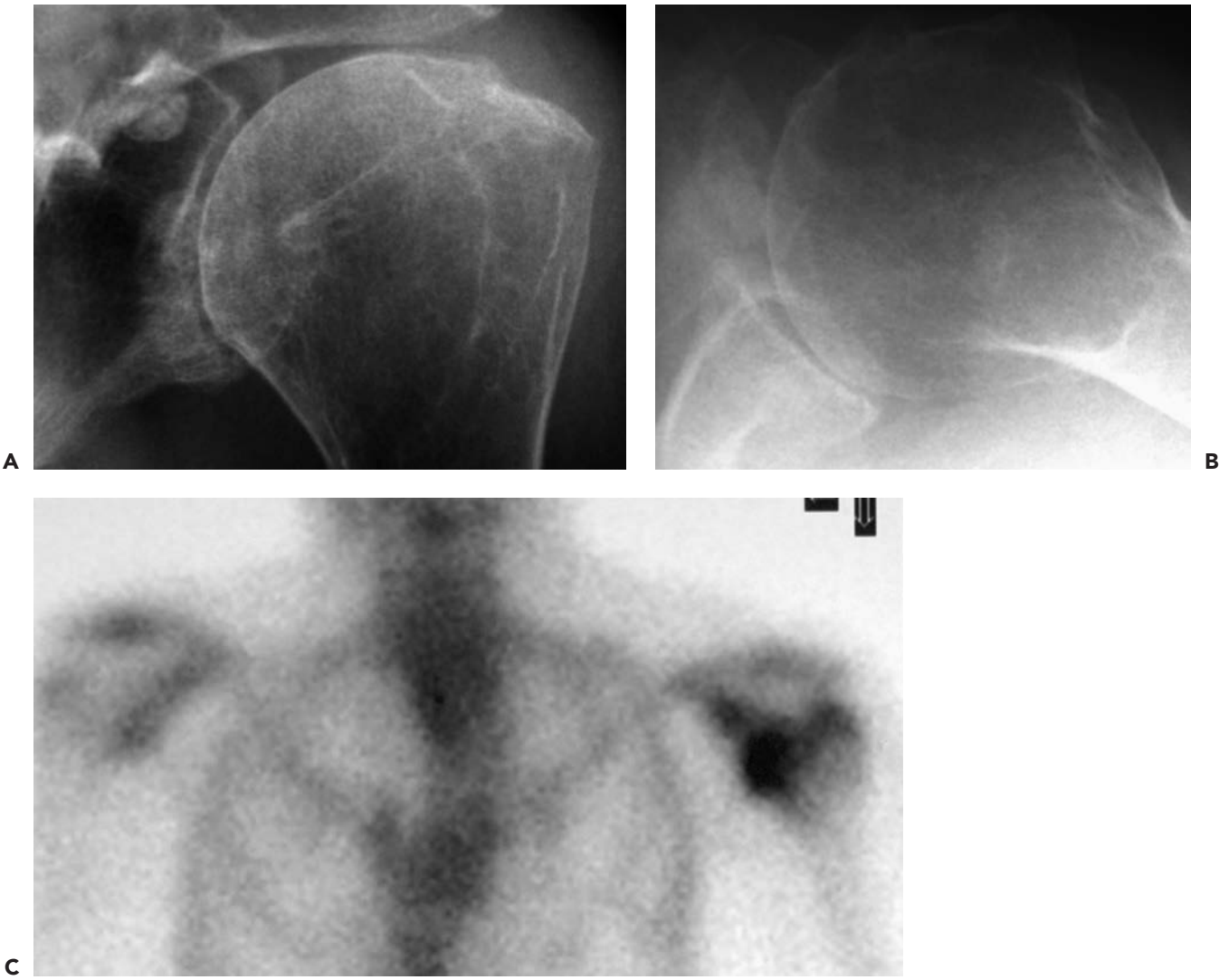


Figure 18-14 **(A,B)** Plain radiographs of mild glenohumeral osteoarthritis. **(C)** Delayed phase of bone scan with increased uptake of radionuclide in the glenohumeral joint.

elements in the pathogenesis of posttraumatic arthritis.[37,109,250,350,524] Major articular surface incongruities that exist at the completion of intraarticular fracture healing will inevitably lead to the deterioration of the joint quality (Fig. 18-15).[328,329] Tolerance of any joint to withstand minor variations in surface contact are dependent on many factors: the severity of the event of injury, surface geometry, surface area, specific load-bearing characteristics, and the integrity of associated joint-supporting soft tissues.[48] The discrepancy in the surface area of the humeral head and the glenoid is assurance that a small portion of the humeral head is in contact with the glenoid at any moment. It would appear, therefore, that a significant glenoid articular surface step-off or gap would have far greater influence on the development of posttraumatic arthritis than a corresponding one on the humeral side. Extraarticular fractures of the proximal humerus (surgical neck) may result in reorientation of the articular segment relative to the shaft or relative to the glenoid (Fig. 18-16). Up to 45 degrees of angulation are seemingly well tolerated without significant posttraumatic arthrosis or functional impairment.[358]

In many instances, the precise cause of posttraumatic osteoarthritis cannot be determined. In others, the cause is more apparent (Fig. 18-17). Varying combinations of joint incongruity from malunion, joint instability from adjacent soft tissue injuries, circulatory disturbance, and intraarticular fibrosis will have a bearing on the fate of the joint (Fig. 18-18). Zyto reported a 64% incidence of osteoarthritis in patients sustaining displaced four-part proximal humerus fractures and 25% for three-part fractures.[559] One of the more common forms of posttraumatic arthritis is that seen following glenohumeral instability.[57,207] This is further discussed in the next section.

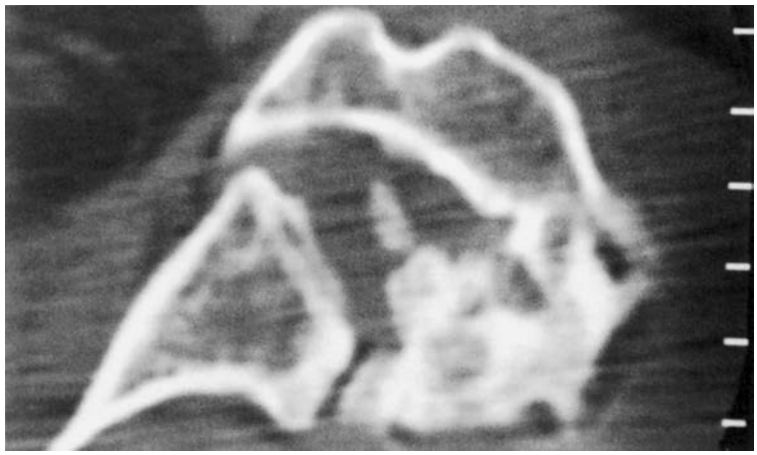


Figure 18-15 Posttraumatic osteoarthritis: Changes resulting from proximal humerus fracture, demonstrating joint incongruity and loss of humeral articular surface.

Chronic dislocations will result in disturbances of circulation and malnourishment of the articular cartilage surfaces (Fig. 18-19). The absence of stress to the underlying bone results in softening. Rapid deterioration of the joint is seen following closed and open reduction of these chronic dislocations, unless the surface abnormalities have been addressed.[191–193]

Proximal humerus fractures and their sequelae are given consideration in the section "Osteonecrosis." This may be the most common complication of proximal humerus fracture that will lead to posttraumatic arthritis. Proximal humerus nonunions may result in the formation of fibrous ankylosis of the glenohumeral joint, predisposing to posttraumatic arthritis.

ARTHRITIS OF DISLOCATION

Definition

Glenohumeral joint dislocations occur more frequently than any other major joint dislocation. Acute complications related to the initial dislocation include fracture, musculotendinous rupture, neurologic injury, vascular interruption, and recurrent glenohumeral instability. A late debilitating sequela to glenohumeral instability and its surgical treatment was, in one form, first appreciated by Hindmarsh and, in another, initially observed by Neer.[199,355] The term "dislocation arthropathy" spawned and included those occurrences of glenohumeral arthritis with a well-documented

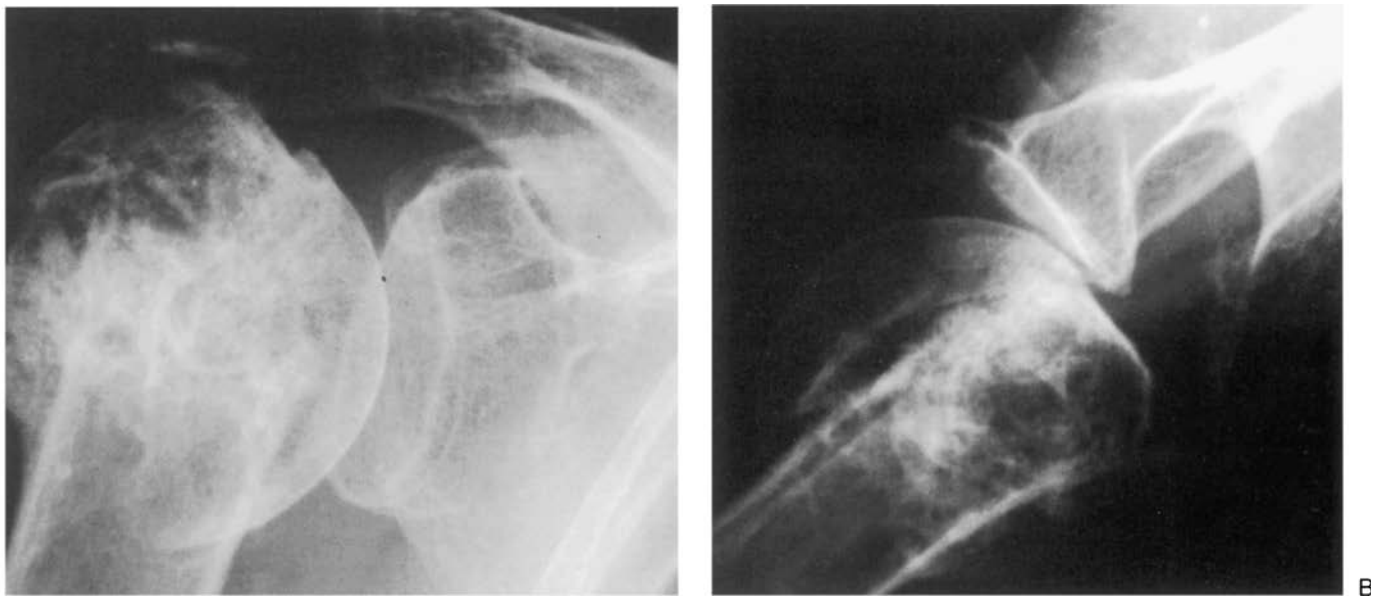
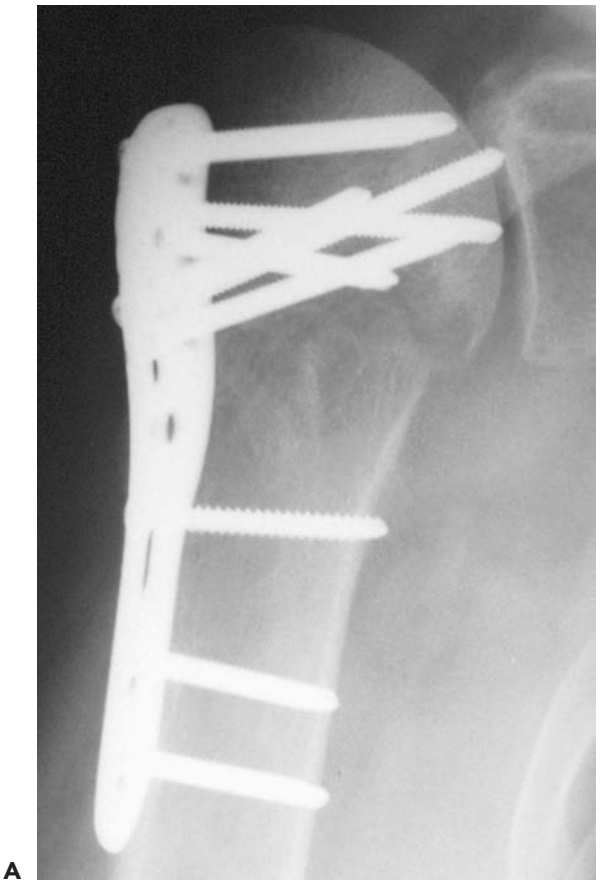


Figure 18-16 **(A,B)** Posttraumatic arthritis. Proximal humeral malunion with distortion of proximal humeral anatomy. Early arthritic changes are observed. Dysvascular changes are not present in the articular segment.



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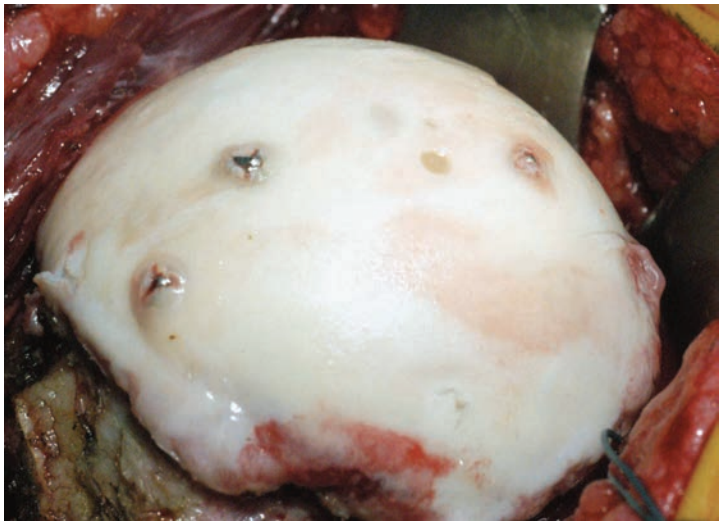
B



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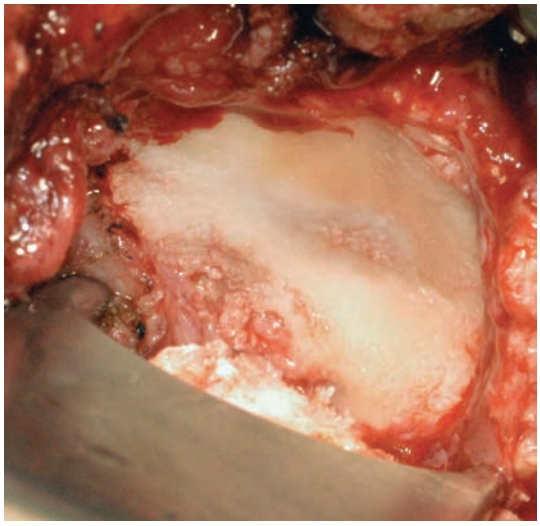


D

Figure 18-17 **(A,B)** Proximal humeral internal fixation screws violate the subchondral bone of the humeral head. **(C)** The thickness of the humeral head cartilage is insufficient to prevent the tips of the screws from projecting "proud" into the glenohumeral joint. **(D)** In this instance, the cause of posttraumatic osteoarthritis is obvious: the destruction of glenoid and humeral head articular cartilage by screws that were too long.

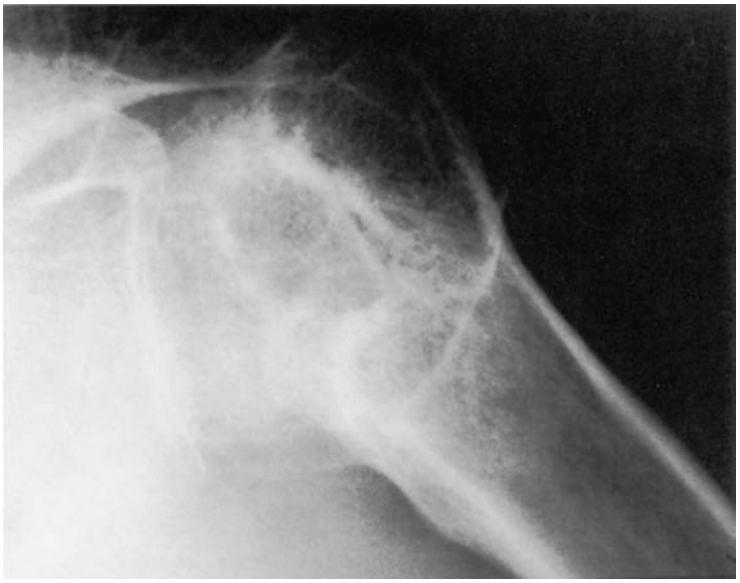


Figure 18-18 Posttraumatic arthritis: Malunited proximal humerus fracture leading to advanced disease.

history of glenohumeral dislocation, often in patients having undergone surgical treatment (Fig. 18-20).[356,445]

Incidence

The incidence of this disorder has not been accurately established. Hovelius et al. performed a 10-year prospective study of patients younger than the age of 40 to assess the outcome of a primary anterior dislocation of the shoulder treated with closed reduction, both with and without immobilization.[207] The incidence of glenohumeral arthrosis was 20%. The degree of involvement was mild in 11% and severe in 9%. Cameron et al. determined the overall prevalence of osteoarthritis after either acute or chronic glenohumeral instability to be small, if not rare.[57] It was not influenced by the direction of the instability. They noted that the risk of osteoarthritis increased with the time from injury. In other series of patients treated surgically for anterior glenohumeral instability, the incidence of preoperative arthritis has ranged from 0% to 20%.[197–200]

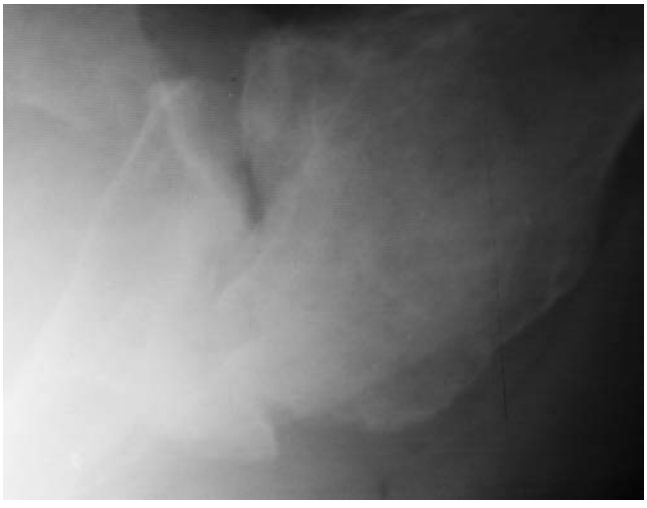


Figure 18-19 Posttraumatic arthritis: Chronic locked posterior dislocation with gross incongruity of the glenohumeral joint.

In the large series (570 patients) reviewed by Buscayret et al., as well as earlier series, the factors of age at the onset of instability, osseous glenoid rim lesions, humeral head impaction fractures, and interval between the onset of instability and surgical treatment correlated with the preoperative development of arthritis.[53,209,445,473] Marx et al. noted that glenohumeral dislocation requiring reduction was found to be associated with a 10- to 20-fold increase in the risk for the subsequent development of severe arthrosis sufficiently severe to warrant shoulder arthroplasty.[307]

By far the most common cause of arthritis of dislocation is iatrogenic. It has been termed "capsulorraphy arthropathy" by Matsen et al.[310] With rare exception, its existence is acknowledged by most experts in the field of shoulder surgery, although its incidence cannot be accurately determined.[315] In series with greater than 10 years follow-up, the incidence of significant arthritis ranges as high as 72%.[8,210,261,433,439,516] Interestingly, in patients with no preoperative arthritis, at a mean 6.5 years after surgical treatment, postoperative arthritis occurred in nearly 20% of patients.[53] Once again, age at the time of the initial instability episode seemed to be most important. Other important factors were age at the time of surgery and a longer interval of follow-up. In contrast to Rachbauer et al., the presence of arthritis was influenced by the number of instability episodes prior to surgery.[410] Findings regarding decreased external rotation were inconclusive. With consideration of these observations, Buscayret suggested that

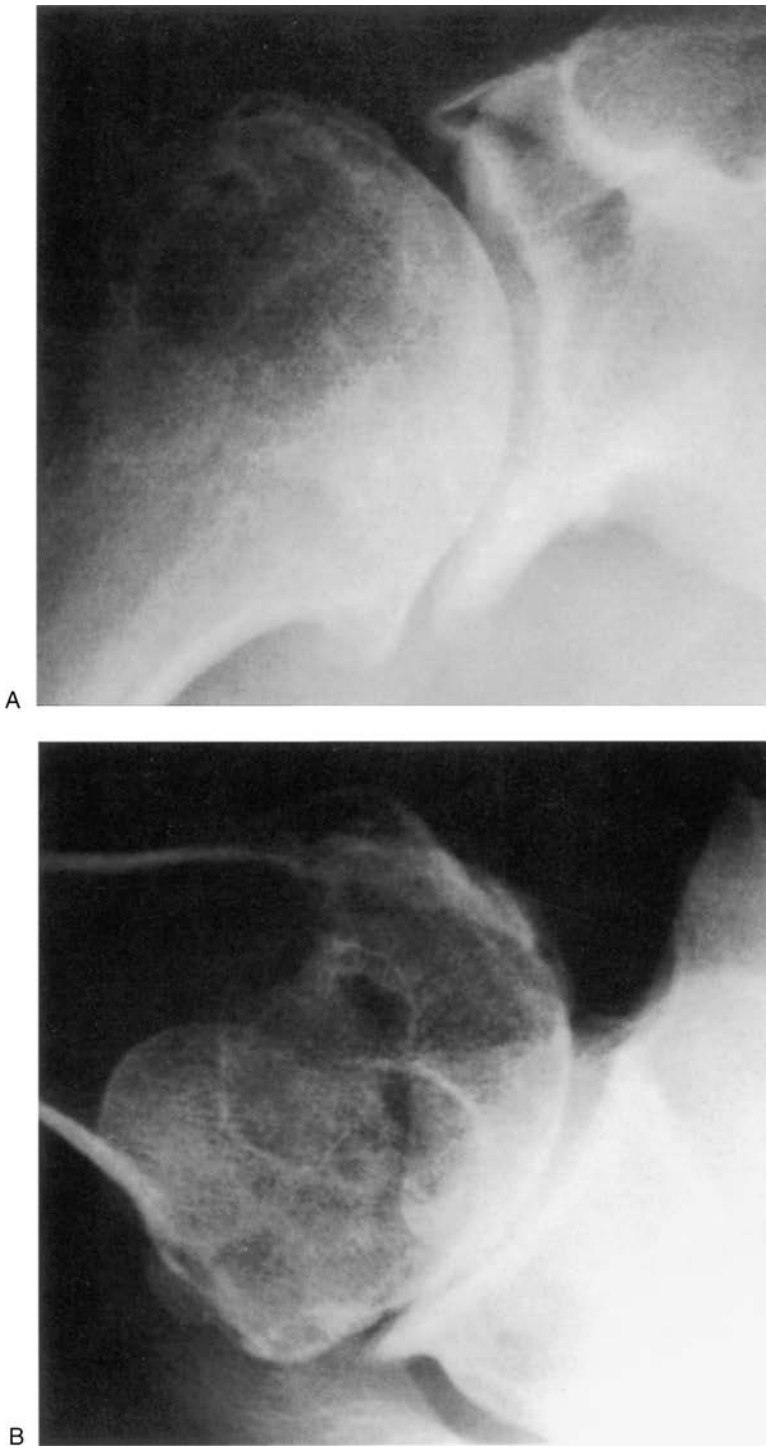


Figure 18-20 **(A,B)** Arthritis of dislocation: The sequelae to two previous surgeries including anterior stabilization and subsequent staple removal.

surgery does not influence the risk factors for the development of arthritis in shoulders that undergo attempts at surgical stabilization.[53]

Pathogenesis

Hovelius' data suggested no relation between dislocation arthropathy and the number of recurrent dislocations or the treatment rendered either operative or nonoperative.[207] The lack of association with the number of dislocations was confirmed by others.[53,307] Hovelius' study pointed out further that advanced glenohumeral arthritis may occur even after a single anterior glenohumeral dislocation treated without surgery.[207] Traumatic shoulder dislocations are the result of a significant force applied to the glenohumeral joint and generally involve damage to soft tissue

including the capsule, labrum, and rotator cuff, as well as bone and cartilage.[498]

Morrey and Janes prudently observed that looseness of the joint played a role in the development of surgical failures after the performance of a unidirectional repair.[343] Factors shared by these patients included a positive family history, occurrence of bilateral glenohumeral instability, and the coexistence of posterior instability in the shoulder that was operated on. Neer introduced the term "multidirectional instability," perhaps for the same subset of patients.[355] He was also the first to draw the association between the disorder and arthritis of dislocation.[356] It was his belief that multidirectional instability existed on the basis of lax ligaments, repeated minor injuries, or a combination of both. The most frequent cause of multidirectional instability was felt to be an acquired laxity, explained on a heritable basis. The initial instability episode in this group of patients most often occurred without high forces, major injury, or significant associated injuries. Typically, if a dislocation was present, it reduced spontaneously or required minimal, usually self-manipulative, effort. Generalized ligamentous laxity was often recognized. Examination of the contralateral shoulder revealed excessive anterior and posterior translations, as well as inferior translation, as evidenced by the "sulcus sign." The presence of this so-called sulcus sign was pathognomonic for multidirectional laxity. Neer concluded that "by far the most frequent etiologic mechanism for the development of arthritis of recurrent dislocation is a 'standard' operative procedure intended to remedy recurrent unidirectional dislocations that is unsuspectingly performed on a loose, multidirectional shoulder."[363] He observed that the "procedures displace(d) the humeral head in a loose shoulder away from the side of the repair, creating a fixed subluxation. The subluxed head wears unevenly on the glenoid, and arthritic changes can develop surprisingly fast."[363]

It was actually Hindmarsh, however, who first implicated the role of stabilization surgery in the production of moderate to severe glenohumeral arthritis.[199] A variety of surgical procedures performed in an attempt to stabilize the glenohumeral joint and prevent recurrences led to glenohumeral arthritis.[188,189,214,297,299,356,369,373,487,536] When a unidirectional repair, such as the Putti-Platt,[208,254,261,410] Eden-Hybinette-Lange,[46,53,536] Magnuson-Stack,[445] Bristow-Laterjet,[8,206,210,295,369,473] DuToit,[373,445,474,558] or Bankart[208,210,369,445] was performed to eliminate instability recurrences, the stage was set for further glenohumeral joint deterioration. The incidence of osteoarthritis was notably less with anatomic repairs (Bankart) when compared with nonanatomic repairs.[433,439]

Pathophysiology

The more widespread recognition of this late sequela to instability reconstruction has led to hypotheses about its mechanism. When unidirectional surgery is imposed on a normal joint, or more often, a multidirectional lax glenohumeral joint, glenohumeral biomechanics are altered.[355,533] This occurrence has been assigned predominant responsibility for the development of the arthritis of dislocation.[41] Even in the presence of normal laxity, a unidirectional repair, resulting in excessive tightening of the soft tissues, forces the humeral head in the direction opposite that of the repair.[183,189] When this is performed for anterior instability, there is a significant limitation of external rotation. As external rotation is performed, contact compression and shear forces at the glenohumeral articulation increase. This results in translation of the humeral head posteriorly, with a shift in the center of rotation. Eccentric loading forces are concentrated on a smaller glenoid surface area with deterioration in the quality and quantity of articular cartilage.[138,481] Over the time period dating from the index procedure, ranging from a few months to more than one-half century, there is a progressive development of glenohumeral degenerative changes.[315,483,516] Those procedures that transpose bone to create a blockade carry the additional risk of humeral articular surface scraping against the bone or metal hardware fixation devices.[19,206,295,536,555]

Samilson and Preito noted that the occurrence of arthritis was much more common after posterior glenohumeral dislocation (Fig. 18-21).[445] This was thought to be because of the delay in diagnosis and treatment of posterior glenohumeral dislocation, especially chronic occurrences. In Samilson and Preito's series, the number of dislocations did not determine the severity of glenohumeral

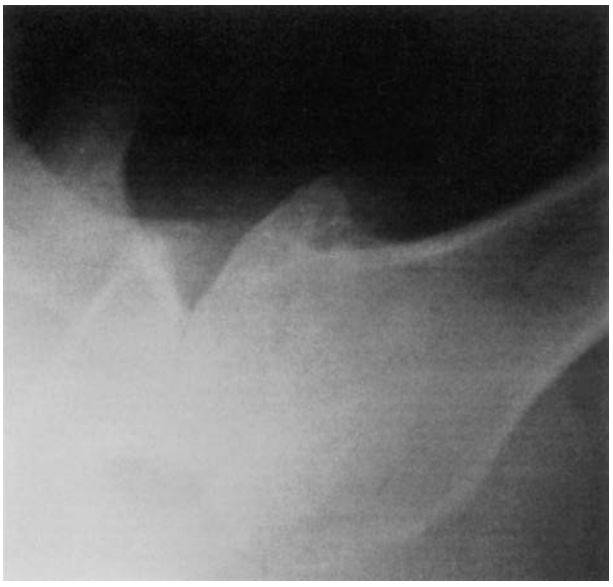


Figure 18-21 Arthritis of dislocation: The result of recurrent posterior glenohumeral instability. Note significant posterior humeral subluxation and eccentric wear of the posterior two-thirds of the glenoid.

joint arthrosis.[445] Of the group of patients with moderate to severe arthrosis, 71% had experienced only one glenohumeral instability event. In this same group of patients, 26% had recurrent instability. No correlation was recognized in the presence of a Hill-Sachs or bony Bankart lesion. Those patients who were slightly older at the time of their index glenohumeral instability event had more severe arthrosis. In Bigliani et al.'s study, 7 of 17 patients continued to have instability in the presence of arthropathy.[31]

The pathophysiologic process that leads to dislocation arthropathy after one or more glenohumeral dislocations is less clear. A more severe arthritis existing in patients with a single dislocation suggests a direct relation between the forces dissipated to the articular cartilage and the restraining soft tissues. In so-called "tight" shoulders, higher forces may be necessary for traumatic dislocation, whereas more lax shoulders require less force. The development of postdislocation arthrosis may result from recurrent episodes of instability because eccentric loading during pathologic translocation transmits greater shear and compression forces at the articular interface. Progressive bone loss in the anteroinferior quadrant, in addition to the loss of the labrum, was found to have the effect of causing further increases in mean contact pressures and peak pressures and a decrease in contact area across the glenohumeral joint.[166] It is possible that surface imperfections and impression fractures of the humeral head could further damage the articular cartilage with each successive dislocation event. The resultant accumulations of particulate joint debris and newly formed osteocartilaginous loose bodies could theoretically contribute a third body wear mechanism. These detrimental effects could coexist and become additive in their potential for joint destruction.

Pathoanatomy

The pathology encountered is often dependent on the index stabilization operation. In general, excessive scarring and adhesions are the predominant finding.[31] Operations involving greater manipulation of the soft tissues, such as the Bristow procedure, often result in encasement of all anterior soft tissue structures, sometimes including the axillary nerve.[31] The subcoracoid, subacromial, and subdeltoid planes of motion are usually scarred. The subscapularis musculotendinous unit will be contracted, as is the anterior joint capsule. Occasionally, the subscapularis integrity will be preserved, such as in a Magnuson-Stack procedure.[301]

Transferred bony blocks may overhang the lateral margin of the anterior inferior glenoid.[8]

The articular surface contours may appear normal with simply posterior subluxation and instability of the humeral head. More often, the glenoid articular surface involvement is quite significant (Fig. 18-22). The anterior glenoid cartilage may be reasonably preserved, whereas the more posterior cartilage demonstrates thinning and erosion to subchondral bone. In advanced cases, posterior glenoid bone erosion and loss is observed. The humeral head shows concomitant wear changes. The penetration of metallic fixation devices into the intraarticular space may be observed in open as well as arthroscopic procedures, with associated articular surface changes, including arthropathy (Fig. 18-23).[19,121,235,373,425,470,555,558] Severe arthrosis is not usually present.

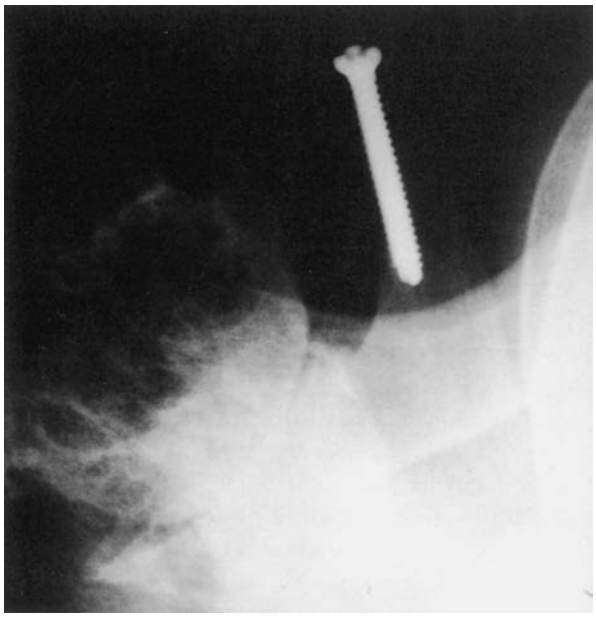


Figure 18-22 Arthritis of dislocation: Capsulorrhaphy arthropathy with significant deterioration of the glenohumeral articulation, total loss of joint space, and secondary changes within the humeral head.

When operative care has not been rendered, the findings are essentially those of glenohumeral osteoarthritis. Preferential glenoid wear or humeral head subluxation may be observed in the direction of the instability. Variable soft tissue changes may exist.

Clinical Evaluation

The patient, often younger than 40 years of age, usually presents to the surgeon for evaluation many years after the glenohumeral stabilization operation or the index dislocation occurrence.[31,42,189,356] Most of these patients have not had interval recurrences or symptoms. Within 2 to 3 years of presentation, shoulder symptoms have begun to evolve. At the time of the evaluation, the chief complaints are related to intense, often disabling pain. Almost all patients have had long-standing limitation of range of motion, especially external rotation.[483] Many develop an internal rotation contracture. Moderate to severe functional disability is present secondary to pain

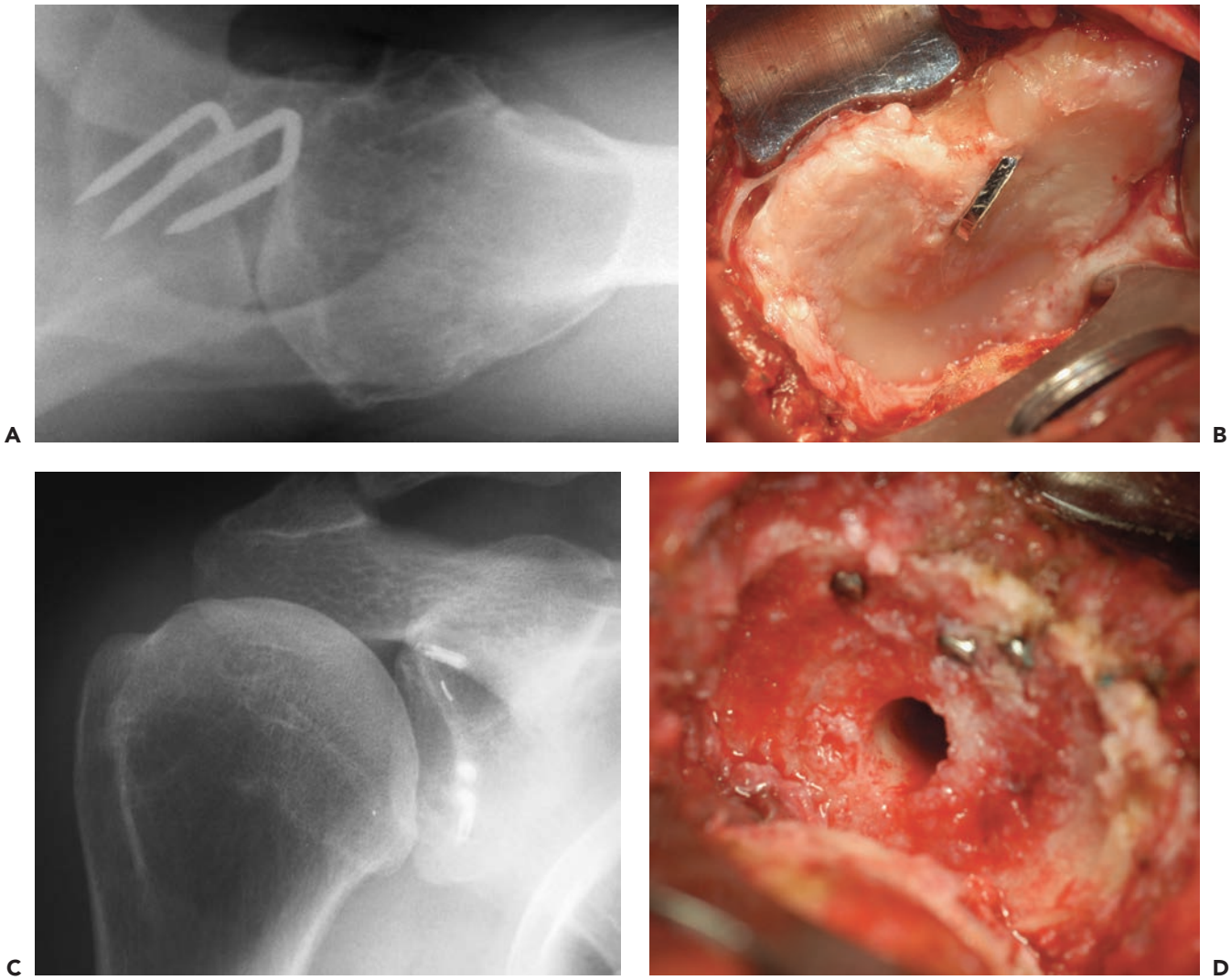


Figure 18-23 **(A,B)** Arthritis of dislocation: Advanced degenerative changes of the glenohumeral joint following prior anterior stabilization. Metallic staples are noted to be intraarticular with secondary effect upon the humeral head. **(C,D)** Arthroscopically placed metallic suture anchors in violation of the glenohumeral joint, necessitating glenohumeral arthroplasty.

and altered range of motion. Some have had multiple surgical procedures.

The physical examination of these patients is consistent. They are usually younger, healthy-appearing men, often muscular with the exception of the affected shoulder. One or more scars may be present, frequently hypertrophic. For those who have undergone anterior reconstructions, the coracoid is often prominent. When viewed from overhead, the prominence of the posteriorly subluxated humeral head may produce posterior fullness. Examination from the side may demonstrate a posterior attitude to the arm as it hangs at the side. Focal tenderness is often present at the posterior joint line. Active and passive glenohumeral joint motions produce crepitation. Examination of the contralateral shoulder will often demonstrate signs of multidirectional laxity including a positive jerk test, positive drawer testing, and a positive sulcus sign. Limitation of external rotation of the symptomatic shoulder is often pronounced; the more advanced the arthrosis is, the more significant the loss of external rotation is.[164] Samilson noted 62 degrees of external rotation with mild involvement, 23 degrees of external rotation with moderate involvement, and 14 degrees of external rotation with severe involvement.[445] This compared with 72 degrees on the contralateral side. Hawkins observed an average of –5 degrees of external rotation and Bigliani identified 17 patients with an average external rotation of –2 degrees.[31,189]

Imaging

Conventional imaging studies that include a true AP view of the glenohumeral joint and an axillary lateral view are

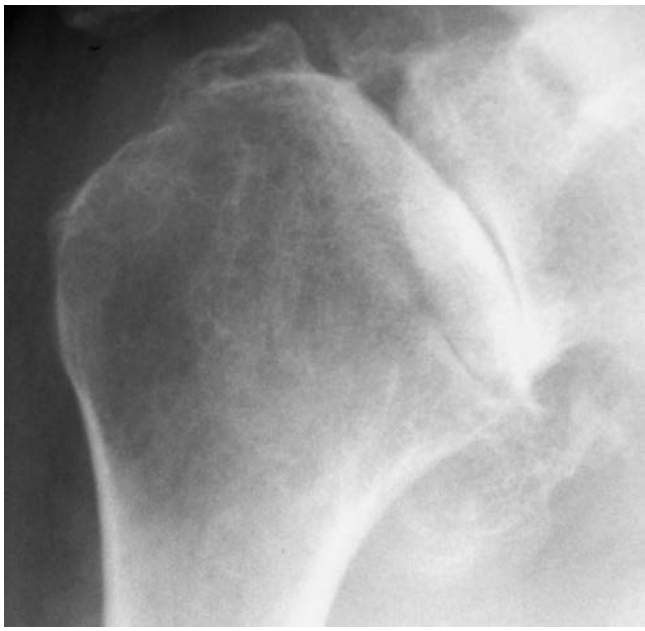


Figure 18-24 Arthritis of dislocation: Grossly altered glenohumeral articulation with large osteophyte formation, obliteration of the joint space, and humeral head flattening.

often all that are needed. The CT scan is helpful when a high-quality axillary lateral cannot be obtained; MRI examination is unnecessary.

The humeral head is usually posteriorly subluxed, with eccentric wear of the glenohumeral joint cartilage. Posterior glenoid erosion may be extreme.[164] The additional findings of glenohumeral osteoarthritis are often severe. Signs of previous surgery also include the presence of periarticular and, sometimes, intraarticular metal fixation devices (Fig. 18-24). Osteopenia from disuse may be present.

Laboratory

Specific laboratory investigation is not necessary.

Clinical Significance

A single common cause does not exist for the arthritis of dislocation. Nonoperative treatment was successful in preventing recurrences in 52% of patients in Hovelius et al.'s study.[207] Nearly one-fourth of the patients with more than two recurrences "spontaneously" stabilized. Their study also showed that glenohumeral arthropathy can result from the trauma of a single dislocation without recurrences. The subset of patients treated nonoperatively was not exposed to the surgical risk factors predisposing to capsulorrhaphy arthropathy. When operative treatment for instability is rendered, additional risks of some degree are inevitably entailed. It is incumbent that the diagnosis of the extent and direction of instability be accurately established. Failure to recognize multidirectional instability and generalized ligamentous laxity can lead to a disastrous outcome when a unidirectional repair is performed, especially if the soft tissues are excessively shortened and remain so permanently. It is therefore important not to treat all glenohumeral instabilities with one operative procedure. The direction of instability must be determined and the appropriate side stabilized appropriately. The surgeon should be familiar with the complications related to the use of metal and avoid the circumstances that place the glenohumeral joint at high risk for penetration.[298,558] Arthroscopic evaluation and treatment of glenohumeral instability may traumatize the joint from direct damage to the articular surfaces with the scope, instrumentation, and fixation devices.

Natural History

With few exceptions, available data have not permitted prospective evaluation of consistently homogeneous groups of patients with glenohumeral instability.[207] It appears that an increased risk of arthritis is not seen in voluntary subluxation of the glenohumeral joint in children.[213].

The work of Edelson identified skeletal specimens with findings of instability.[118] Coexistent findings of diffuse arthritic changes about the glenohumeral articulation were not observed. The data did not support the concept of prophylactic stabilization to prevent arthritic changes from developing. Hovelius' study would lend support to this concept, because a number of shoulders, even after one or more recurrences, stabilized over time without surgery.[207] Inappropriate stabilization in these cases may have led to the unnecessary development of dislocation arthropathy.

The natural history of established arthritis of dislocation is the progressive development of severe wear and, ultimately, glenoid deficiency (Fig. 18-25). Prevention would have the greatest influence on this destructive entity of the glenohumeral joint. Accurate diagnosis of the unstable shoulder and, when necessary, appropriate surgical treatment are mandatory. Early recognition of the condition plays a role if the joint is to be surgically salvaged without implant arthroplasty.[297,299]

OSTEONECROSIS

Definition

Osteonecrosis, sometimes termed avascular necrosis or aseptic necrosis, is simply bone death, both the osteocytes

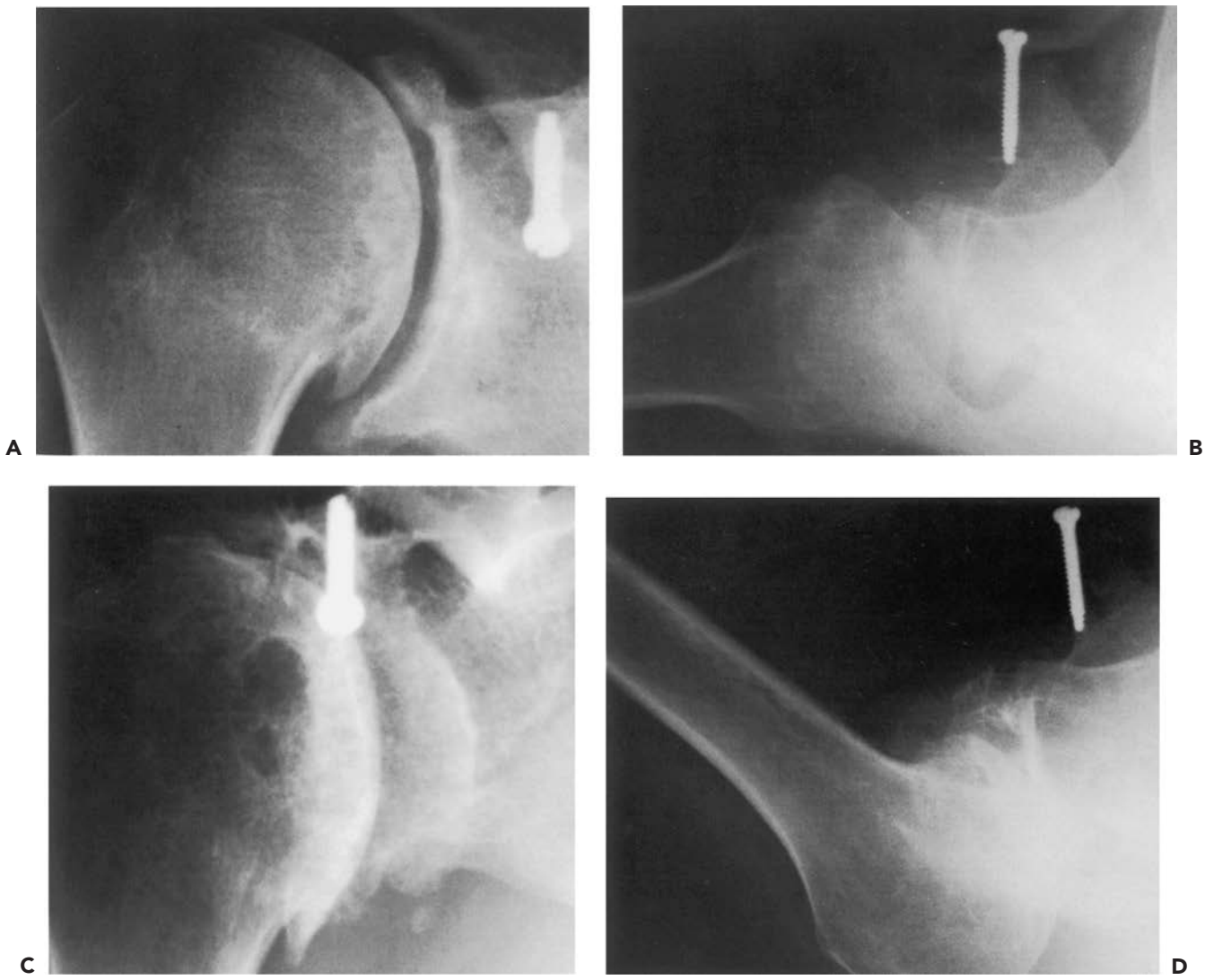


Figure 18-25 **(A,B)** Arthritis of dislocation: Ten-year interval following anterior stabilization procedure with moderate glenohumeral arthritis. **(C,D)** Now 18-year interval depicting further progression, with the formation of increased subchondral density, subchondral cysts, osteophytes, and loose bodies. Significant posterior wear and subluxation are seen.

and the marrow contents. However, it is the bone's failure of repair that leads to destructive changes within the joint. The earliest reports of implant arthroplasty for the shoulder include patients with osteonecrosis after proximal humerus fracture or dislocations.[268,353]

Osteonecrosis of the humeral head is best classified as traumatic or atraumatic. Fractures of the proximal humerus or fracture dislocations involving the glenohumeral joint place the articular segment of the proximal humerus at risk for osteonecrosis. The atraumatic category includes those occurrences of osteonecrosis that result from disease processes, highlighted in one of the earliest reports on the subject by Cruess.[87]

Incidence

The incidence of posttraumatic osteonecrosis has been difficult to determine. Factors including the severity of the fracture, the amount of displacement, and, to some extent, the type of treatment rendered will have influence on the occurrence of posttraumatic osteonecrosis. The incidence of osteonecrosis is highest after three- and four-part fractures, but can also occur after certain two-part fractures and even open anterior dislocations.[114,140,165,173,258,267,284,381,490,491] Closed treatment of displaced three-part fractures has resulted in a 3% to 14% avascular necrosis rate.[173] In four-part fractures treated closed, the incidence has ranged

from 13% to 34%.[114,173,258,359,397,462,484,492,559] For any method of treatment, anatomic neck fracture occurrences approach 100%.[99,220,358,359]

It has been suggested that operative treatment carries a higher risk of osteonecrosis because of the wide surgical exposure necessary to provide for adequate internal fixation.[490] The incidence of avascular necrosis in three-part fractures that have been treated operatively ranges from 12% to 34%.[173,190,266,346,490] Four-part fractures treated with open reduction and internal fixation have a much higher rate of avascular necrosis and malunion.[173,221,266,267,359,381,449,484,490,492,496,534] Neer believed the incidence to be as high as 90%.[359]

Osteonecrosis of the humeral head has been described in an adolescent treated surgically for recurrent anterior glenohumeral instability.[386]

Although it can be associated with many different conditions, the incidence of atraumatic osteonecrosis cannot be determined.[92,184,294] It is probably the third or fourth most common indication for glenohumeral implant arthroplasty. From a group of over 1,000 patients managed for osteonecrosis of any joint, humeral head was 7%.[340] A high incidence of corticosteroid use (82%), hip involvement (81%), and bilateral disease (74%) was noted in the same cohort. For patients with humeral head involvement, L'Insalata et al. reported a 76% incidence of osteonecrosis at another site.[292]

TABLE 18-2
ETIOLOGY OF OSTEONECROSIS

Relationship clearly established
Traumatic
Proximal humerus fractures
Chronic glenohumeral dislocations
Nontraumatic
Dysbaric disorders
Gaucher's disease
Sickle cell
Radiation
Relation probable
Traumatic
Repetitive injury
Nontraumatic
Steroids
Alcohol ingestion
Tobacco
Cushing's disease
Lipid metabolism disorders
HIV infection
Fatty liver
Pancreatitis
Organ transplants (renal)
Systematic lupus erythematosus
Osteomalacia
Lymphoma
Cytotoxic drugs

Pathogenesis

Some common causes of osteonecrosis and their relation to the disorder are summarized in Table 18-2. Proximal humerus fractures and dislocations have most clearly been established to jeopardize the circulatory integrity of the humeral head.

Neer's classification of proximal humerus fractures and glenohumeral fracture dislocations gave better understanding of the pathomechanics and the prognosis following treatment of these injuries.[358] He cited the importance of the vascularity of the articular segment and the relation of the articular segment to other parts of the proximal humerus and to the glenoid.

When the proximal humerus fractures, the wider the displacement of the articular segment from the shaft or from the tuberosities is, the higher the incidence of osteonecrosis is. More than 80% of the proximal humerus fractures are minimally displaced, according to Neer's criteria, keeping them at low risk for osteonecrosis (Fig. 18-26).[360] The unusual and unexpected trauma to the humeral head received at the time of extracorporeal shock wave lithotripsy for calcifying tendonitis resulted in the developments of osteonecrosis of the humeral head.[115]

Disturbance of microcirculation can be the result of elevated intraosseous and interstitial pressures, such as with marrow hypertrophy, infiltration, or replacement.[92,215,429,448,497] Venous congestion as a mechanism has been popularized.[216] Although most of this information has come from studies of the femoral head, there is less knowledge of the specific occurrences within the humeral head.[339] Occlusion of the microcirculation may take place also by infiltration of hematopoietic marrow spaces with metabolic byproducts.

Thrombosis of vessels may occur in vascular disorders or coagulopathies. The process of intravascular coagulation is capable of initiating the cascade of events that result in osteonecrosis and may be triggered by various conditions.[154,155,230,231] Jones et al. assessed 45 patients with nontraumatic osteonecrosis for nine coagulation factors and identified a high incidence of thrombophilic and hypofibrinolytic coagulation abnormalities.[232] Nearly half the patients had abnormal concentrations of two or more factors. A hypercoagulable state has been suggested as a mechanism for osteonecrosis in HIV infection, as have alterations in lipid function.[18,45,151] Kubo et al. reported a possible relationship between type I congenital thrombophilic antithrombin III deficiency and multifocal osteonecrosis.[269] Embolization phenomena with vasocclusion occur in sickle cell disorders (sickled cells), caisson disease (nitrogen bubbles), and possibly microscopic lipid droplets.[67–69,93,136,226-229,239,335,464,543] Repetitive trauma may play a role.[245]

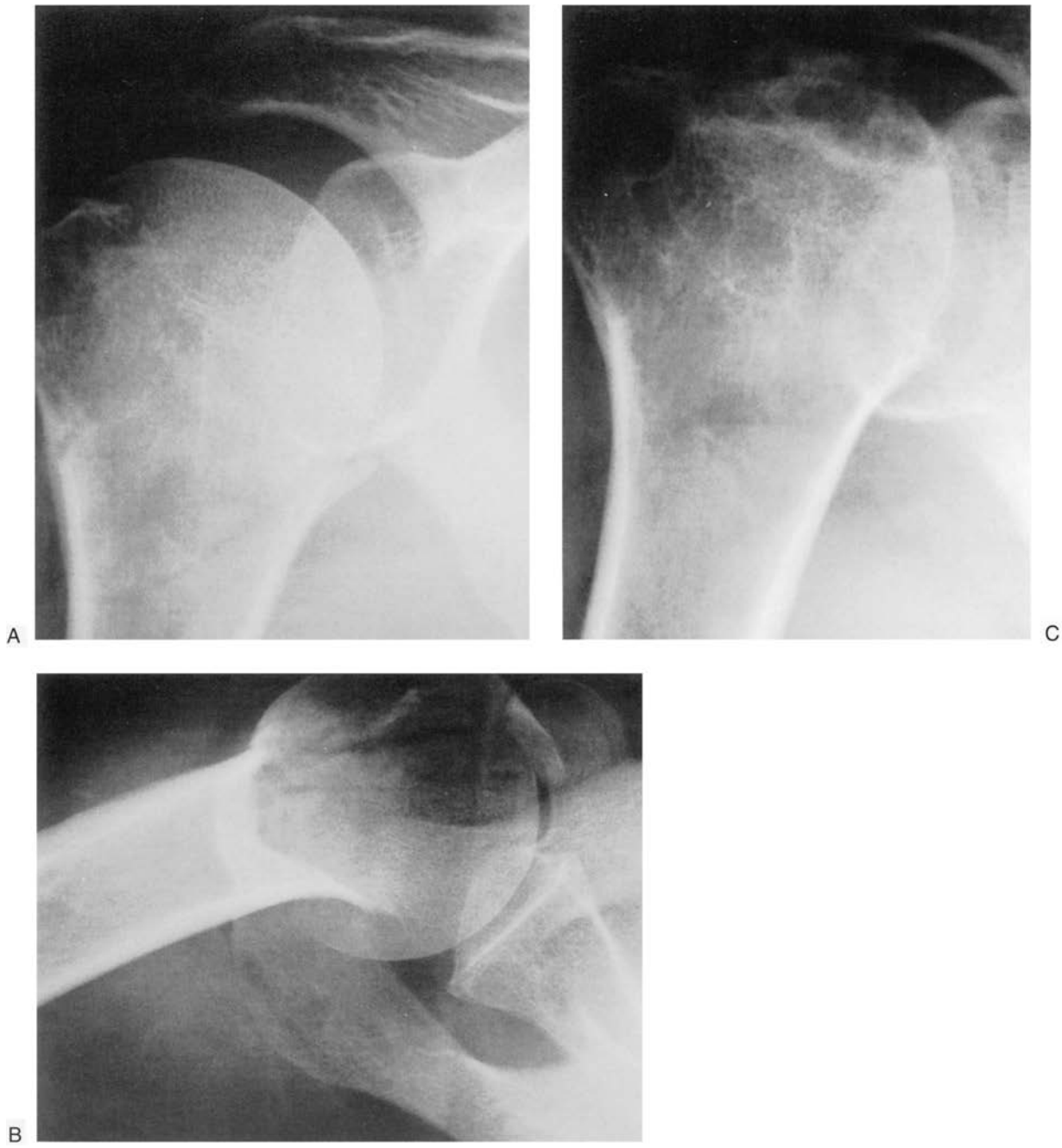


Figure 18-26 **(A,B)** Osteonecrosis: Minimally displaced fracture of the proximal humerus, managed with closed treatment. **(C)** However, posttraumatic segmental osteonecrosis developed, requiring hemiarthroplasty reconstruction.

Steroid ingestion is the predominant identifiable risk factor.[84,340,515] The precise mechanism of occurrence is unknown, although an initial reduction of blood flow has been reported when high-dose methylprednisolone is administered to a porcine model.[86,87,113,529] There is alteration in serum lipid content and concentration with associated fatty change within the liver. Systemic fat emboli may occur.[137,229] Osteonecrosis may follow short-term use as well as intraarticular injections.[11,85,128,279,371,378,499]

L'Insalata et al. reviewed a series of patients who had symptoms for 9 years.[292] The shortest interval from the completion of steroids to the onset of symptoms was 1 year. The shortest course of steroids was 1 week. In Cruess's study, the interval to the development of symptoms was not less than 6 months from the onset of steroid ingestion, the longest interval being 18 months.[85] L'Insalata et al. observed that 76% of patients using steroids developed osteonecrosis at sites other than the shoulder: the hip and

knee, most commonly.[292] Mont et al. observed that the incidence could not be correlated with the dosage or the duration of steroid ingestion.[340] In 74% of cases, bilateral disease was identified.

The excessive intake of alcohol is associated with the development of osteonecrosis.[216,219,377] In an experimental study with rabbits, alcohol ingestion induced adipogenesis, decreased osteogenesis in bone marrow stroma, and produced intracellular lipid deposits resulting in the death of osteocytes.[530]

Gaucher's disease is a well-known cause of osteonecrosis.[429,497] This autosomal recessive disorder is characterized by the accumulation of sphingolipid within the macrophages of the reticuloendothelial system (Gaucher's cells) due to the deficiency of glucocerebroside hydrolase. Gaucher's cells tightly pack the hematopoietic marrow leading to circulatory embarrassment of the surrounding bone. Risk factors for osteonecrosis in patients with type I Gaucher's disease are splenectomy and male gender.[429]

Other causes of osteonecrosis include sickle cell disease and other hemoglobinopathies, tobacco intake, hyperbaric exposure, decompression sickness, pancreatitis, familial hyperlipidemia, renal or other organ transplants, lymphoma, bismuth encephalopathy, gout, Cushing's disease, radiation, electrical shock, chemotherapy, Hodgkin's disease, myxedema, peripheral vascular disease, psoriasis, pregnancy, renal dialysis, systemic lupus erythematosus, and idiopathic diseases.[124,160,184,196,228,278,294,327,408,435]

Pathophysiology

The blood supply of the proximal humerus plays a critical role in the development of posttraumatic avascular necrosis; the incidence is not uncommon after displaced fractures of this region (Fig. 18-27). Most of the blood supply

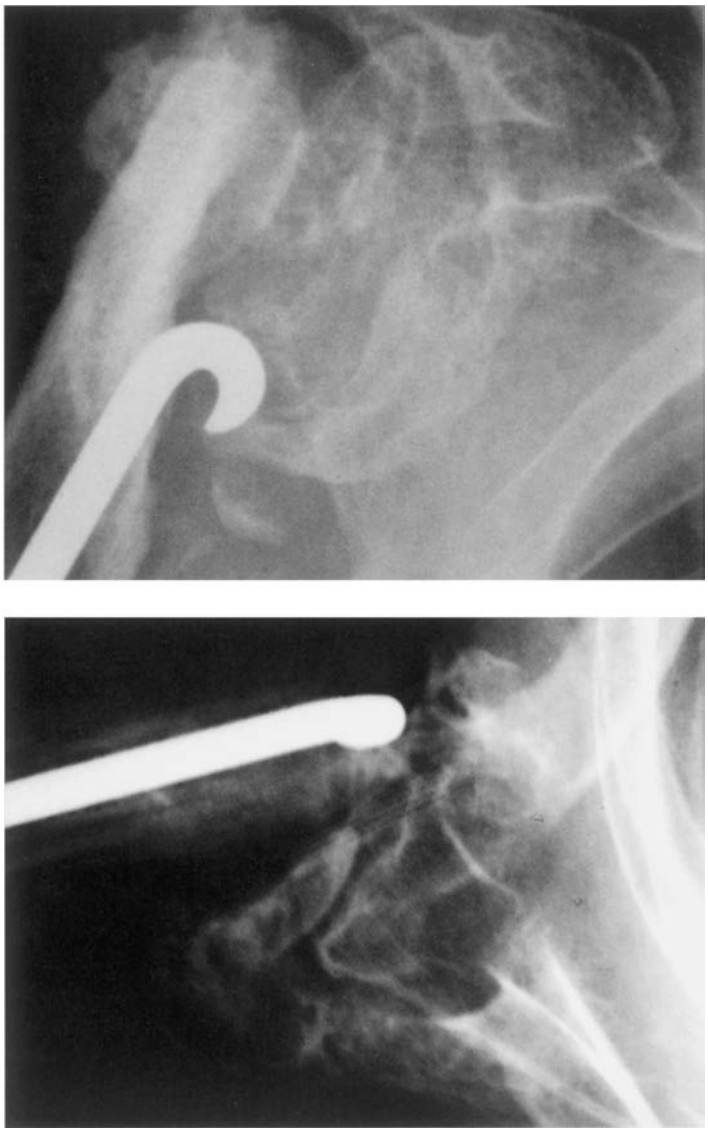


Figure 18-27 **(A,B)** Osteonecrosis: Displaced comminuted fracture of the proximal humerus, treated by open reduction and internal fixation (ORIF), resulting in total resorption of the avascular articular segment.

to the humeral head derives from the anterior humeral circumflex artery through its ascending branch.[275,344,417,436] Entry into the humeral head is in the region of the biceps groove. The arcuate artery is the intraosseous communication that arborizes and becomes the major blood supply to the head (Fig. 18-28).[150] Collateral circulation, normally not present to a great extent in the shoulder, will become insufficient if the fragments are widely displaced. The posterior humeral circumflex artery through rotator cuff attachments will provide a small amount of collateral flow. In addition, the more anterior portion of the cuff will receive other branches of the anterior humeral circumflex, which provides collateral circulation for the articular segment.[344,417,436]

It is possible that the event of circulatory arrest leading to humeral head osteonecrosis occurs at the moment of injury. Forceful manipulative attempts at reduction or surgical intervention may further disturb precarious circulation to the articular segment. When indirect reduction techniques were utilized, the incidence of osteonecrosis was reduced to 4%.[195] Although commonly felt to favor prevention of osteonecrosis in the hip, the effect of prompt reduction and stabilization on the incidence of osteonecrosis of the humerus is unknown. Because the vascular supply plays such a critical role in proximal humerus fractures, classifications based on this fact alone have evolved, allowing prognosis after injury and treatment.[220]

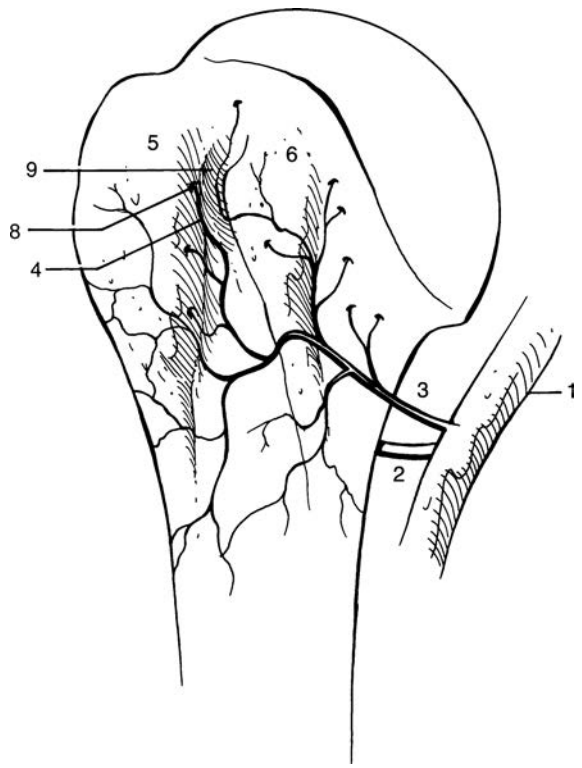


Figure 18-28 Graphic representation of the anterior aspect of the humeral head depicting the arterial vascularization: **(1)** axillary artery, **(2)** posterior circumflex artery, **(3)** anterior circumflex artery, **(4)** anterolateral branch of the anterior circumflex artery, **(5)** greater tuberosity, **(6)** lesser tuberosity, **(7)** constant site of entry of the anterolateral branch into bone, **(8)** intertubercular groove. (From Gerber C, Schneeberger A, Vinh TS. The arterial vascularization of the humeral head. *J Bone Joint Surg Am* 1990;72: 1489, with permission.)

Whereas the underlying diagnosis in femoral head osteonecrosis is often elusive and determined to be idiopathic, the cause of nontraumatic osteonecrosis of the humeral head is often identifiable. Despite the etiologic factors involved in the development of osteonecrosis, a common pathway is followed as the disease process evolves. Unlike the usual arthritides that begin with the involvement of the articular cartilage, osteonecrosis initially involves the subchondral cancellous bone in the portion of the humeral head under the greatest load. Later, the articular surface may be altered and, eventually, more extensive damage extends to the glenoid.

Osteonecrosis from disease processes probably results from repetitive insults leading up to a threshold of vascular embarrassment. The resultant ischemia of the marrow elements and osteocytes eventually leads to infarction. Pain may or may not be present at this stage. Discomfort may be related to increased intraosseous pressure in the absence of collapse of the articular surface. What role increased intraosseous pressure plays at this point is uncertain, although its presence has been clearly documented.[134,264]

Pathoanatomy

The repair process is initiated soon after the initial vascular insult. The response is vascular ingrowth in attempt to remove necrotic marrow and to rebuild necrotic bone. The repair process is ongoing, even continuing in the presence of vascular insults. Continued stress on viable bone resulting in microfractures, trabecular collapse, and compression may establish a front impenetrable by the repair granulation tissue. At the time of collapse, the overlying subchondral bone and cartilage remain intact. Initially, this occurs in the area of maximum joint reaction force and creates a gap, which radiographically is known as the "crescent sign." With further loading, there is complete segmental failure with incongruity of the subchondral bone.

Articular surface damage may take place even though the cartilage remains viable (Fig. 18-29). Depending on the extent of involvement, the humeral head undergoes progressive secondary degeneration. As a result of bearing against the morphologically altered humeral head, the glenoid eventually develops osteoarthritic changes.

Clinical Evaluation

The chief complaint of patients with osteonecrosis is pain. The onset is usually insidious and discomfort is not initially severe. In sickle cell disorders, the clinical distinction

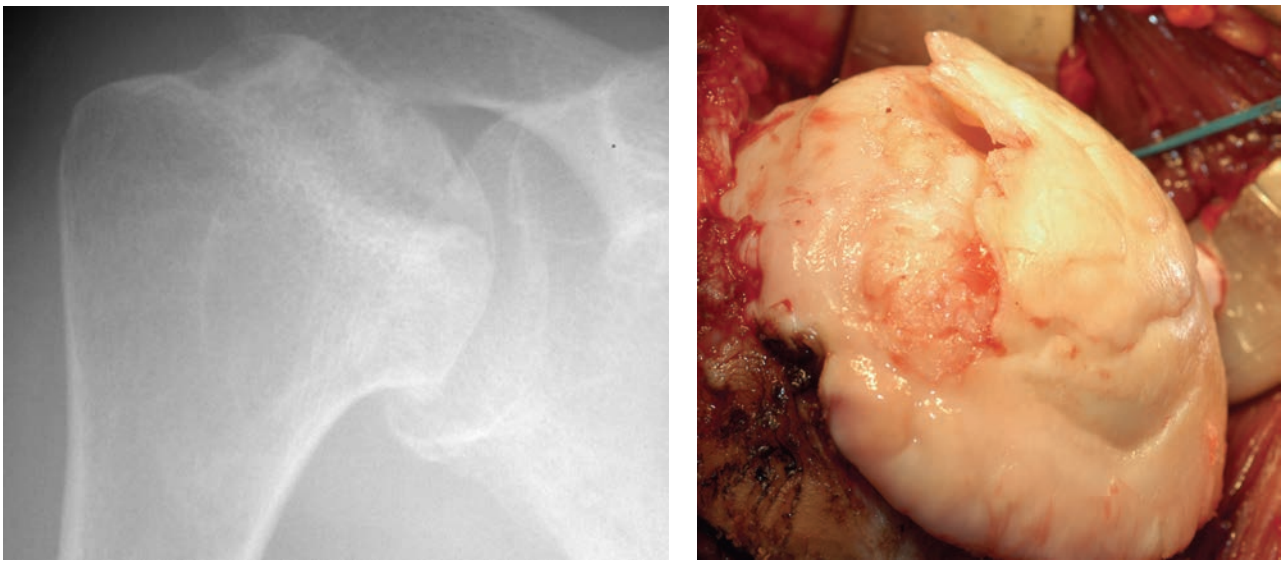


Figure 18-29 **(A)** Stage IV osteonecrosis of the humeral head. **(B)** The articular cartilage appears viable despite near-complete detachment from the underlying subchondral bone. Secondary osteoarthritic changes are present.

between a crisis, bone or synovial infarction, and incipient osteonecrosis may be extremely difficult.[452]

Proximal humerus fractures, with anatomic or near anatomic reductions and stable fixation, may achieve a satisfactory result even in the presence of osteonecrosis.[149,200,534] Hattrup and Cofield reported a series of 200 patients with humeral head osteonecrosis in which more than 25%, regardless of stage, were managed nonoperatively in a satisfactory manner for 2 to 14 years.[186] Greater deformity of the humeral head is better tolerated by the shoulder due to the extent of loading ("non–weight bearing") and because of the maintenance of scapulothoracic motion.[335,515] For other patients, shoulder pain may later intensify with progressive involvement of the head or owing to alterations of the articular surface. Not only will there be an intense synovial response, but mechanical symptoms of locking or catching ensue. Early in the disease, the shoulder range of motion is maintained with guarding at the extremes. Gradually, the range diminishes to that required for essential functional tasks, later accompanied by atrophy and weakness. Crepitation of articular origin can be elicited as the shoulder actively or passively rotates.

Imaging

Imaging studies include tomography, MRI, bone scintigraphy, and CT to assess the amount of humeral head involvement, bone loss, and the distortion of underlying anatomy. McCallum and Walder offered a classification scheme that separated humeral head, neck, and shaft involvement from juxtaarticular lesions.[316] In the latter, the earliest findings were dense areas with an intact cortex. Spherical segmental opacities and linear opacities were noted. When structural failure occurred, a translucent subcortical band could be seen. Later, the articular cartilage would collapse and the cortex would be separated. The final stage was osteochondrosis.

The radiographic progression of the disease has been more recently staged according to the system of Ficat and Arlet, as modified by Cruess (Fig. 18-30).[85,86,134] Stage I includes subtle changes within the humeral head. Pain may or may not be present. The plain films show that the humeral head is round, with some mottling present: MRI examination will pick up this early finding. Stage II is the development of focal sclerosis without collapse. The anteroposterior view of the humerus in external rotation is often diagnostic at this stage, as is the MRI. The cartilage surface can be round, but there is some ballotability of the affected segment. Stage III is the development of collapse or a crescent sign (Fig. 18-31). There is no displacement of the articular surface more than l or 2 mm. The cartilage may loosen as a flap, creating symptoms of internal derangement. At this point, the glenoid is normal. Stage IV results in a displaced cartilaginous flap accompanied by humeral head softening and collapse (Fig. 18-32). Stage V is wear of the cartilage and the bone of both the glenoid and humerus. The joint becomes incongruent with secondary degenerative changes (Fig. 18-33). The MRI findings of humeral and femoral heads with osteonecrosis are similar.[285] In femoral heads with MRI-proven osteonecrosis, bone scintigraphy demonstrated sensitivity, specificity, accuracy, positive predictive value, and negative predictive value of 65%, 81%, 77%, 54%, and 87%, respectively.[444] In femoral heads, subchondral fractures have been more

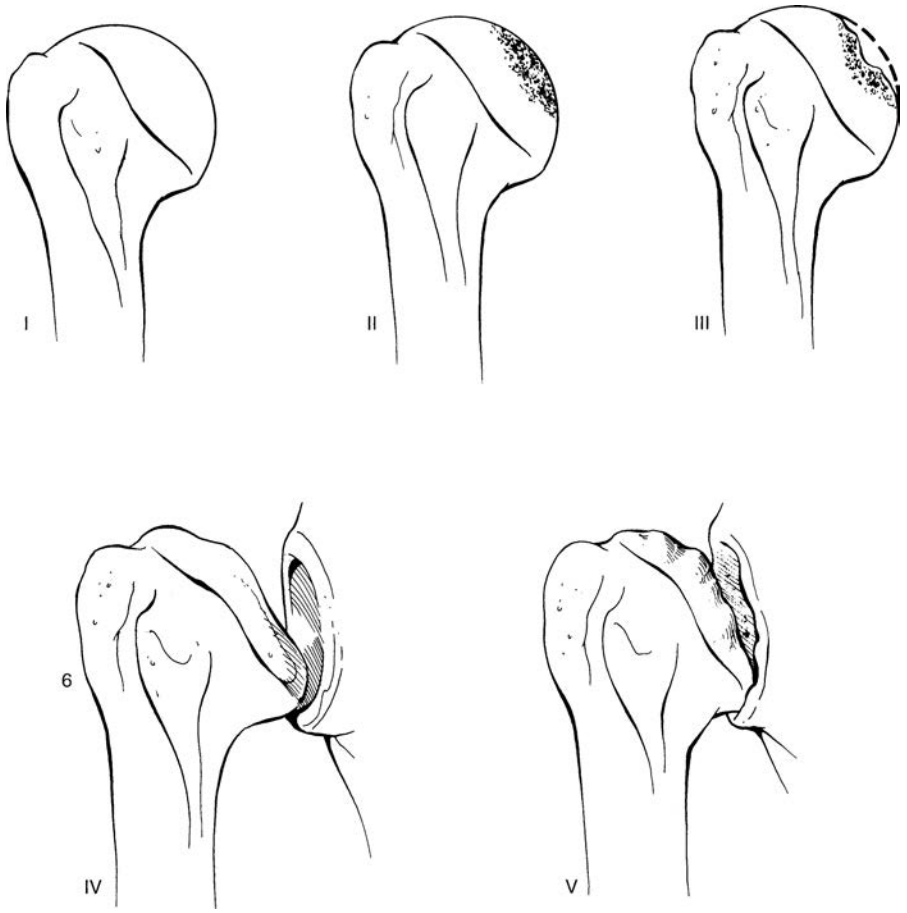


Figure 18-30 Osteonecrosis: Artist-rendered figure of staging system for humeral head osteonecrosis. (Modified from Cofield RH. Osteonecrosis. In: Friedman RJ, ed. *Arthroplasty of the shoulder.* New York: Thieme, 1994:174.)

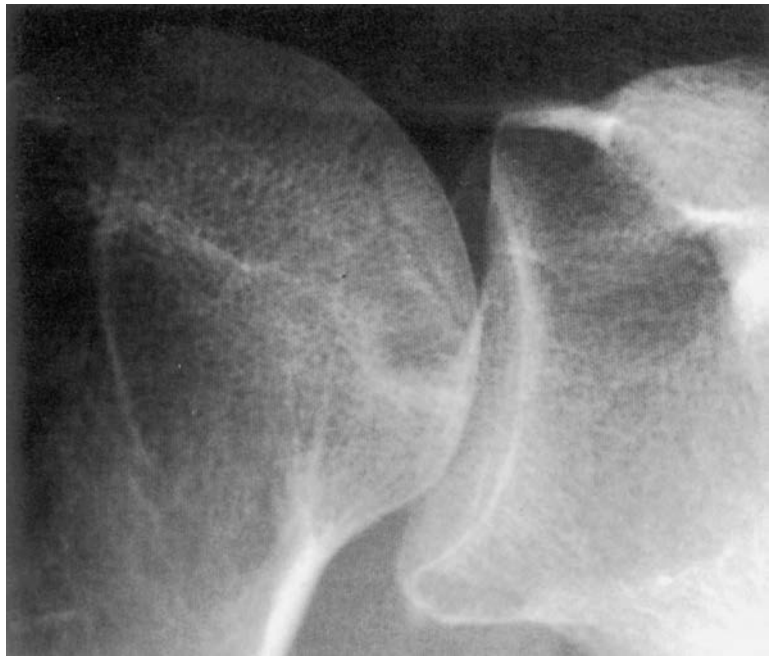
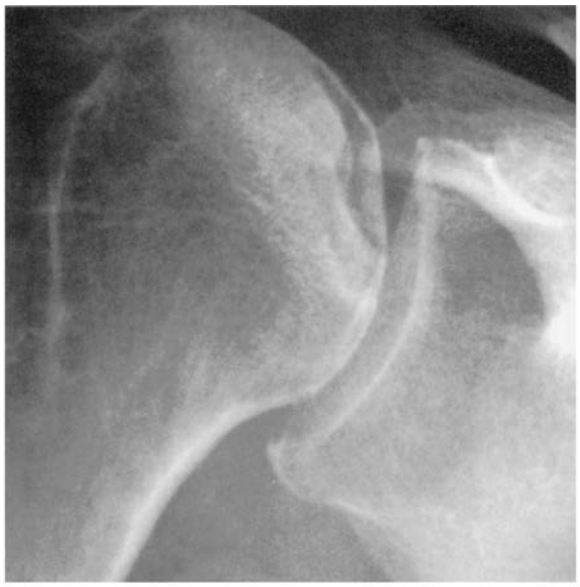
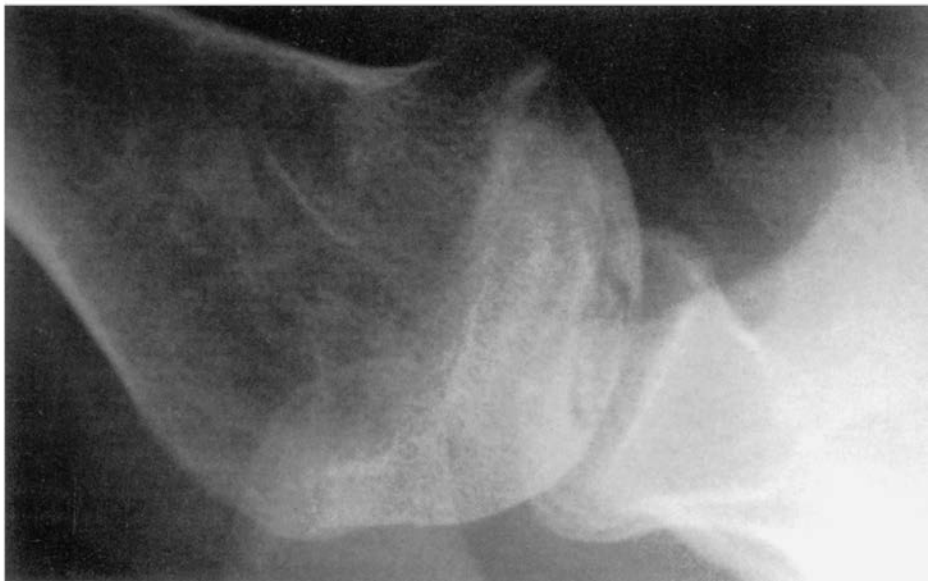


Figure 18-31 Osteonecrosis: Stage III. A "crescent sign" is seen. There is preservation of the subchondral articular margin.



A



B

Figure 18-32 **(A,B)** Osteonecrosis: Stage IV osteonecrosis of the humeral head with localized collapse and irregularity of the articular surface.

reliably demonstrated with CT scan rather than MRI.[489] Kishida et al. showed that the diagnostic ability of three-dimensional spoiled gradient-echo (3D SPGR) MRI provided more accurate measurements of the area and volume of a necrotic lesion than T1-weighted SE imaging.[253]

Laboratory

Laboratory studies may be important for the detection of underlying diseases.

Natural History

The necrotic segment size may vary in traumatic occurrences of osteonecrosis. Smaller areas of involvement may be sufficiently revascularized and stabilized before the stage of collapse. Larger necrotic segments remain isolated from incoming circulation, inevitably progressing to collapse and eventual head destruction. Sometimes, however, the glenohumeral joint congruity will be preserved, allowing maintenance of adequate function.[149,284,346]

Almost three-fourths of patients with atraumatic osteonecrosis will have progression of their disease to the extent that will require surgery, or will result in significant pain or disability.[85,87,426] At the time of presentation, 20% of patients in L'Insalata et al.'s series required surgery on the basis of pain alone.[292] Thirty-four percent responded initially to conservative care for an average period of 2 years, and then required surgery. Forty-six percent responded to conservative care for an average of 10 years.

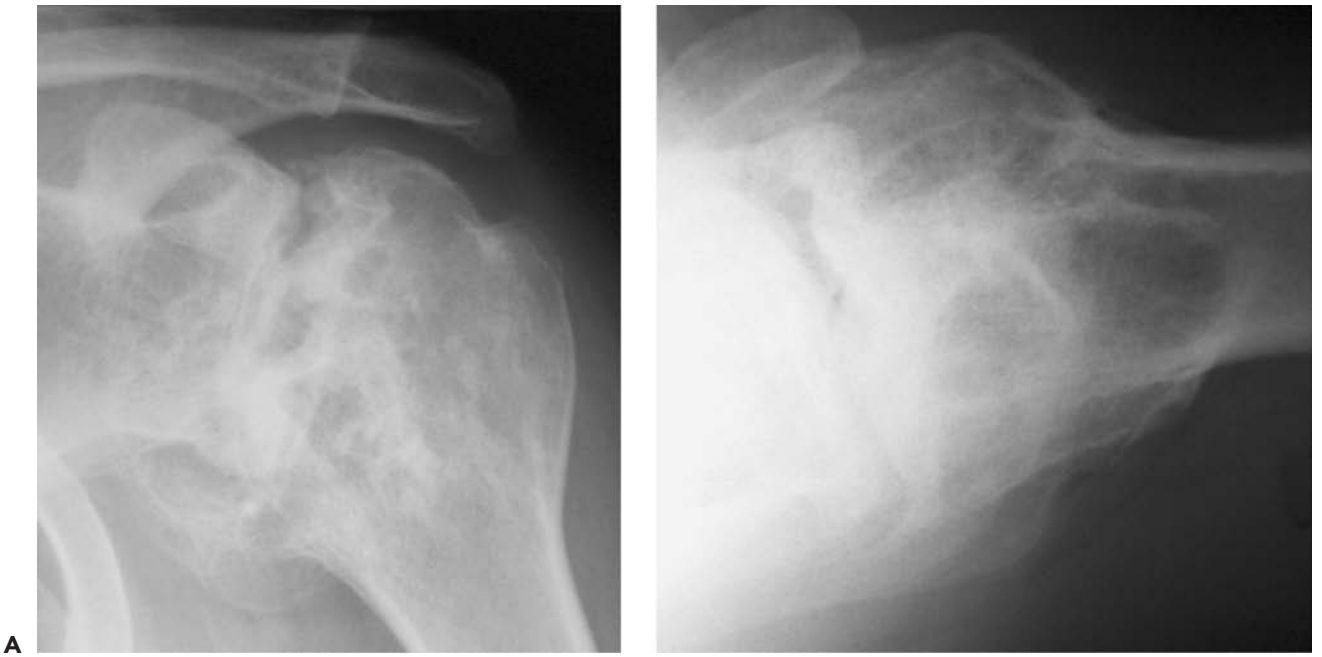


Figure 18-33 **(A,B)** Osteonecrosis: Stage V. Secondary osteoarthritic changes of the humerus and the glenoid following proximal humerus osteonecrosis.

Half of these patients were satisfied and half were unsatisfied with their outcome. At 4.5 years after presentation, 80% of Rutherford and Cofield's patients with stage II and III disease had no clinical progression.[442] In a follow-up study at the same institution, Hattrup and Cofield remarked that the need for shoulder arthroplasty was dependent on the underlying diagnosis and the extent and Cruess stage of humeral head involvement.[186] At a 3-year interval, traumatic etiology versus steroid intake was more likely to result in surgical treatment, 80% versus 43%. Three-fourths of patients initially treated nonoperatively continued to be pain-free, or have only moderate discomfort, almost 9 years after diagnosis. Clearly, radiographic progression is commensurate with a poor outcome. This is particularly true with radiographic stage III, or greater, and evidence of disease progression. L'Insalata et al. observed that poor outcomes were more common in women than in men and that patients with steroid-induced disease had a better prognosis.[292]

RHEUMATOID ARTHRITIS

Definition

Rheumatoid arthritis is a systemic autoimmune disorder of unknown cause. It is believed to occur in individuals who are genetically predisposed in response to an arthritogenic agent or antigen, perhaps environmental. Although its effects may be systemic, its major distinctive feature is chronic, often symmetrical erosive synovitis of the peripheral joints.

Incidence

Rheumatoid arthritis is the most prevalent inflammatory arthropathy in adults, occurring in 1% to 2% overall and 2% to 2.3% in the geriatric population.[415] It is most prevalent in the fourth and fifth decades.[88] There is considerable variation in the prevalence and incidence of rheumatoid arthritis among different populations. In Northern Europe and North America, the prevalence of the disease is 0.5% to 1.0%, with the mean annual incidence of 0.02% to 0.05%.[4]

The incidence of rheumatoid arthritis is approximately 20 to 40 in 100,000 adults. Women have a two- to threefold greater risk, and the disease is more prevalent with advancing age.[203]

The shoulder is invariably involved, usually as a clinical feature of progressive disease.[167,336] It is not commonly one of the first manifestations of the disease; never is it the only one. Its presence is usually bilateral.[88] The actual incidence of glenohumeral involvement, depending on which patient group or what stage of the disease is selected or studied, ranges from 20% to 90%.[82,288,394] The incidence is 50% to 60% when polyarticular rheumatoid disease exists.[289,427,482] The incidence is highest in patients undergoing surgical intervention, whereas shoulder occurrences are much less frequent with those patients who manifest minor symptoms of the disease.

It may be the first joint to be involved in approximately 4% of the cases with rheumatoid arthritis as well as the most common large joint to be affected at the time of presentation.[185,375] When it is the presenting joint (up to 21% of cases), the illness soon becomes systemic and polyarticular.[375] Therefore, only rarely can rheumatoid arthritis account for a chronic monarthritis of the shoulder.

Pathogenesis

Although connective tissue, wherever it is present, is susceptible to the effects of rheumatoid disease, its greatest effect is on the internal aspects of the joint, especially the synovial membrane. A single primary cause has not been determined.[336] Risk factors are genetic susceptibility, sex and age, smoking, infectious agents, hormonal, dietary, socioeconomic, and ethnic factors.[4] Infectious agents, autoimmunity, and heritable factors, their activities perhaps interrelated, may have a role.[336] Rheumatoid factor (IgM) may be found in 3% of healthy people. Its role in rheumatoid arthritis may be to amplify rheumatoid inflammation, but it does not appear to serve as a primary etiologic agent or as a trigger.[453]

Pathophysiology

The exact sequence or cause of events leading to symptomatic rheumatoid arthritis has not been determined. The initial pathologic event appears to be activation and injury of subsynovial microvascular endothelial cells.[458,476] T lymphocytes transgress the vascular endothelium and come to lie in the subsynovial areas around small capillaries, where their surfaces acquire antigens, processed and presented by tissue macrophages.[222,405] These cells, as well as B-cell lymphocytes and their products—proinflammatory cytokines (interleukin-1 [IL-1], tissue necrosis factor-alpha [TNF-α], and immunoglobulins)—proliferate in a poorly restrained manner.[494] Metalloproteinases, nitrogen oxide, hydrogen peroxide, TNF-α, and interleukin-beta accumulate in cytotoxic concentrations, further activating collagenase and stromelysin.[476] Complex pathways of oxygen metabolism resulting in reactive oxygen species exhibit toxicity to cellular and molecular components and contribute to the mediation of the inflammatory response.[202] The normally flimsy, one- to two-cell–thick lining layer and sublining synovial membrane may thicken by as much as 100 times through the process of hypertrophy and activation of the connective tissue stroma.[382] Joint fluid volume increases with activation and proliferation of the synovial cells. Inflammatory cells and chemical mediators of inflammation accumulate and weaken the supporting soft tissue structures of the synovium.

The synovitis resembles both tumor growth and wound healing, relying on new blood vessel proliferation—angiogenesis—to sustain its expansion.[270,488] These abnormal cells develop abrasive qualities that take on the behavior of malignancy and advance onto the adjacent cartilage and bone.[127] This tissue, which continues to proliferate, develops invasive properties, and bears resemblance to granulation tissue, is known as pannus.[182] Whether its initiation is pathologic or reparative has not been clarified.[182] It can invade adjacent tendons, tendon sheaths, ligaments, bone, and articular cartilage. As the cartilage is eroded, breaks in the underlying bone may develop. Cysts may form and subsequently coalesce into large subchondral defects with potential for collapse in areas of greatest load.

Pathoanatomy

Crossan identified three pathologic phases of synovial inflammation: exudative, infiltrative, and degradative.[83] The exudative phase begins at the articular cartilage margin. There is increase in capillary permeability, initially with the efflux of fluid into the interstitial space. The infiltrative phase is marked by the escape and movement of cells into the subsynovial and periarticular tissues. Monocytes and lymphocytes invade the inflamed synovium when earliest biopsies are obtained. The development of lymphoid follicles may occur, but they rarely have germinal centers. When this takes place, an increased number of plasma cells follow. Multinucleated giant cells may exist in the subsynovial layers. The activated synovial surface appears villous and papillary as different portions exist in different stages of development. The degradation phase begins with peripheral marginal injury to the avascular hyaline cartilage. The result is disorganization and loss of the intercellular matrix, yielding to limited repair attempts by fibrosis. Chondrocyte and matrix loss results in cartilage thinning. Pannus may undermine the subchondral and marginal bone and result in osteoclastic bone destruction, observed as the periarticular erosion seen on radiographs.

The entire shoulder structure may become involved in the rheumatoid process. Earliest changes are in the adjacent soft tissue, including the subacromial bursa, rotator cuff tendons, and the long head of the biceps. In the early stages, the rotator cuff is vulnerable to infiltration by rheumatoid synovium. This may result in attenuation or rupture of the cuff and the formation of subacromial bursal effusions. From 20% to 50% of patients acquire full-thickness tears and a smaller number, partial-thickness tears.[125,143,356,365] In patients undergoing total shoulder arthroplasty for rheumatoid arthritis, approximately one-third will have a full-thickness rotator cuff tear.[143,356,406] Arthrography fails to demonstrate up to one-third of intertubercular grooves, implying biceps tendon stenosis within the canal.[104] In later stages, when destruction of the humeral articular cartilage is prominent, up to two-thirds of the biceps tendons rupture.[383] Poor bone quality, subchondral cysts, and soft tissue contractures are often recognized.

The characteristic erosions of glenohumeral rheumatoid arthritis occur in the superior aspect, medial to the greater tuberosity at the synovial reflection, and correspond to the most common site of rotator cuff tearing. The humeral head may ascend with secondary erosive changes owing to glenohumeral incongruity. A classification of shoulder disease in 100 patients with rheumatoid arthritis undergoing lower extremity surgery described these changes.[83] Forty percent of shoulders initially manifested only erosive changes, often involving the acromioclavicular joint. The humeral head remained spherical, and the glenohumeral articulation remained normal. In 40%, proximal subluxation of the humeral head occurred. In half of these, the acromiohumeral interval was less than 6 mm as a result of significant soft tissue damage. The humeral head remained spherical and the glenoid preserved in approximately one-half of the cases. End-stage disease occurred in the remaining 20%. The humeral head was destroyed with associated destructive changes of the glenoid. Resorption was a common feature. The acromiohumeral interval was significantly diminished, and there were gross distortions in glenohumeral relations.

Clinical Evaluation

The symptoms and manifestations of glenohumeral rheumatoid arthritis usually have an insidious onset and slow progression. Adaptive and compensatory mechanisms by the elbow and wrist enable most patients to maintain their functional status while the disease progressively takes its toll on the glenohumeral joint and soft tissues. As a result, advanced changes often manifest at initial presentation. The shoulder symptoms typically do not correlate with the general severity or activity of the disease. In the later stages of the disease process, patients with rheumatoid arthritis have a tendency toward more significant impairment of shoulder function than patients with osteoarthritis, while their overall health status is significantly worse.[308]

Pain is the most common symptom reported by patients with glenohumeral rheumatoid arthritis and is reported by 67% to 91% of patients.[286] In 20% of these patients, it is the result of moderate or severe glenohumeral joint destruction within the first 15 years of the disease.[286] The pain may be intense, even before radiographically advanced changes of severe bone loss, osteopenia, erosions, and humeral head translocations are observed. Unlike other involved joints, night pain is more typical when the shoulder is involved. Patients sense an awareness of stiffness. Functional loss may occur early in the disease.

The physical findings include those related to inflammation: tenderness, often diffuse, but sometimes more localized to the joint line posteriorly, as well as the infracoracoid region corresponding to the anterior joint line. Tenderness is seen more commonly in older patients and in more severe disease.[375] There may be increased overlying cutaneous warmth when compared with the adjacent areas. Erythema is not common. Swelling from a glenohumeral joint effusion may be present, although not always prominent. More often, the swelling is observed superiorly in the subacromial bursa from an extension of articular fluid through a defect in the rotator cuff. Atrophy of the shoulder girdle may be present, although the presence of swelling and effusions may make recognition more difficult. Motion restriction may be attributable to several causes. Reflex activity limitations because of discomfort will accompany most conditions that cause pain. Active range-of-motion losses and weakness may occur because of rotator cuff tears. Some motion loss may be noted from weakness because of muscle atrophy. Joint capsule contractures may inhibit both active and passive range of motion. Large effusions and articular surface changes may contribute to diminished range of motion.

Imaging

Larsen et al. presented the first radiographically refined and standardized grading system for the radiographic severity of rheumatoid arthritis.[280] This method was applied to multiple small and large joints, including the shoulder (Table 18-3).

Radiographically, Neer identified three clinical types of rheumatoid glenohumeral arthritis.[364] The dry type is characterized by loss of the joint space, formation of subchondral cysts, sclerosis, and marginal osteophytes, not unlike osteoarthritis (Fig. 18-34). Marginal erosions are infrequent. Patients have more difficulty mobilizing the shoulder involved with this type of rheumatoid arthritis. A wet type results from the proliferation of exuberant granulation tissue at the articular margins (Fig. 18-35). Marginal erosions, sometimes quite large, are characteristic of this pattern. The proximal humeral architecture, altered by a gradual loss of humeral head and tuberosity contours, acquires a "pointed appearance." The wet and resorptive phase is the most destructive, characterized by "centralization" (Fig. 18-36). This phenomenon evolves in response to a rapid and severe articular cartilage and bone loss. Medial migration with the loss of bone mass results in a loss of the normal physical contour of the shoulder (Fig. 18-37). In 1954, Laine et al. proposed a similar classification based upon clinical and radiologic findings.[274]

The radiographic findings are dependent on the duration and extent of the disease, and may reflect the quality of medical management. Symmetry is characteristic of rheumatoid arthritis. The initial radiographic features may be normal, but later, because of disuse or local inflammation, osteopenia may be observed (Fig. 18-38). Osseous erosions appear at sites of synovial tissue concentrations, most commonly the anatomic neck, superior and medial

TABLE 18-3
LARSEN RADIOGRAPHIC STAGING FOR RHEUMATOID ARTHRITIS

Grade	Description	Anatomic findings
0	Normal	
I	Slight abnormality	One or more: periarticular soft tissue swelling, periarticular osteoporosis, slight joint space narrowing
II	Definite early abnormality	Erosion and joint space narrowing
III	Medium destructive abnormality	Erosion and joint space narrowing
IV	Severe destructive	Erosion and joint space narrowing; bone deformity
V	Mutilating	Disappearance of original articular surfaces; gross deformity

From ref. 166, with permission.

to the greater tuberosity. Lehtinen et al. observed that a significant joint space reduction did not occur until Larsen grade 4 destruction.[287] At the same time, they observed that extensive erosions on the glenohumeral joint are followed, not preceded, by joint space narrowing. With the gradual concentric loss of articular cartilage and erosion of the subchondral bony plates, the glenoid and the humeral head move closer together (Fig. 18-39). In rare instances, osteophytes may secondarily occur. Invasive biceps tenosynovitis may produce surface erosions of the intertubercular groove.

The position of the humeral head relative to the glenoid and to the acromion is controlled by several factors. Initially, there may be ascent of the humeral head because of weak muscles. Later, with attrition and rupture of the rotator cuff, the acromiohumeral interval narrows further. It has been observed that upward migration precedes medialization and gross destruction of the glenohumeral articular surfaces.[286] Progressive upward migration is an inevitable consequence of rheumatoid destruction in the glenohumeral joint. A significant step in this process was observed between the Larsen grades of 3 and 4, where the mean distance turned negative, indicating rotator cuff disease.[287] During the end stages of disease, the acromiohumeral interval may become obliterated with the formation of a new acromiohumeral articulation. At this point, gross destruction of the glenohumeral articulation may be observed. Large cysts, subchondral sclerosis, and secondary

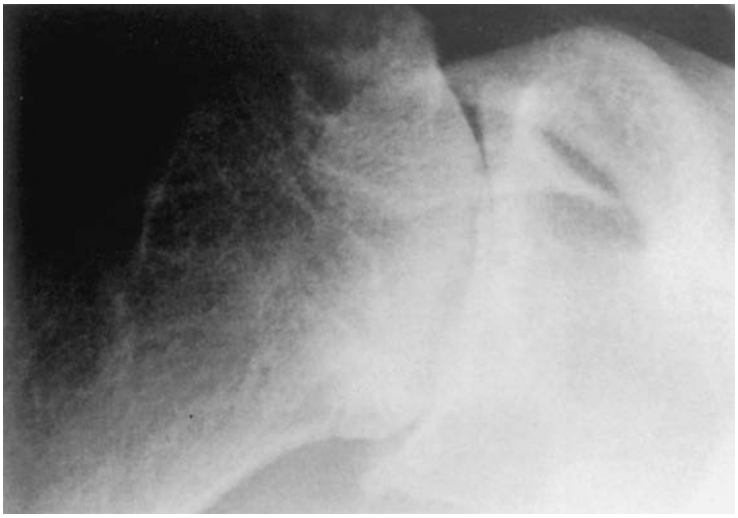


Figure 18-34 Rheumatoid arthritis: Dry type, characterized by loss of joint space, sclerosis, and marginal osteophytes.

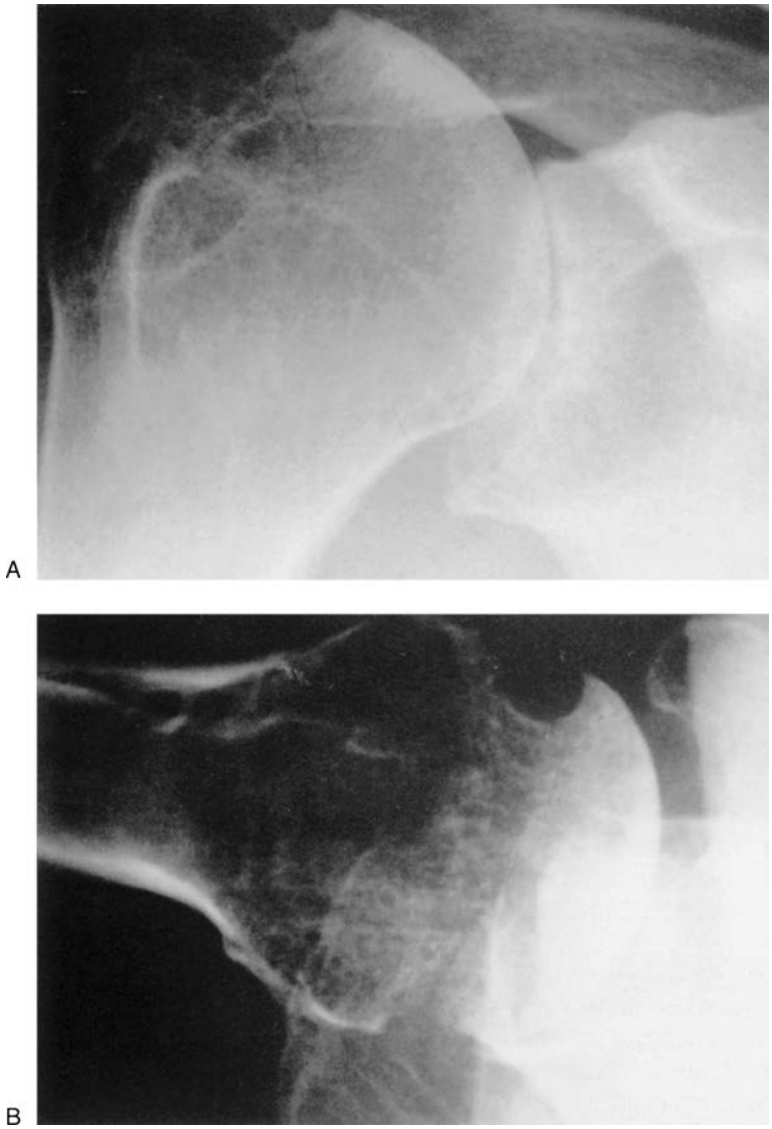


Figure 18-35 **(A,B)** Rheumatoid arthritis: Wet type, with large erosions characteristically found at synovial attachment sites.

osteoarthritis may develop (Fig. 18-40). There may be overall centralization of the joint due to extreme glenoid erosion (Fig. 18-41). The humeral head may resorb and collapse onto the glenoid (Fig. 18-42).

Hirooka et al. observed that certain radiographic findings apparent at 5 to 10 years are capable of predicting the prognosis for shoulder joint destruction at 15 to 20 years, implying the opportunity for treatment stratification.[201]

Computed tomography scanning will help to better define the extent of the erosions and cyst formation within the humeral head and glenoid.[7] A determination of the volume and orientation of the glenoid vault can be made in preparation for arthroplasty reconstruction.[142] Significant centralization may result in a wafer-thin glenoid, which would yield to the preparation and placement of a prosthetic glenoid component. Mullaji noted that only one-half of the surface area of the rheumatoid glenoid was supported by sufficient bone.[348] Humeral bone quality in rheumatoid arthritis may be underestimated by CT.[7]

Arthrography can be used to discriminate an acute rheumatoid flare from a full-thickness rotator cuff tear. When there is significant stiffness, adhesive capsulitis can be assessed. Chronic synovitis is often diagnosed on the basis of filling defects within the joint. Distension of the biceps tendon sheath may be observed.[103]

When the initial radiographs are unrevealing, sonography and MRI can provide supplementary information about the disease process.[6,193] With sufficient experience, sonography can be useful to image the osteoarticular changes about the shoulder resulting from rheumatoid arthritis.[193] Sonographic abnormalities may be present both in patients with and without shoulder complaints as well as in patients with normal findings on physical

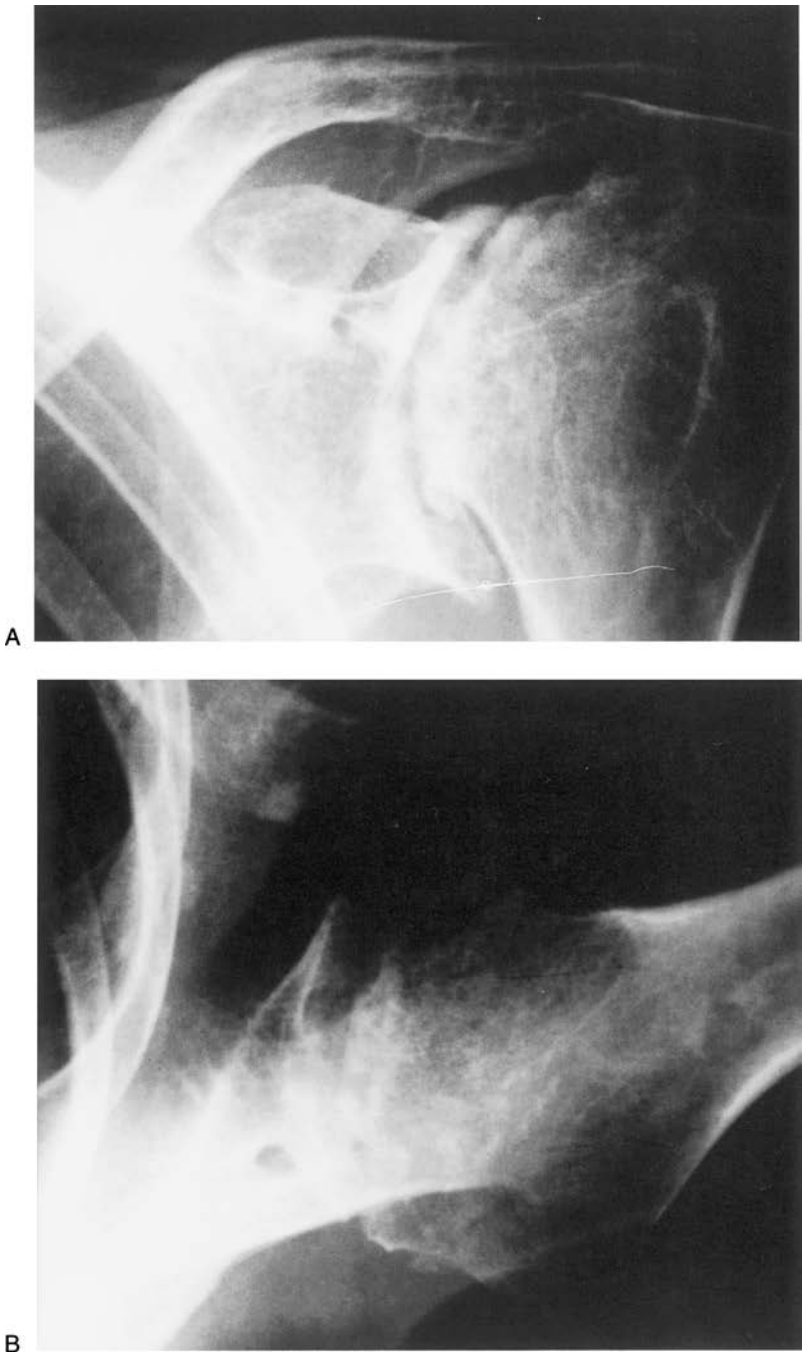


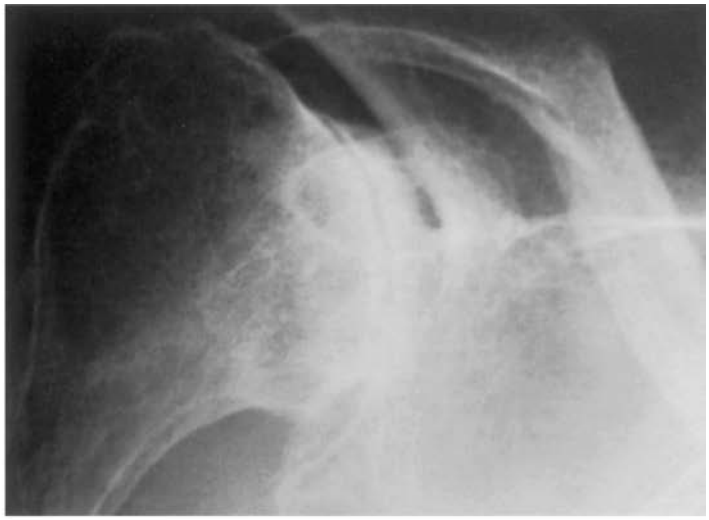
Figure 18-36 Rheumatoid arthritis: **(A,B)** Wet and resorptive type. Subchondral cyst formation and erosion of the glenoid and humeral head.

examination.[352] Sonography has not proven useful to distinguish glenohumeral synovitis from a joint effusion.[528] It has been shown to be more useful early in the disease process before the normal anatomy and motion are significantly altered.[5]

MRI is probably the best overall imaging modality for rheumatoid arthritis capable of detection with a high degree of accuracy and detection of the earliest changes in the soft tissue and damage to articular cartilage and bone.[480] An MRI scan can accurately determine the presence of synovial fluid, estimate its volume, and trace the extent of its dissection through the periarticular tissues. The rotator cuff integrity and quality can be assessed. In the rheumatoid knee, subchondral cyst contents and dimensions may become more apparent with MRI, their detection dependent on size, location, and perhaps biologic activity.[399]

Laboratory

Blood work is important for the evaluation of patients suspected of having rheumatoid arthritis.[64] Anemia is



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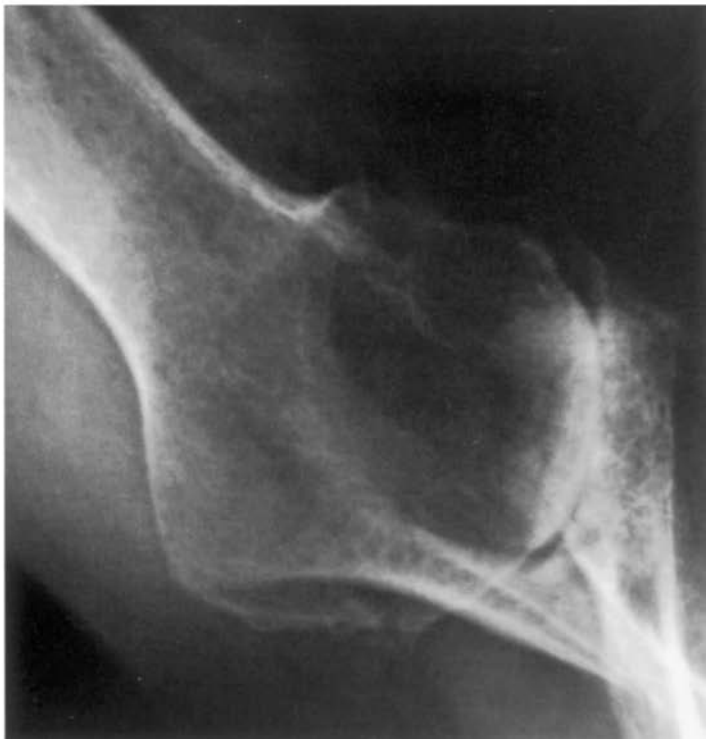


D

Figure 18-36 (*continued*) **(C,D)** More severe involvement with advanced centralization of the joint is noted with generalized loss of the glenoid.

sometimes present, but generally there is a normal white blood cell count. Rheumatoid factor is positive in 75% to 90% of cases.[14,545] Acute-phase reactants (ESR, CRP) may be present and elevated.[23,453] Their usefulness may be more appropriate for monitoring the activity of rheumatoid arthritis.[379] Synovial fluid analysis is confirmatory for inflammation.

Natural History

The rate of progression of rheumatoid disease involvement of the shoulder is unknown. Three main patterns of shoulder involvement have been suggested.[83] These include acute rotator cuff tear, progressive cuff attrition, and medial head displacement. In a few patients, acute rotator cuff tear occurs. This is usually the result of acute synovitis that has involved the synovium adjacent to the insertion of the supraspinatus tendon. The synovium will infiltrate the adjacent tendon, with resultant spontaneous rupture. Progressive cuff attrition is the result of end-stage disease. With progressive muscular and tendinous weakening, there is ascent of the humeral head and diminution in the available acromiohumeral interval. The glenohumeral joint becomes incongruous and

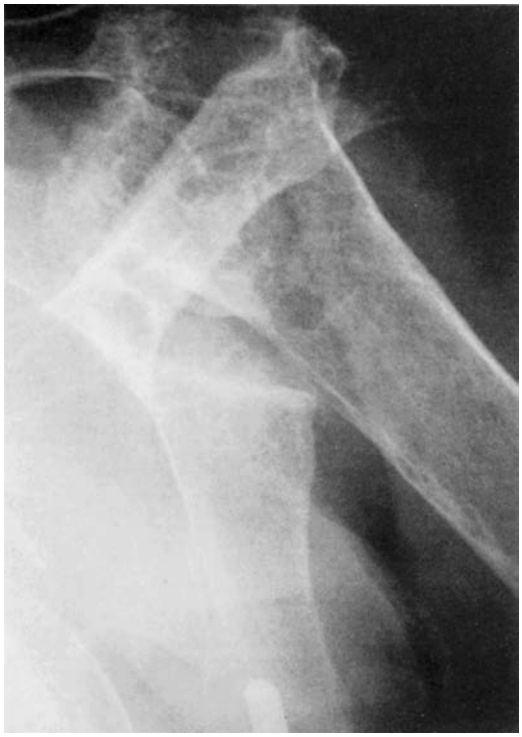


Figure 18-37 Rheumatoid arthritis, end stage, with total distortion of glenohumeral anatomy.

develops progressive erosion. Glenohumeral deformity soon follows. Medial head displacement is rarely an early event. Only when there has been significant cuff attrition and proximal migration does medial head displacement occur. A significant toll is taken on the articular and subchondral osseous tissues. Pannus formation within the glenoid fossa results in a significant loss of supporting bone stock. As the shoulder comes under load, the forces progressively collapse the weakened glenoid. Repair cannot keep pace with the mechanical overload of the weakened glenoid. The humeral head assumes a more medial position as the glenoid gradually disappears.

Neer pointed out the pathologic variations in glenohumeral rheumatoid arthritis.[364] A low-grade type of involvement showed the slow development of articular surface changes and bone loss. An intermediate type was associated with the formation of osteophytes, indicating a response to the ongoing process. In the most severe pattern, there was rapid loss of the articular space and collapse of the underlying bone, with narrowing of the acromiohumeral interval.

Non-Hodgkin's lymphoma has been reported as an unexpected diagnosis made in the surgical specimen of a patient with long-standing rheumatoid arthritis taking immunosuppressive medication and being treated with total shoulder arthroplasty.[16]

CRYSTALLINE ARTHRITIS

Calcium Pyrophosphate Dihydrate Deposition Disease

Calcium pyrophosphate dihydrate (CPPD) deposition disease is a disorder of the articular tissues resulting from the liberation and deposition of CPPD crystals. A prerequisite for diagnosis of the disorder is identification of the deposition of CPPD crystals into fibrocartilage and hyaline cartilage. The relation between CPPD and osteoarthritis is unclear, but notably strong.[131] Unlike gout, there is not a metabolic disturbance.

Incidence

The incidence of clinically symptomatic disease is at least half that of classic gout, with the knee most commonly involved, followed by the wrist, then the shoulder.[104] Clear associations with end-stage secondary osteoarthritis of the shoulder have been reported.[349] Okazaki et al. reported an incidence of up to 50%.[374] CPPD deposition disease does not favor one gender. A familial form is common in the shoulder.[181]

Clinical Evaluation

Two stages of the disorder have been recognized.[109] First is the occurrence of acute attacks of synovitis precipitated by the liberation, accumulation, and reactivity from calcium pyrophosphate crystals within the intraarticular space. Termed "pseudogout," these attacks clinically resemble gout.[109,260] Often the acute events are imposed on a joint with chronic arthropathy. Patients present with an acute severely painful shoulder with motion limitation. Tenderness, swelling, and increased warmth overlying the joint may be appreciated. Fever is not uncommon. If left untreated, symptoms may persist for weeks.

In the second stage, CPPD arthropathy will evolve from repeated attacks and the accumulation of deposition pyrophosphate crystals within the articular cartilage. Secondary degenerative arthritis will develop, accompanied by chronic pain. A propensity for elderly women is recognized with this form.[332]

Pathogenesis

The mechanism of crystal shedding into the joint is unclear. The association of trauma, infection, or illness has not been established, but there may be a predisposition for joints with degenerative changes.[117] McCarty proposed six types: sporadic, pseudorheumatoid arthritis (multiple joints), pseudoosteoarthritis, asymptomatic, chondrocalcinosis, and neuropathic-like.[319] Occurrences at the time of surgery

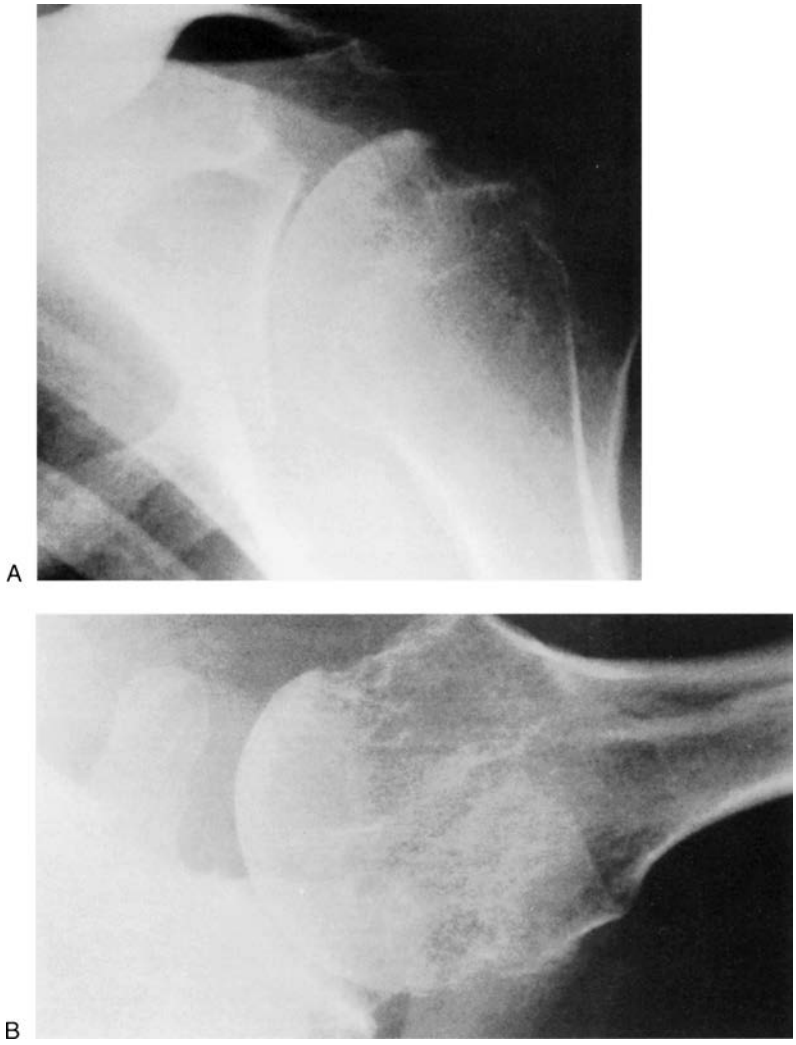


Figure 18-38 **(A,B)** Rheumatoid arthritis: Early disease with preservation of the glenohumeral articular surface; periarticular osteoporosis, particularly in the humeral head.

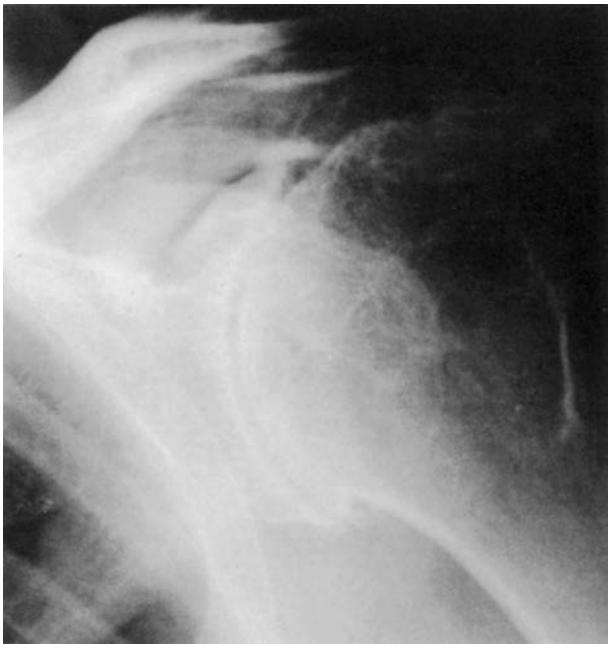


Figure 18-39 Rheumatoid arthritis: Concentric loss of the articular space and osteopenia. Erosions are not prominent.

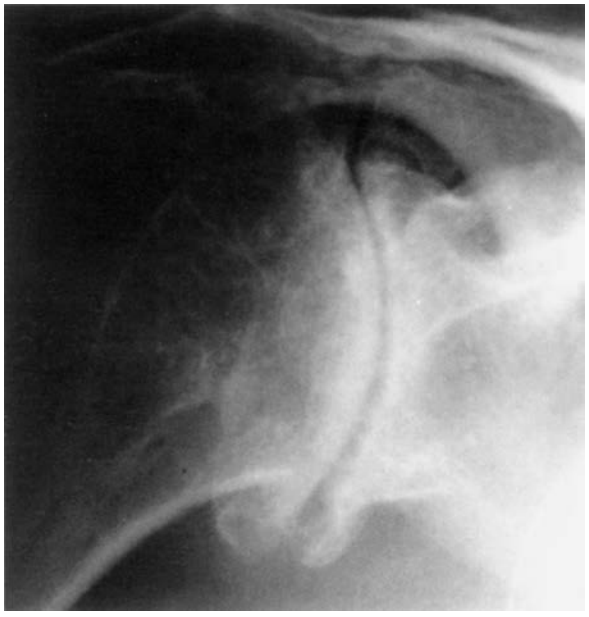


Figure 18-40 Rheumatoid arthritis: Secondary osteoarthritic changes are present.

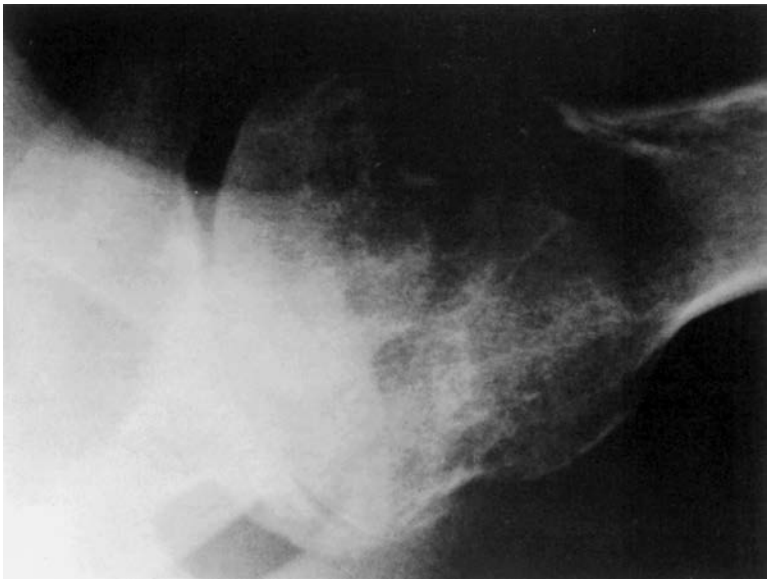


Figure 18-41 Rheumatoid arthritis: The morphology of the humeral head is satisfactorily preserved. There is significant "centralization."

are not uncommon. An increasing incidence with age suggests that the disorder may be a part of the aging process.

Chondrocytes within the articular cartilage possess enzyme systems capable of regulating pyrophosphate and CPPD concentrations within the cartilage.[547] If chondrocyte damage occurs, normal pyrophosphate homeostasis is disturbed. The crystal form accumulates and is liberated into the joint when concentrations exceed a critical threshold. A synovial inflammatory response ensues: Chemotactic factors are released and propagated by polymorphonuclear phagocytosis. Further articular cartilage damage leads to the additional release of calcium pyrophosphate crystals.

Imaging

The disorder may present incidentally on plain radiographs (Fig. 18-43A). Known as chondrocalcinosis, these opacities in articular and fibrocartilage are observed

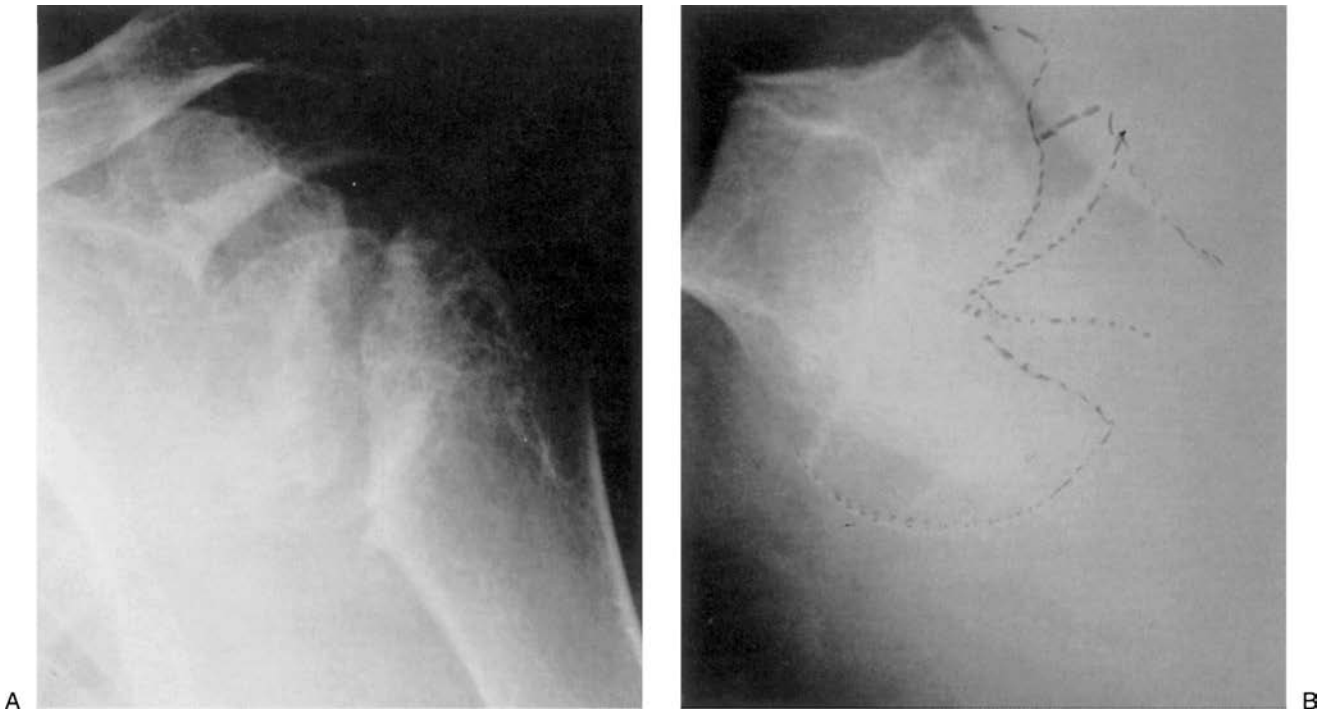


Figure 18-42 **(A,B)** Rheumatoid arthritis: Significant osteopenia is observed. There has been collapse of the humeral head in the absence of significant trauma, in essence, a pathologic fracture dislocation.

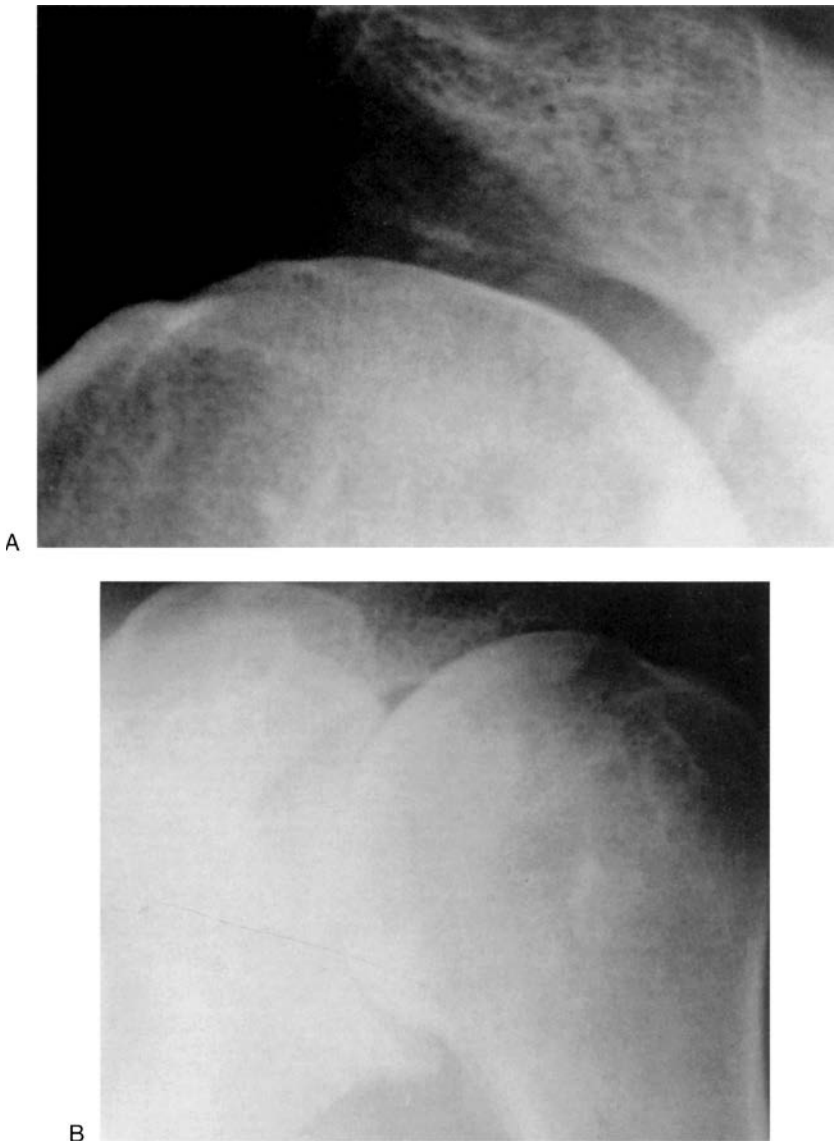


Figure 18-43 Calcium pyrophosphate dihydrate deposition disease: **(A)** Crystal deposition is observed in the articular cartilage of the humeral head. **(B)** Progressive deterioration of the glenohumeral articulation is seen.

in CPPD as well as diabetes, hyperparathyroidism, hemochromatosis, ochronosis, gout, and hypophosphatemia syndromes.[181] Radiographs of CPPD arthropathy are essentially comparable with osteoarthritis (Fig. 18-43B). Particular radiographic features of calcium crystal deposition disorders help to distinguish them from each other and from other arthropathies.[30,486]

Laboratory Findings

Laboratory investigation might include routine screening serum studies for the foregoing disorders, especially if the patient is 55 years or younger with atypical osteoarthritis or polyarticular chondrocalcinosis.[549] Appropriate screening would include serum calcium, alkaline phosphatase, magnesium, ferritin, and thyroid function tests. The diagnosis is made on the basis of synovial fluid analysis. The fluid is often thicker than normal and, at times, turbid, especially in elderly women. At other times, recurrent hemarthroses may exist.[332] It may be indistinguishable from purulent joint fluid. The cell count is representative of inflammatory rather than infectious fluid. Characteristic rhomboid-shaped positively birefringent crystals are identified (Fig. 18-44). Some are intracellular, following engulfment by polymorphonuclear cells. Alizarin red staining enhances the opportunity to identify CPPD crystals in synovial fluid specimens.[551]

Gout

Gout is a disease characterized by hyperuricemia and resultant accumulation of sodium urate crystals within tissues and joints. The clinical manifestations of gout caused by urate crystal accumulations include acute and

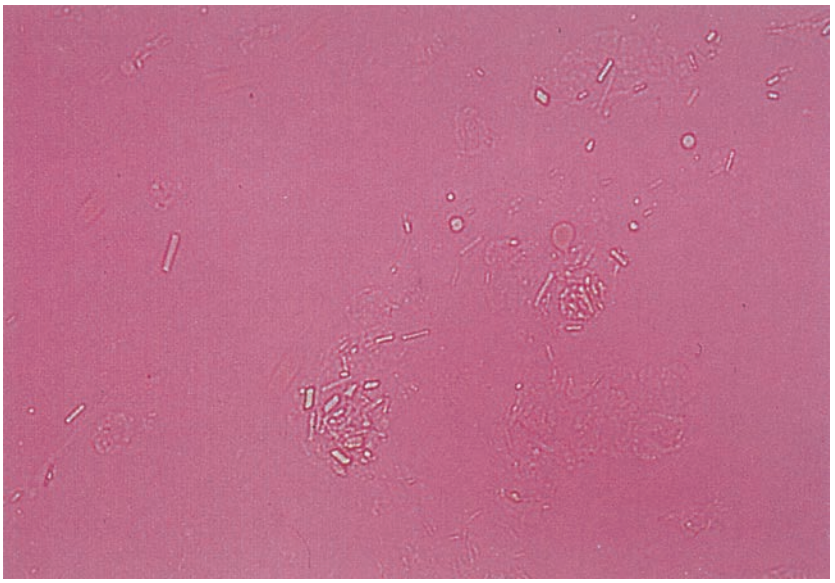


Figure 18-44 Calcium pyrophosphate dihydrate deposition disease: Rhomboid-shaped, positively birefringent crystals.

chronic arthritis, tophi (tissue deposits of crystals), and nephropathy.

Incidence

The incidence is difficult to determine because of the relapsing–recurring nature of the disorder. However, it is believed that the incidence is increasing and is related to changes in dietary habits that lead to the development of the insulin resistance syndrome.[385] Males significantly outnumber females (20:1). It is the most common inflammatory arthropathy in men older than 40 years of age and peaks in the fifth decade.[438] Ninety percent of initial acute attacks of gout are monarticular and rarely involve the glenohumeral joint, unless widespread disease is present.[172,242] In some reports, glenohumeral involvement is not even mentioned. When it is affected, it is rarely early in the disease. It may be the presenting joint in a postoperative flare and in postmenopausal women. Glenohumeral occurrences are much more common in elderly patients, particularly those taking diuretics.[36]

Pathogenesis

Uric acid is the end product of purine catabolism. Humans, as a species, lack the enzyme uricase that governs the degradation of uric acid to allantoin.[75] This conversion is necessary to eliminate the highly insoluble uric acid in the form of the highly soluble compound allantoin. In human tissues, therefore, urate metabolism does not exist, necessitating elimination. Two-thirds of uric acid disposition is by the kidney and approximately one-third is by the gut by bacterial oxidation.[75] Hyperuricemia may be the result of overproduction or undersecretion. Overproduction results from increased synthesis or, much less commonly, alterations in the purine metabolic pathway. The elimination capacity of the kidney is exceeded with supersaturation of serum with uric acid. Up to 90% of cases are the result of deficits of renal excretion.[290]

Most evidence supports the concept of crystal formation in acute attacks of gouty arthritis.[321,461] The mechanisms leading to precipitation of urate crystals are still not well understood. Serum and synovial fluid urate concentrations typically, but not always, are at supersaturated levels.[320] Underlying joint disorders may favor urate crystallization.[495] Proteoglycan concentration, cation (Na^+, Ca^+) concentration, synovial fluid dynamics and pH, intraarticular temperature, and other local factors may play a role.[238,293,471,472,535] Liberation of monosodium urate crystals may also be explained on the basis of tophi present in the synovium.[252] These result from uric acid precipitation caused by increasing circulatory levels. Polymorphonuclear cells invade the synovium and target and then phagocytose the crystals.

Whether the crystal–white blood cell interaction takes place within the joint or within the synovium, a common pathway ensues. The monocytes–macrophages present in the synovial fluid appear to modulate the consequences of the crystal–cell interaction.[385] Mediators of inflammatory response, including chemotactic factors, lysozymes, collagenase, oxygen radicals, and prostaglandins, are released. The synovium is stimulated and proliferates; an acute inflammatory cellular infiltrate rapidly develops. When the joint has been subject to repeated attacks, a local foreign body reaction ensues with the synovium and results in the local resorption of the adjacent cartilage and bone. Radiographically, this is what is seen as the "punched-out" lesions without an associated bony response.

The pathology of the tophi is a chronic foreign body granuloma around a urate crystal. The tophi are often encapsulated. There is an associated inflammatory reaction that involves monarticular cells and giant cells.

Clinical Presentation

With glenohumeral joint involvement, patients will present with an acute onset of shoulder pain. They may previously have had symptoms attributable to gout in another joint. These patients are often obese; most are men and, sometimes, have excessively ingested alcohol. A previous history of renal dysfunction may be obtained, and diuretics may be in use. Occasionally, there is an abrupt reduction in serum uric acid, as might occur with hyperuricemic treatment or the cessation of alcohol ingestion. These factors will result in the dissolution of uric acid from disrupted tophi.[252]

Physical findings of acute gouty arthritis include decreased range of motion of the shoulder, overlying warmth, tenderness, and, perhaps, swelling. Occasionally, these patients will have fever. Chronic gout often exists in the presence of secondary arthritis, the symptoms of which have been discussed earlier.

Imaging

Plain radiographs are usually unremarkable, unless there have been repeated attacks. In such cases, radiographs will show juxtaarticular osteopenia and perhaps juxtaarticular tophi. Sharply outlined erosions, punched out with sclerotic margins and overhanging edges, will be present. Chronic arthropathy from gout results in secondary osteoarthritis and is indistinguishable radiographically (Fig. 18-45).

Laboratory

Only a minor fraction of individuals with elevated serum uric acid levels will develop symptomatic gout. Therefore, the diagnosis cannot be made on the basis of uric acid alone. Sometimes leukocytosis is present. Synovial fluid analysis is diagnostic, with the presence of monosodium urate crystals, often intracellular, visualized with a polarizing microscope. The needle-like crystals are negatively birefringent (Fig. 18-46).

Apatite Deposition Disease

This disorder, sometimes known as apatite gout, has been described by many authors.[106,144,236,317,456] It is associated with the presence of intraarticular hydroxyapatite crystals. The link between the crystals, arthropathy, and osteoarthritis is unclear. There is a predisposition for large joints. McCarty et al. popularized the condition as "Milwaukee shoulder."[317]

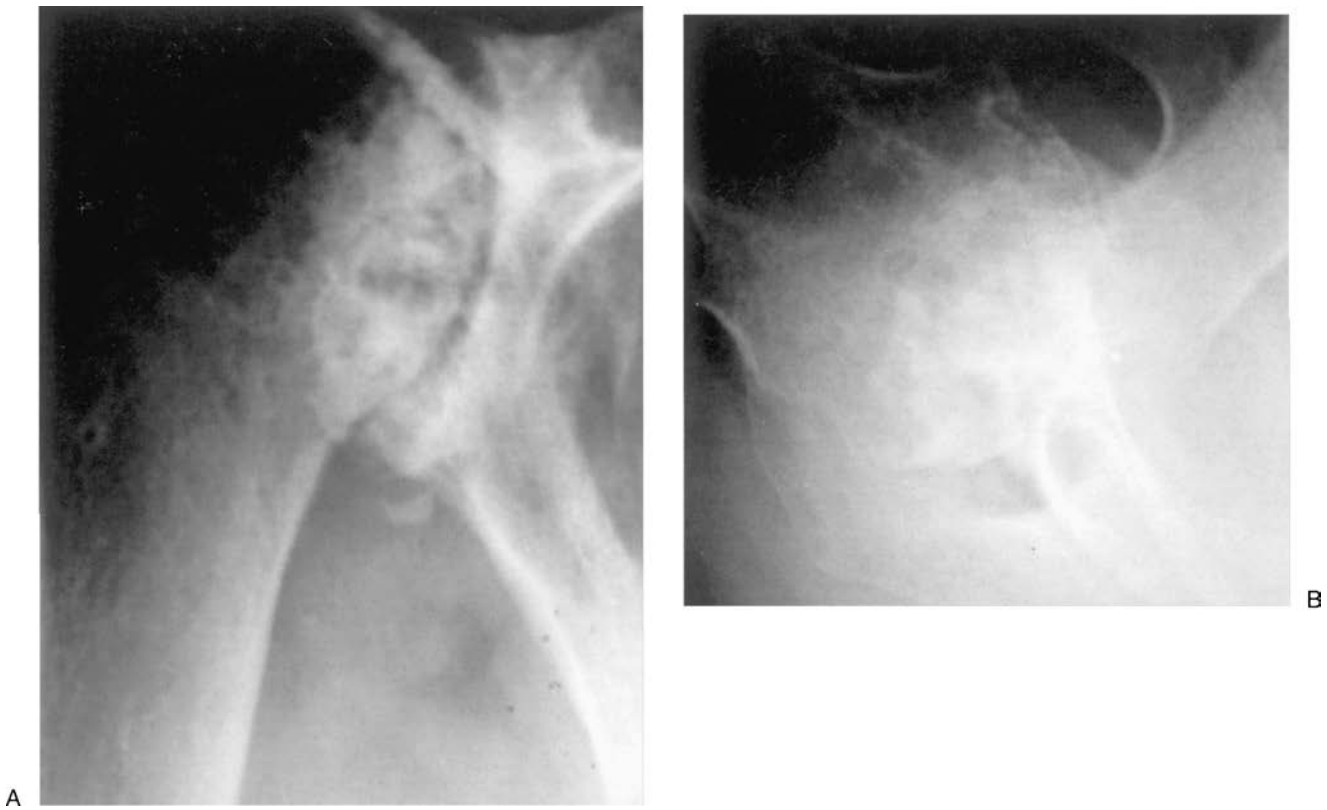


Figure 18-45 **(A,B)** Gout: Arthropathy with loss of glenohumeral joint cartilage and subchondral cyst formation.

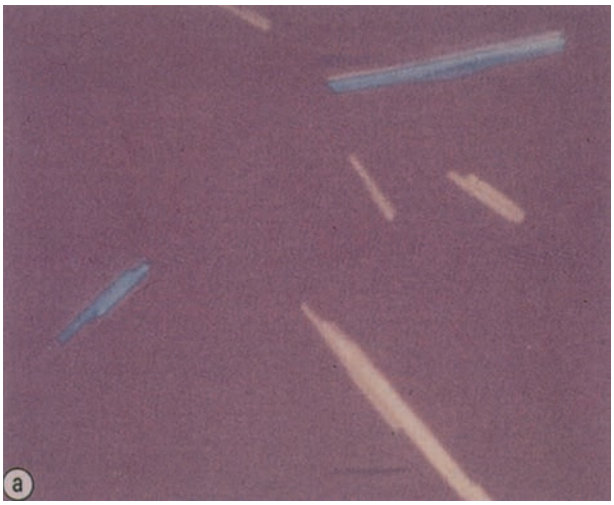


Figure 18-46 Gout: Monosodium urate crystals—negatively birefringent.

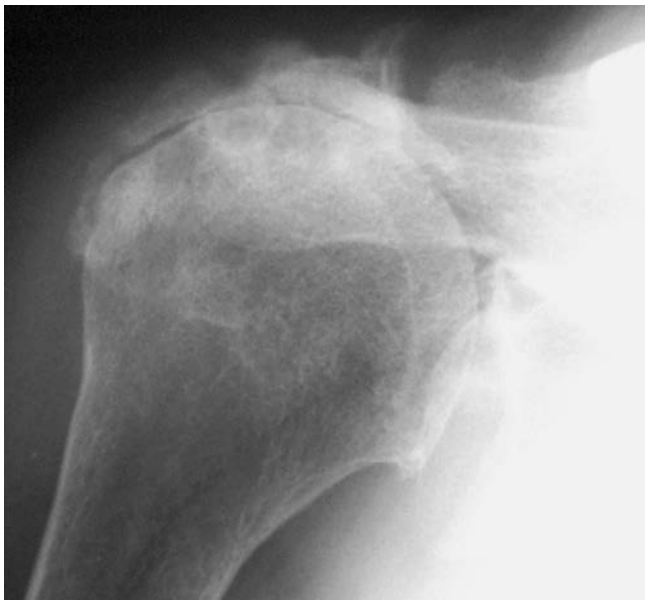


Figure 18-47 Rotator cuff tear arthropathy. Of note is the superior subluxation of the humeral head with obliteration of the acromiohumeral interval. The joint has incurred secondary osteoarthritic changes.

It typically involves the shoulder and occurs in older women.[60] Hydroxyapatite crystals do not excite the same degree of synovial response as do sodium urate crystals.[60] As a result, over a period of weeks or months, chronic mild discomfort typically evolves. Physical examination is characterized by a boggy effusion and limitation of motion. This is associated with radiologic soft tissue calcification, ascent of the humeral head, and destruction of the glenohumeral relations. Joint fluid often contains debris as well as calcium crystals, but relatively few inflammatory cells, predominantly monocytes.[105] Blood-stained fluid is common.[317] The effusion may rupture into the adjacent soft tissue planes, creating additional symptoms and signs, sometimes interpreted as venous occlusive disease.[546] A rotator cuff tear is common. The calcium hydroxyapatite crystals are not seen by light microscopy because of their size. They require alizarin red S staining of the synovial fluid preparation.[194] Electron microscopy may be more reliable, but is generally less available. Synovial collagenases and neutral proteases are present in high concentrations, indicative of underlying enzymatic action.[177]

Further discussion will be given to this entity in the next section.

ROTATOR CUFF TEAR ARTHROPATHY

A special condition originating in the tendinous socket of the shoulder, which leads to arthritic changes in the glenohumeral joint, has been recognized clinically for over a century (Fig. 18-47).[2,3,477,478] It is characterized by recurrent, often hemorrhagic, effusions containing biochemically active enzymes; the complete absence of the rotator cuff; advanced degenerative glenohumeral joint changes with humeral head collapse; and erosion of the acromion process, the acromioclavicular joint, and the distal clavicle. Codman made reference to a case of rotator cuff tear with hygroma.[70] McCarty and associates in 1981 recognized this clinical entity and it became popularized as the Milwaukee shoulder.[144,178,317] Neer and others in 1983 recognized a subset of patients with similar clinical and radiographic findings and designated the lesion "cuff arthropathy."[354]

The pathogenic role of calcium phosphate crystals (hydroxyapatite) in the deterioration process was touted by McCarty and continues to draw interest.[179,317] Neer, on the other hand, believed that glenohumeral instability resulted from the massive rotator cuff tearing, with resultant impaired cartilage nutrition, bone softening, collapse, and bone destruction.[354,362] Its genesis continues to be a source of debate and speculation, but essential elements of each theory are often present within the same shoulder.

Milwaukee shoulder is also known as apatite gout and has been mentioned earlier. Forming in the synovium and articular cartilage, hydroxyapatite, octacalcium phosphate, and tricalcium phosphate crystals, whose origin is unknown, are released into the joint fluid.[323] A phagocytic response is induced, leading to the release of matrix-degrading metalloproteinases, collagenase, and stromelysin and subsequent destruction of the rotator cuff and articular cartilage. With further destruction, the cycle is propagated. It is characteristically found in older female patients. Often, their symptoms are bilateral and have been chronic for many years, with periodic exacerbations of more

intense discomfort. Physical examination is remarkable for atrophy, weakness, and shoulder effusion. Synovial fluid analysis by electron microscopy or alizarin red is diagnostic but difficult, and usually not necessary. Imaging studies will demonstrate full-thickness rotator cuff tears.

The destruction of the osteoarticular elements of the shoulder appears to result from the progressive formation of a massive, irreparable, rotator cuff tear and its mechanical sequelae.[79,309,354] Joint debris and biomechanical products of an active synovial lining also play a role in disease progression. Their presence is the likely result of glenohumeral instability, a consequence of losing rotator cuff integrity.

Progressive narrowing of the acromiohumeral interval by ascent of the humeral head is a prominent feature of rotator cuff arthropathy. With the loss of the stabilizing effect of the rotator cuff, the humeral head displaces upward.

On the basis of their anatomic positioning, the musculotendinous units of the rotator cuff serve as glenohumeral joint compressors, as opposed to acting as humeral head depressors. The force generated by the muscles pulls the humeral head into the concavity of the glenoid and serves as its primary stabilizer. The creation of this efficient fulcrum enables the humeral head to resist the upward force vector of the contracting deltoid muscle. By this mechanism, the hand can be placed overhead or away from the body. An entirely intact rotator cuff is not necessarily required; many patients are capable of overhead mobility with full-thickness defects. The arm can be elevated, as long as there is sufficient muscle activity for compressive stabilization of the humeral head.

A secondary humeral head–stabilizing system includes the acromion, coracoid process, coracoacromial ligament, and distal clavicle. This coracoacromial archway is the final functional restraint to superior humeral head migration, when the primary stabilizing system has failed.[283]

Pathophysiology and Pathoanatomy

Muscular weakness and degenerative tendon fiber failure are age-related changes that inevitably occur within the rotator cuff.[71,311] Initially, only the supraspinatus tendon is involved. Eventually, further interstitial tendon fiber failure, often clinically interpreted as tendonitis, may lead to partial- and, later, full-thickness tears of the supraspinatus. With continuing activity, the tear may enlarge as adjacent fibers are loaded beyond their elastic limit, and it predictably extends to involve the infraspinatus and, later, the subscapularis. Subtle superior humeral head migration occurs as the concavity compression forces diminish. Initially, this phenomenon is satisfactorily tolerated in terms of comfort and presents little or no functional deficit.

Once the superior forces have exceeded the medially directed forces, abrasive contact occurs against each side. Impingement of the rotator cuff tissue against the acromion accelerates the loss of tendinous tissue. When the cuff tendon tissue superior to the humeral head axis of rotation has vanished, contracture of the lower half of the cuff and the deltoid will create a fixed "boutonniere" deformity of the humeral head against the coracoacromial arch.[312] As a result of the increased friction against the nonarticular and nonconforming undersurface of acromion, the articular surface of the humerus begins to wear.

In response to increased pressure, tensioning of the coracoacromial ligament may result in the formation of an anterior or inferior acromial traction spur. This bony projection may potentially gouge the underlying tendinous tissue and articular cartilage, inducing further damage. In response to load, the acromion process develops the characteristic changes of eburnation and erosion, whereas the coracoacromial ligament may hypertrophy. Stress fractures of the acromion process resulting from unremitting load by the upwardly thrust humerus have been reported (Fig. 18-48).[98] When the primary stabilizers have failed, limited although purposeful function may be maintained, in spite of the unopposed action of the deltoid, by the secondary stabilization within the coracoacromial arch.[283] Although initially adequate, further articular cartilage degradation and further rotator cuff tendon damage are incurred.

This disorder does not spare the glenohumeral articulation. As a failure of containment, the humeral head subluxes superiorly with force concentration on the narrower

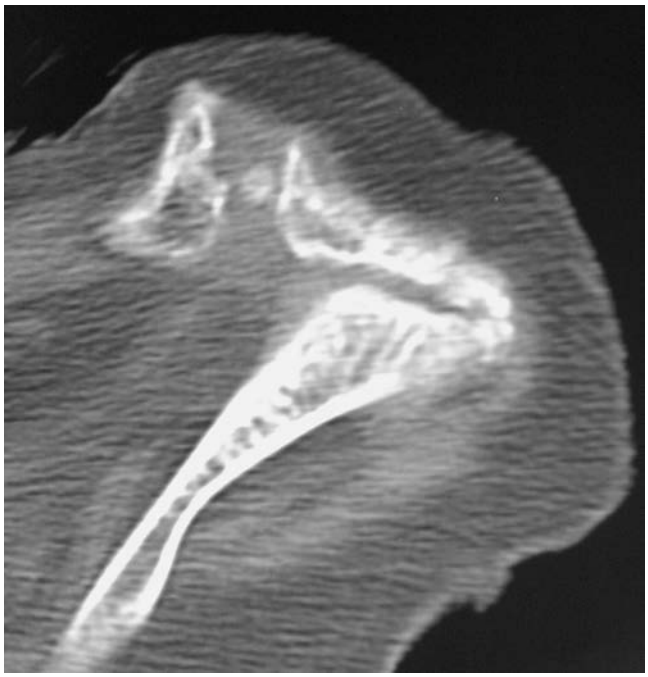


Figure 18-48 Acromial insufficiency fracture due to extreme overloading of the unyielding acromion process by the upwardly displaced humeral head.

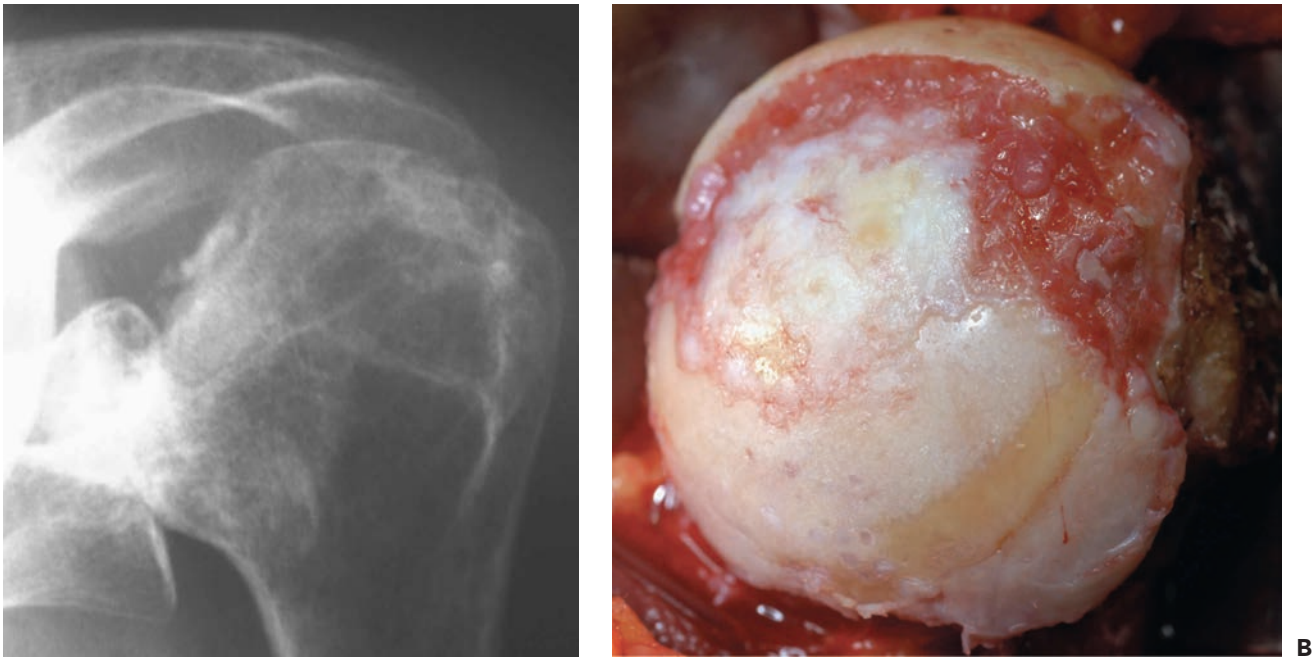


Figure 18-49 **(A)** The superiorly subluxed humeral head manifests collapse and superior glenoid and acromial humeral erosion along with early secondary osteoarthritis. **(B)** The gross specimen at the time of humeral head replacement demonstrating collapse, erosion of the articular cartilage with reparative granulation tissue, and eburnated bone.

superior glenoid. An even greater demand is placed on the primary and secondary stabilizers. Articular surface wear is accelerated and is soon followed by a collapse and degradation of the underlying subchondral bone of the humeral head.

When articular cartilage fragments and underlying areas of softened, and often nonviable, bone are exposed, particulate debris is shed into the joint (Fig. 18-49).[177,317] In response, the synovium proliferates with the development of large effusions, often contained only by a weakened, and sometimes surgically altered, deltoid muscle. "Subcutaneous effusions" may evolve as the fluid herniates through the soft tissue pathways of least resistance. Sometimes, the fluid accumulates superior to the acromioclavicular joint, recognized as the "geyser sign" on shoulder arthrogram.[81]

The effusions may be predominantly bloody.[24,141,218,322,475,546] The source of bleeding may be a crater in the humeral head resulting from impingement on the medial acromion process.[447]

Rotator cuff arthropathy is not likely an obligate end stage of the so-called impingement syndrome and rotator cuff tendon failure. Neer et al. stated that only 4% of rotator cuff tears will progress to develop cuff tear arthropathy.[354] From a purely mechanical basis, as long as the humeral head can be maintained concentrically opposed to the glenoid by the balanced action of residual intact columns of the anterior and posterior cuff, the shoulder is protected from the requisite instability that may lead to rotator cuff arthropathy.[52] A limited degree of superior subluxation of the humeral head can apparently be tolerated without progressively erosive changes of the bony surfaces. Certain rotator cuff tears may be at risk for progression to arthropathy if elevated crystal levels are identified in the joint fluid.[12]

Clinical Evaluation

When presenting for evaluation, patients with rotator cuff arthropathy complain bitterly of unrelenting pain. For years before the acute and intolerable exacerbation, the disorder has been in slow evolution with periodic exacerbations. Not only is the pain felt in the depth of the shoulder, but it is often referred to the deltoid insertion. Weakness, crepitation, and sudden sharp pain often limit the attempted use of the extremity for lifting or overhead activities. Significant night pain exists. Minor traumatic experiences are common, and patients will often deny a history of major shoulder injury. Although symptoms may exist bilaterally, one shoulder is often more significantly affected. One or more arthrocenteses accompanied by corticosteroid instillation may have been performed. The typical patient is a woman and is older than 70 years of age. In 60% of cases, the disorder will be bilateral.[223] In some instances, the family history is curiously positive for the same disorder among the older generations.[400]

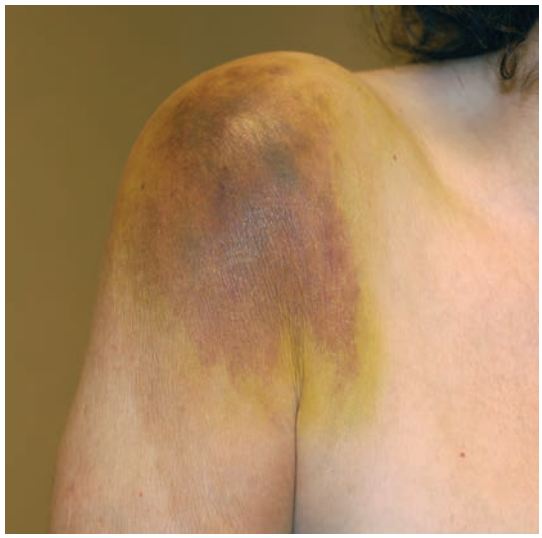


Figure 18-50 The characteristic ecchymotic appearance of the cutaneous manifestation of recurrent shoulder hemarthrosis. Intense staining of the tissue with the products of blood breakdown may persist long after resolution of the acute ecchymosis.

The disorder is characterized by physical findings indicative of the chronic nature of the disorder. Atrophy of the spinati and, sometimes, the deltoid musculature is prominent. Sometimes, a large effusion will obscure the presence of atrophy. The effusion may contribute to the formation of a unique dermatitis.[113] The localized vesicular rash is created by the subcutaneous seepage of "dermal mucin" in the absence of frank fistula formation. Recurrent hemarthrosis may result in an ecchymotic appearance of the shoulder and upper brachium (Fig. 18-50). A superiorly subluxed humeral head may rest directly beneath the deltoid muscle anterior to the acromion process. Profound weakness results in a significant restriction in active range of motion. Restraints to passive movement include pain, contracture, and glenohumeral incongruity. Invariably, crepitation, arising from many different sources, often indistinguishable, will be present. The physical findings may exist bilaterally.

Imaging

Sophisticated imaging studies are unnecessary to diagnose rotator cuff tear arthropathy or to adequately identify the pathology, especially in its later stages (Fig. 18-51).[79] Plain films, which may include the weighted abduction Grashey's view, represent the most cost-effective means of evaluation, as well as the most accurate (Fig. 18-52). Osteopenia, superior migration of the humeral head, narrowing or obliteration of the acromiohumeral interval, acromial erosions, and superior glenoid bone loss are the characteristic findings.[446] The humeral head may be collapsed, a finding felt by Neer to necessary for the diagnosis of cuff tear arthropathy (Fig. 18-53).[354] Osteophytes, irregular cystic formation, subchondral sclerosis, and loss of tuberosity prominence may also be seen. The glenohumeral joint takes on the appearance of the hip.[313] "Acetabularization" results from sculptured erosion of the acromion, superior glenoid, and coracoid bone and contact of the humeral head against the coracoacromial arch.[313] Likewise, reciprocal "femoralization" of the proximal humerus includes tuberosity round-off and loss of the bicipital groove, resulting in a large spherical head.[313] The findings on MRI are often dramatic but simply reiterate the findings noted on the physical examination and plain radiographs (Fig. 18-54).

Visotsky et al. presented Seebauer's analysis of the radiographs of patients with cuff tear arthropathy in which Seebauer recognized variations that implied biomechanical differences among shoulders with cuff tear arthropathy and conceived a radiographic classification for cuff tear

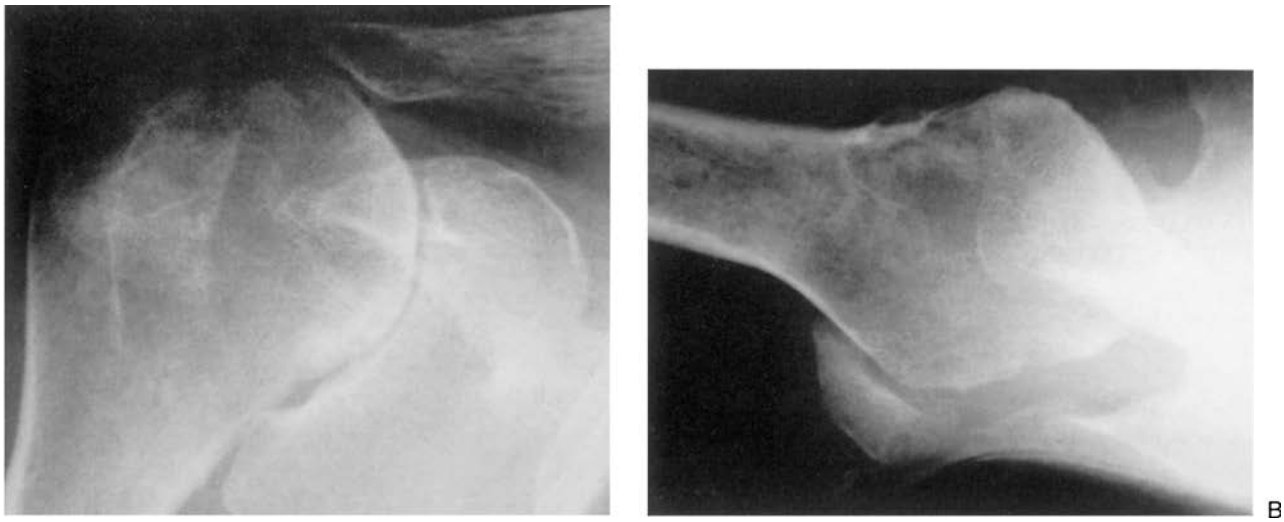


Figure 18-51 **(A,B)** End-stage rotator cuff arthropathy with significant superior glenoid erosion into the acromioclavicular joint and insufficiency fracture of the acromion process.

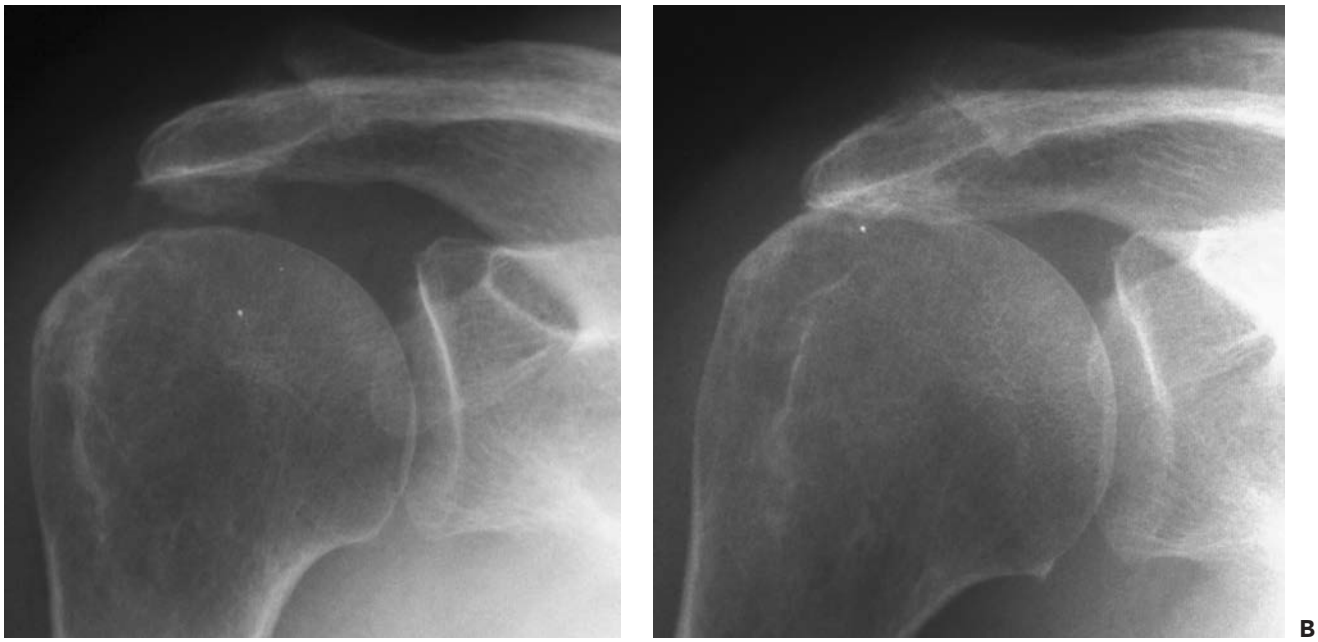


Figure 18-52 **(A)** Grashey's view. **(B)** Weighted abduction Grashey's view. The acromiohumeral interval is obliterated as the humeral head ascends as a result of muscle imbalance due to rotator cuff tear.

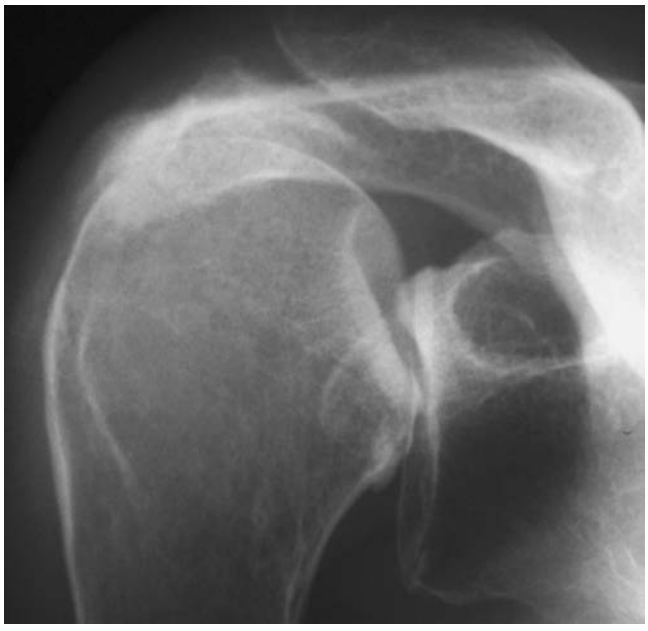


Figure 18-53 Rotator cuff tear arthropathy with humeral head collapse.

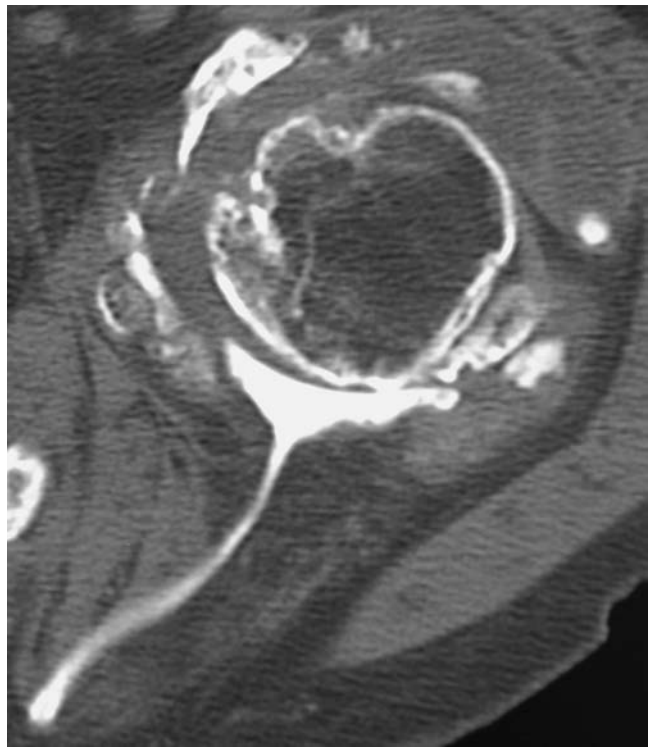
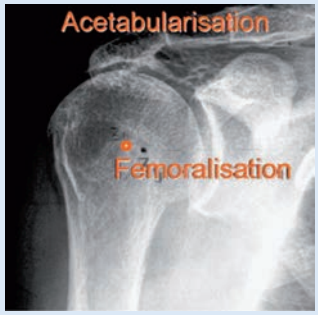
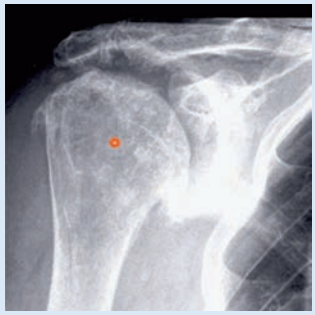
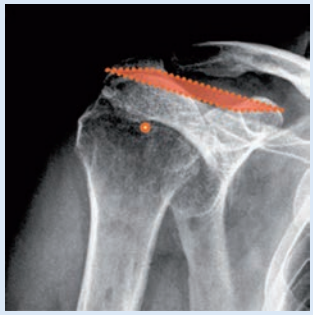
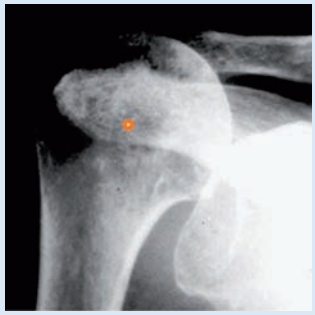


Figure 18-54 Magnetic resonance imaging of advanced rotator cuff tear arthropathy. There is loss of articular cartilage, disruption of normal anatomic soft tissue planes, and erosion of the coracoid process with disintegration of the humeral head and glenoid.

TABLE 18-4
SEEBAUER CLASSIFICATION FOR ROTATOR CUFF TEAR ARTHROPATHY

Type IA-Centered Stable	Type IB-Centered Medialized	Type IIA-Decentered Limited Stable	Type IIB-Decentered Unstable
			
• Intact Anterior Restraints • Minimal Superior Migration • Dynamic Joint Stabilization • Acetabularization of CA Arch and Femoralization of Humeral Head	• Intact Anterior Restraints – Force Couple Intact/Compensated • Minimal Superior Migration • Compromised Dynamic Joint Stabilization • Medial Erosion of the Glenoid, Acetabularization of CA Arch, and Femoralization of Humeral Head	• Compromised Anterior Restraints—Compromised Force Couple. • Superior Translation • Insufficient Dynamic Joint Stabilization • Minimum Stabilization by CA Arch, Superior–Medial Erosion and Extensive Acetabularization of CA Arch and Femoralization of Humeral Head.	• Incompetent Anterior Structures • Anterior–Superior Escape • Absent Dynamic Joint Stabilization • No Stabilization by CA Arch—Deficient Anterior Structures.

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arthropathy based on the location of the center of rotation of the humeral head, the sufficiency of restraining structures, and the degree of instability (Table 18-4).[521] The classification may prove to be a useful tool in the treatment decision making for rotator cuff tear arthropathy.

Laboratory

Synovial fluid analysis is usually not necessary. If it is performed, the fluid is often blood streaked or straw-colored. Routine demonstration of calcium phosphate crystals is extremely difficult, requiring special staining techniques or electron microscopy.

OTHER ARTHROPATHIES

Rheumatoid Arthritis of the Elderly

This condition presents typically in older persons (Fig. 18-55). Involvement is most often confined to the shoulders and wrists. Over a 4- to 6-month course, it can become very disabling. The rheumatoid factor is negative and ESR

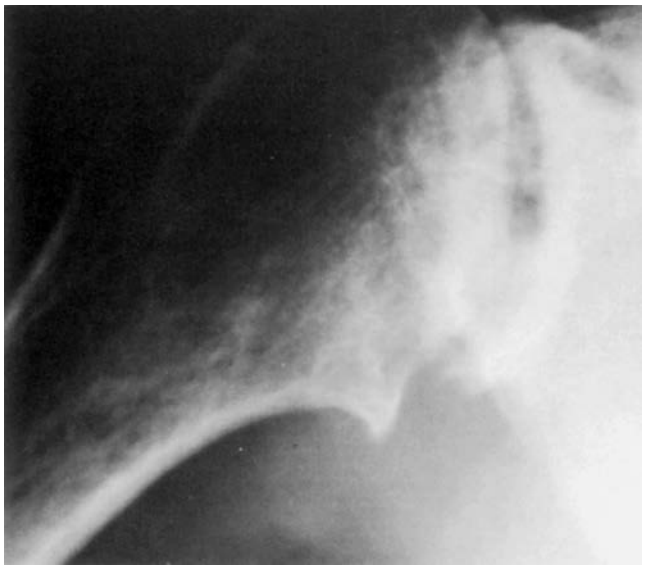


Figure 18-55 Rheumatoid arthritis of the elderly.

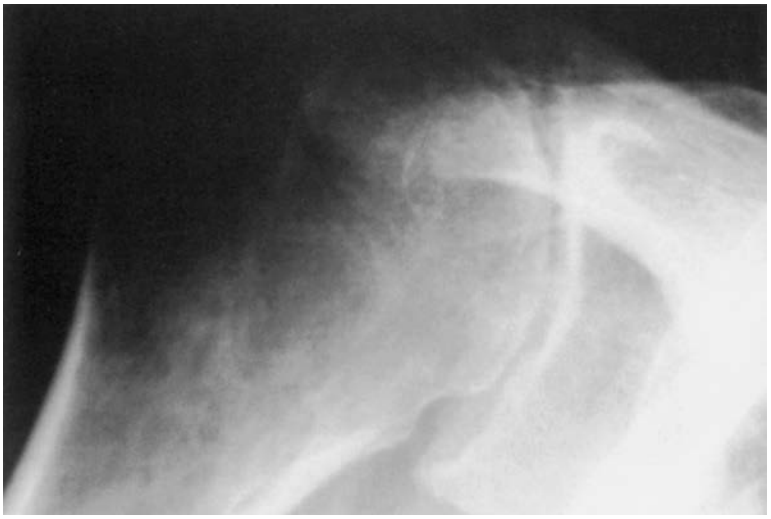


Figure 18-56 Adult-onset Still's disease with distortion of the glenohumeral articulation and moderately severe glenohumeral arthritis.

is elevated. A significantly higher frequency of shoulder involvement is observed in elderly-onset rheumatoid arthritis (more than 60 years old).[22]

Juvenile Rheumatoid Arthritis

The shoulder is not typically involved early in the various forms of juvenile rheumatoid arthritis (JRA). When juvenile spondylitis or the polyarticular form exists, shoulder involvement is more likely, up to 25%. It is less common in the pauciarticular form. Typically, when the patients are seropositive, a rapidly destructive and severe glenohumeral involvement can be observed.[437]

Adult Still's disease, a rare inflammatory disease accompanied by polyarthritis with features similar to the systemic form of JRA, may affect the shoulder in up to 43% of cases at the time of presentation (Fig. 18-56).[90,97,404,548] Thirty percent to 50% of patients with more chronic arthropathy will have glenohumeral involvement.[281,404]

Rheumatoid Syndrome

Neer identified a select group of patients who manifested symptoms, signs, and radiologic changes consistent with rheumatoid arthritis.[366] Members of this group were almost exclusively women between the ages of 35 and 55. The involvement spared other joints and was almost always confined to the shoulder. Bilaterality was common; involvement was of the dry low-grade type. The rheumatoid factor was positive. Radiographic changes include glenohumeral joint space loss, cysts, and sclerotic changes (Fig. 18-57). Progressive disease over a short time was observed.

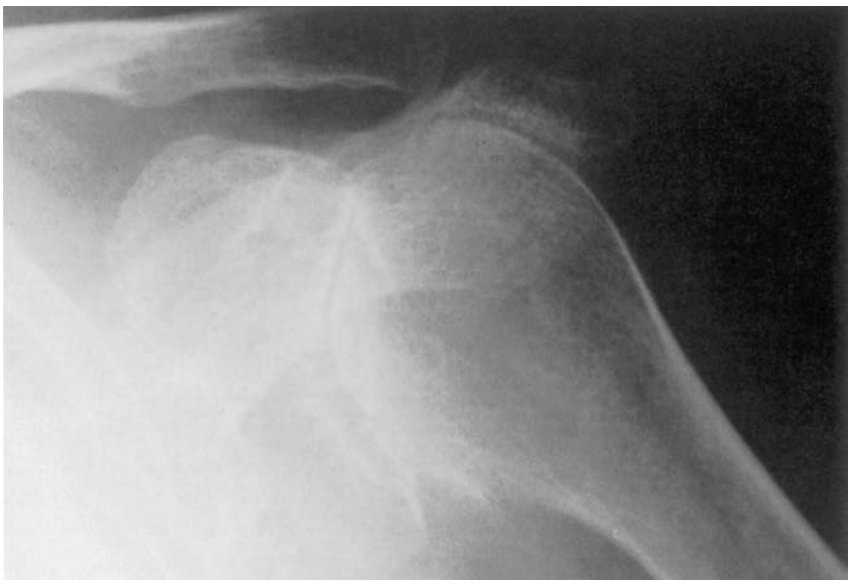


Figure 18-57 Rheumatoid syndrome with a "dry-type" appearance to the glenohumeral articulation.

Rapidly Destructive Articular Disease

Rapidly destructive articular disease (RDAD), an unusual form of arthropathy recognized most often in the hip, has been suspected of causing a similar condition in the shoulder.[33,58,255,282,368,434,443,504] It is most commonly observed in elderly females. Osteoporosis-induced subchondral insufficiency fracture may play a significant role in the pathogenesis of the disorder.[504,552,553] It is similar radiographically, in its final stages, to rotator cuff tear arthropathy and Milwaukee shoulder (Fig. 18-58). Sometimes, RDAD shares certain gross, microscopic and synovial fluid features of those disorders.[58,255,282] Crystals or large cuff defects, as well as the typical findings of osteonecrosis and osteoarthritis, may be notably absent.[504]

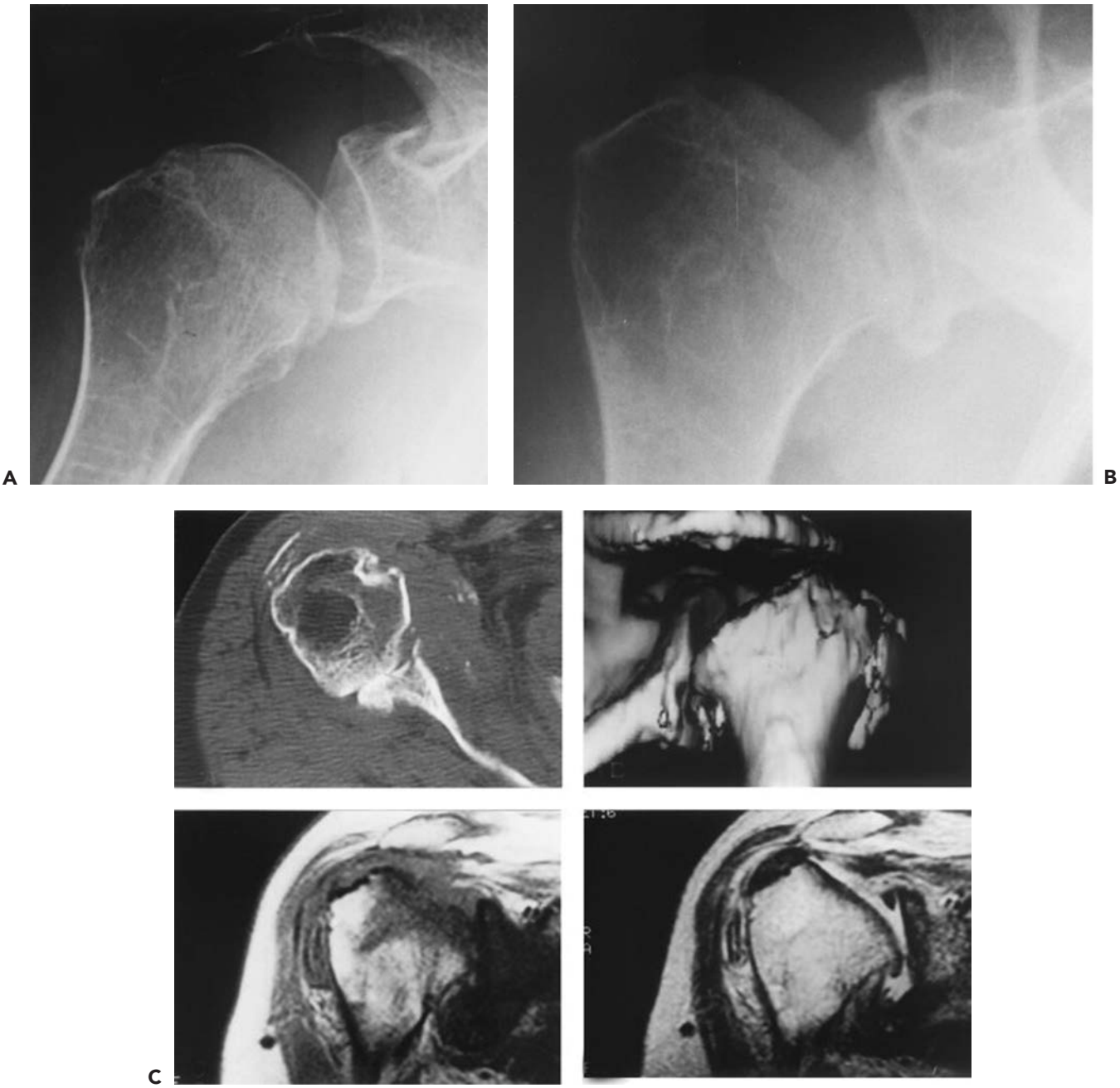


Figure 18-58 **(A)** Rapidly destructive articular disease at the time of presentation. **(B)** Three months later. **(C)** Advanced destruction well depicted with computed tomography (CT), three-dimensional CT, and magnetic resonance imaging.

Dialysis Arthropathy

As many as 50% to 69% of patients receiving treatment with long-term dialysis for end-stage renal disease experience musculoskeletal problems.[26,65,420] The most common of these is "dialysis arthropathy" and may affect the shoulder up to one-third of the time.[271,465] The extent of involvement parallels the duration of dialysis and are manifest earlier in older patients.[271] Both end-stage renal disease and the process of dialysis contribute to the pathophysiology of dialysis arthropathy. Most frequently implicated is amyloid depositions, oxalosis, nonbacterial infections (including hepatitis C), apatite crystal deposition, and iron or aluminum overload.[26,133,271,420,465,469] Clinical manifestations are painful, diminished range of motion. Early in the course of the disease, even including patients requiring surgical treatment, the plain radiographic findings are unremarkable.[78] At this time, MRI will demonstrate synovial proliferation. Periarticular bone cysts, articular erosions, and destructive spondyloarthropathy are often late findings, even persisting after successful renal transplantation (Fig. 18-59).[27,420,465]

Spondyloarthropathy

Spondyloarthropathies are a group of environmentally triggered diseases identified in those who are genetically

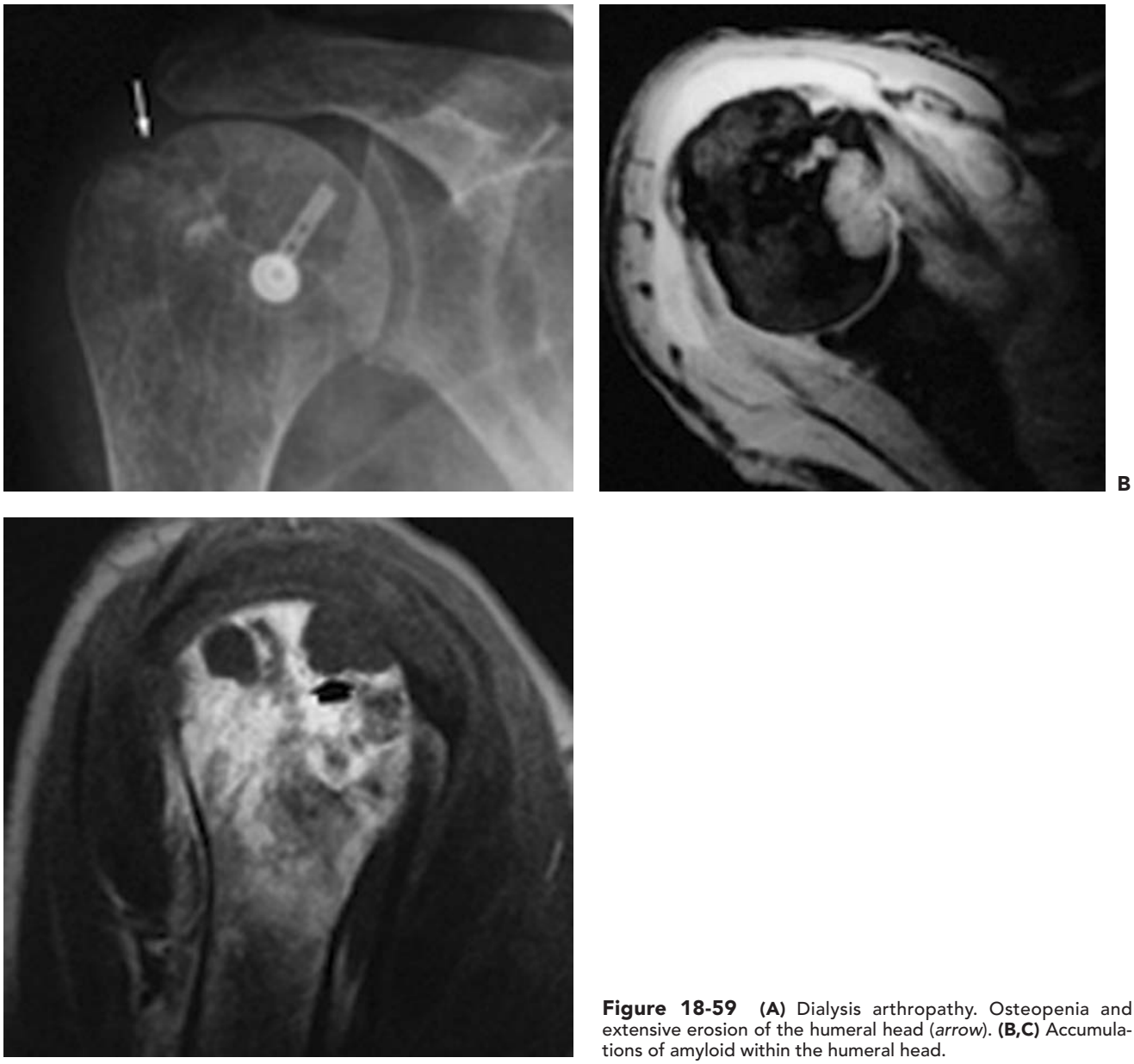


Figure 18-59 **(A)** Dialysis arthropathy. Osteopenia and extensive erosion of the humeral head (*arrow*). **(B,C)** Accumulations of amyloid within the humeral head.

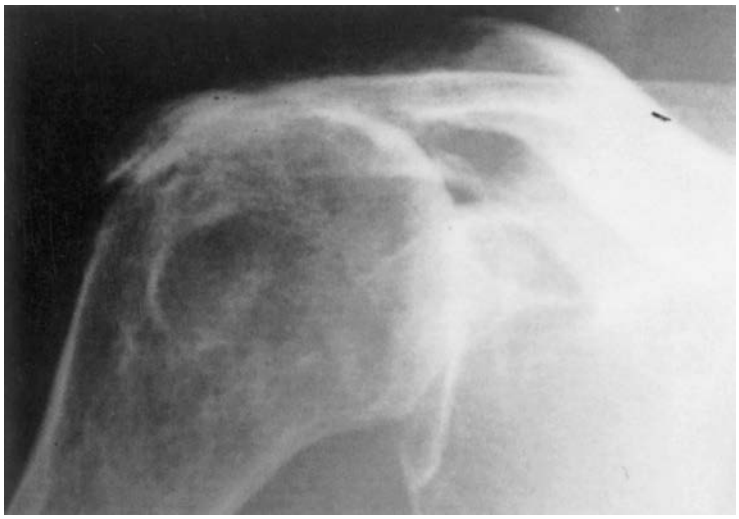


Figure 18-60 Ankylosing spondylitis with accompanying loss of the rotator cuff.

predisposed.[249] The axial joints are typically involved, including the shoulder. Involvement of the shoulder typically occurs somewhat early in the disease process. Entities such as psoriatic arthritis, enteropathic arthritis, and ankylosing spondylitis are known to involve the shoulder.[139]

In ankylosing spondylitis, the frequent involvement of the hip and shoulder (50%), although clinically mild, follows the appearance of spinal disease.[123,248,305] Shoulder symptoms and loss of shoulder mobility are common but not frequently disabling.[537] Sometimes, the first symptoms are in the shoulder. Bilateral involvement is common. The radiographic changes include osteoporosis, joint space narrowing, and bone exostosis (Fig. 18-60).[276,423] The "hatchet sign," a characteristic destructive abnormality, involves the superior lateral aspect of the humeral head.[432] A rotator cuff tear often accompanies this lesion.

Psoriatic arthritis involves the shoulder in 30% to 50%.[500] A rapidly destructive form of glenohumeral change may occur.

Reiter's disease may present as an acute arthritic attack, often following an enteric or urogenital infection. There may be an associated fever. There appears to be some genetic susceptibility. Acute shoulder pain will occur in 8%.[519] Seventy percent of patients are HLA-B27 positive. The shoulder is not involved to the extent of the subtalar, ankle, and hip joints. If the attack of Reiter's disease is protracted, patients are more likely to have shoulder involvement.[519] Clinically, this cannot be distinguished from other seronegative arthropathies.

Enteric arthritis parallels the activity of the inflammatory bowel disease.[198,338] Shoulder involvement occurs in 5% to 10% and is usually associated with sacroiliitis and spondylitis.

Neuropathic Arthropathy

Neuropathic arthropathy may affect the shoulder.[25,187] Syringomyelia is the most common cause; arthropathy occurs in 20% to 40% of patients with the disease, the shoulder more often than the elbow.[55,538] Other causes are diabetes, syphilis, and peripheral neuropathies.[9] The pathogenesis is uncertain but favors the theory of "neurotrauma," whereby joint destruction ensues in response to altered sensory innervation of the joint, and the theory of "neurovascular," whereby the neurologic changes of the underlying disease induce hypervascularity within the subchondral bone with resultant bone atrophy, subchondral bone collapse with disorganization of the joint.[9] Mild pain and neurologic symptoms and signs are usually present.[187] Deformity secondary to instability is sometimes present. More often, the physical signs are subtle (Fig. 18-61). The imaging data varies with the course of the disease. Plain radiographs may suggest glenohumeral instability, often confirmed with CT scanning (Fig. 18-62). Advanced imaging, at this point, is often dramatic (Fig. 18-63). With time, the joint is unrecognizable (Fig. 18-64).

Hemophilia Arthropathy

Glenohumeral arthropathy has also been observed as a sequela to hemophilia.[15,54,126,300] Properties of the synovium are pivotal in the pathophysiology with invasive capacity not unlike rheumatoid arthritis or pigmented villonodular synovitis.[296] Blood and products of blood metabolism are the pathologic agents of the disorder.[431,485] Hemosiderin deposition in the synovium induces inflammatory hypertrophy, which inevitably leads to arthropathy

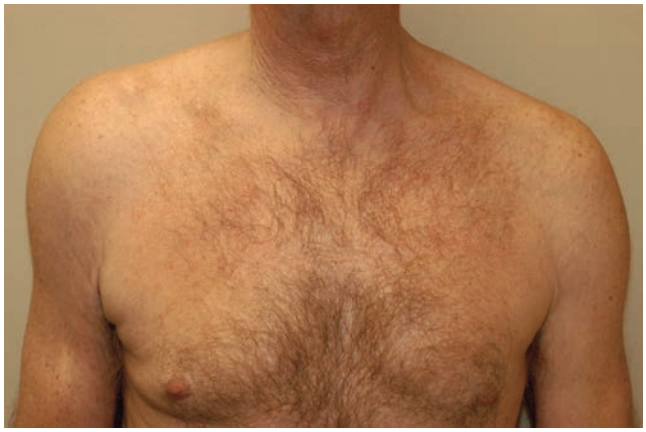
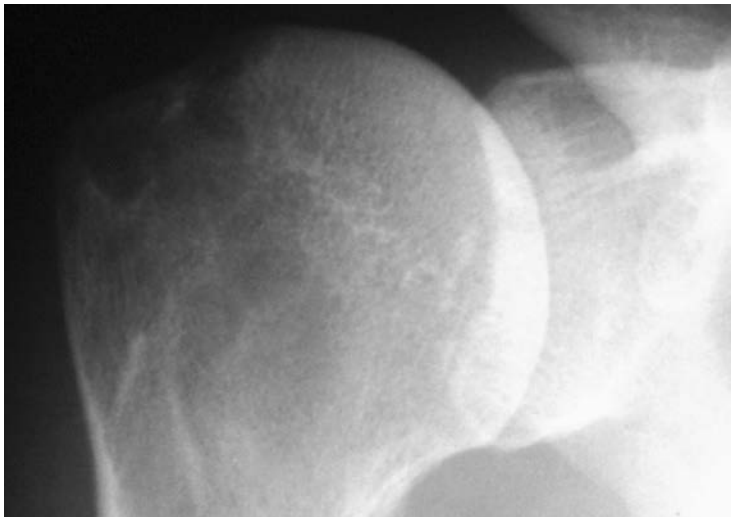


Figure 18-61 Neuropathic arthropathy. The right shoulder is swollen.



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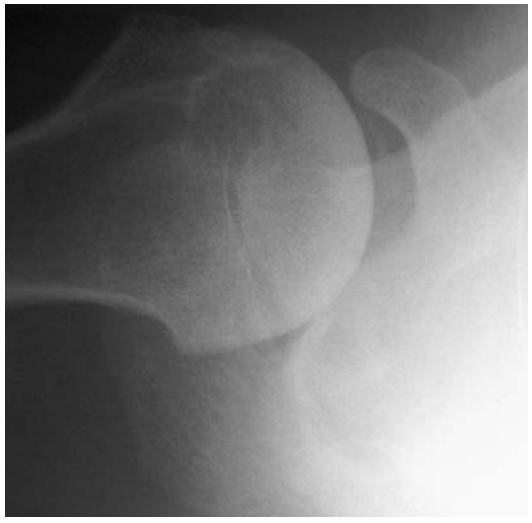

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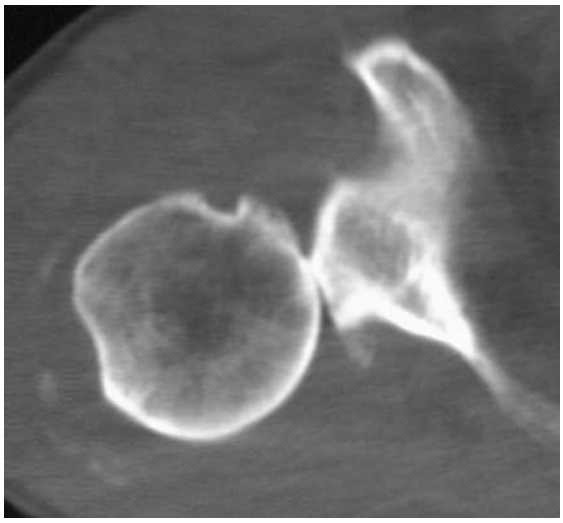


C

Figure 18-62 **(A)** Neuropathic arthropathy. The anteroposterior radiograph is unremarkable. **(B)** The axillary lateral reveals anterior subluxation of the humeral head, **(C)** whereas the computed tomography scan documents posterior dislocation in the same shoulder.

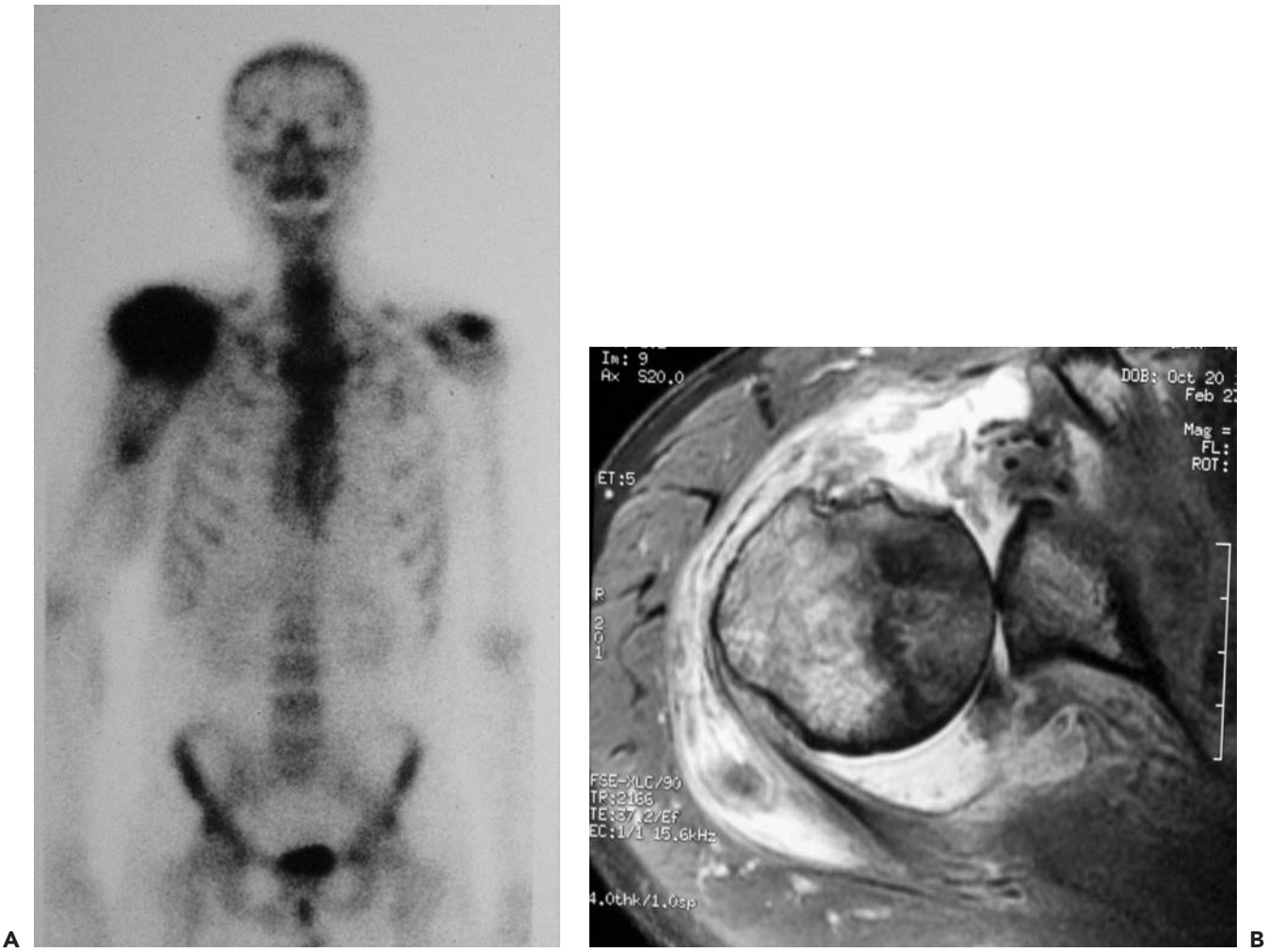


Figure 18-63 **(A)** Neuropathic arthropathy. The bone scan shows extremely radionuclide uptake in the shoulder. **(B)** Magnetic resonance imaging reveals fluid accumulation, disruption of normal soft tissue planes, and arthropathic changes of the glenohumeral articulation.

and arthrofibrosis.[296,485] As with other arthropathies, the severity of symptoms correlates directly with the radiologic involvement (Fig. 18-65).[54] Shoulder involvement is more common in adults than in juveniles and proceeds slowly over the years and manifests as erosions and thinning of the glenoid.[296]

Endocrine and Metabolic Arthropathy

Arthropathy associated with hemochromatosis can affect the shoulder, typically late in the disease course.[116,180,233,341,414] The mechanism whereby this inborn error of iron metabolism leads to arthritis is unknown. Ochronosis arthropathy affecting the spine and hips precedes shoulder involvement by many years.[161] Acromegaly can result in peripheral joint arthropathy, most commonly occurring in large joints, including the shoulder.[28,32,398] Articular cartilage hypertrophy, a condition unique to this disorder and prone to degeneration, leads to eventual osseous disruption (Fig. 18-66). Glenohumeral arthropathy has also been observed as a sequela to hyperparathyroidism.[370]

Synovial-based Arthropathy

Synovial chondromatosis, a cartilaginous metaplasia of synovium, rarely affects the glenohumeral joint (Fig. 18-67).[224,523] Secondary arthropathy associated with coarse intraarticular crepitation can follow years of intermittent or continuous pain (Fig. 18-68).[326,403,518] Malignant transformation has been reported.[94,389]

Pigmented villonodular synovitis has the potential to cause destructive arthropathy of the shoulder sufficient to warrant shoulder arthroplasty (Fig. 18-69).[505]

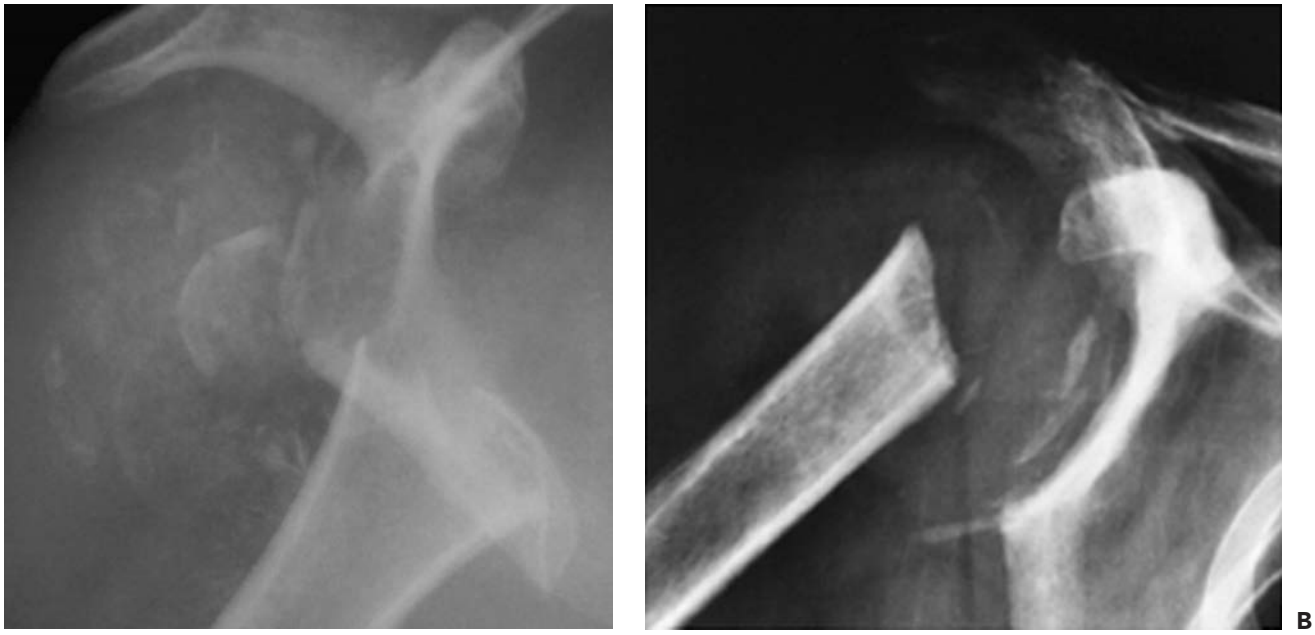


Figure 18-64 **(A,B)** Neuropathic arthropathy: The late stage of the disease with complete destruction of the normal joint architecture.

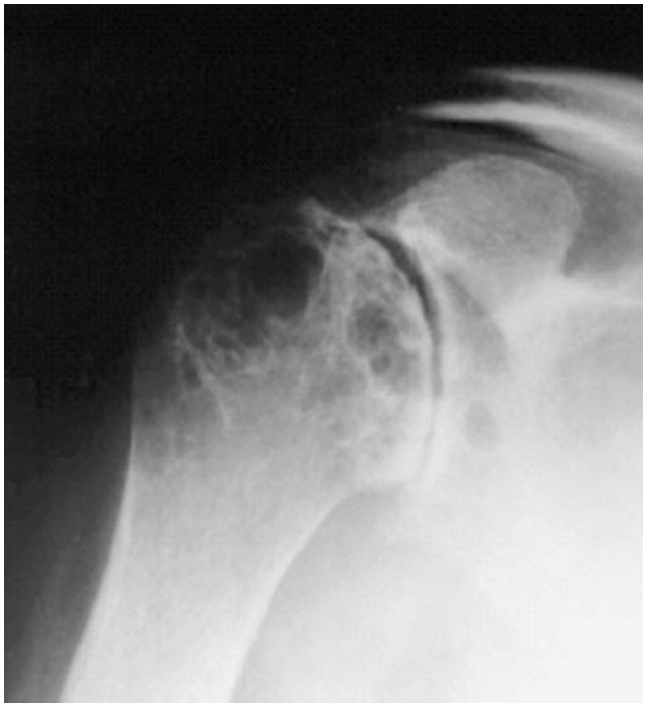


Figure 18-65 Hemophilia arthropathy. Cystic destruction of the humeral head is accompanied by progressive loss of articular cartilage.

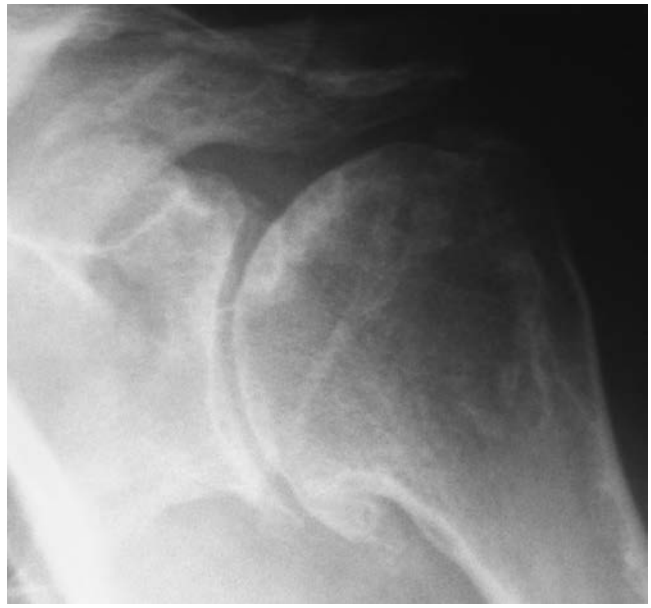


Figure 18-66 Acromegaly causing glenohumeral arthropathy.

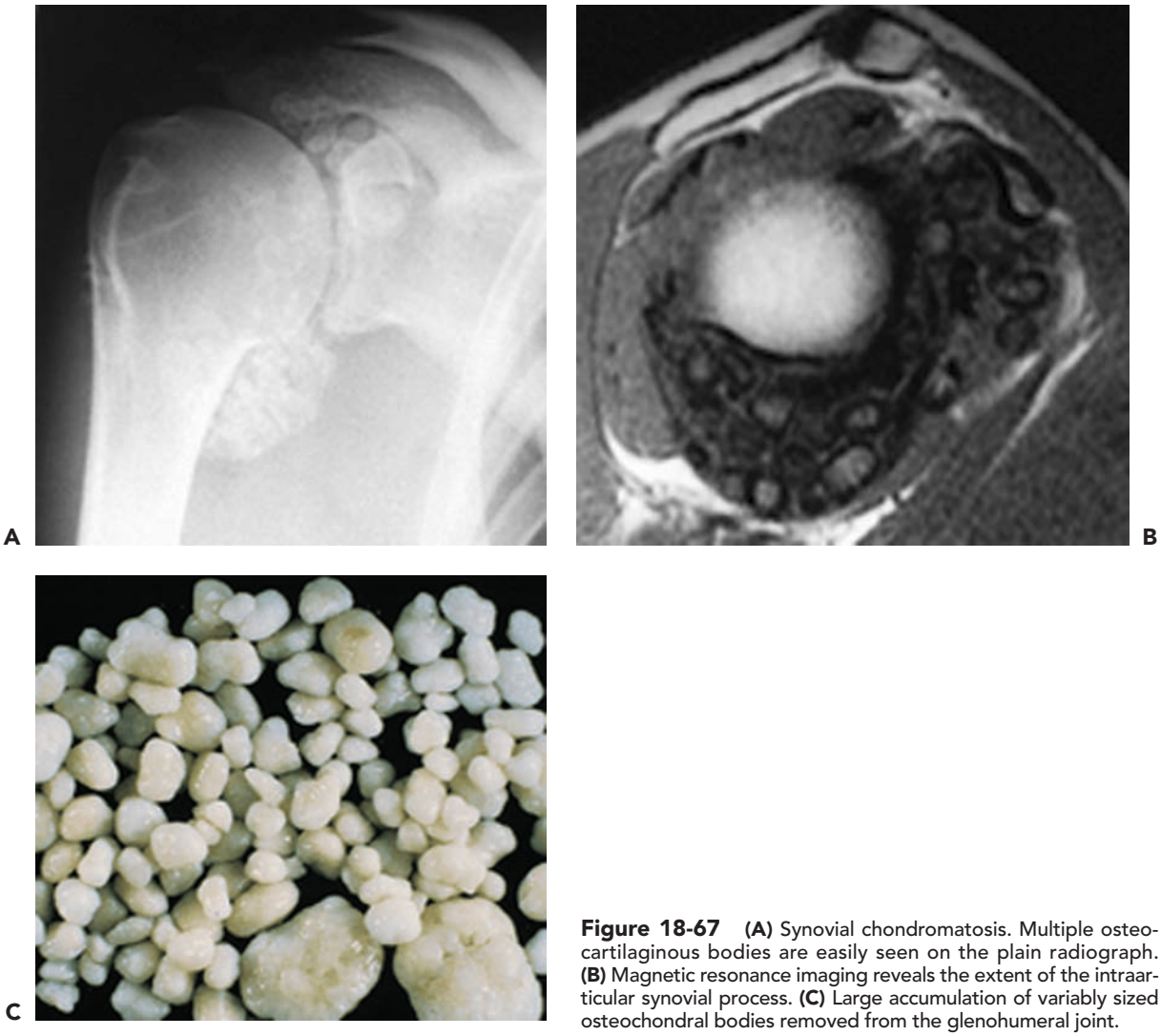


Figure 18-67 **(A)** Synovial chondromatosis. Multiple osteocartilaginous bodies are easily seen on the plain radiograph. **(B)** Magnetic resonance imaging reveals the extent of the intraarticular synovial process. **(C)** Large accumulation of variably sized osteochondral bodies removed from the glenohumeral joint.

Miscellaneous Arthropathies

Erythema nodosum may result from sarcoidosis, tuberculosis, drug ingestion, inflammatory bowel disease, Behçet's disease, and beta-hemolytic streptococcal infections. Acute shoulder involvement can be as high as 36%.[508]

Patients with sarcoidosis, a heterogeneous multisystem granulomatous disease, will develop arthritis in 15% to 25% of cases.[506,522] One-fourth of these patients will have shoulder involvement (Figs. 18-70 and 18-71).[169,539]

Systemic lupus erythematosus (SLE) and other connective tissue diseases may have significant shoulder involvement, but these occurrences are uncommon. Sixty percent to 90% of SLE patients will develop arthritis, the most common manifestation of the disease.[273] Osteonecrosis occurs in 10% to 52%, not always associated with steroid ingestion.[1]

Amyloid deposition disease will involve the shoulder in 68% of cases.[89,129] Usually this is not related to glenohumeral destruction, but is a problem in the soft tissues.[237] The pathognomonic "shoulder pad pattern" is always associated with shoulder involvement. Osteopenia with associated joint destruction is a very late phenomenon.[74]

Polymyalgia Rheumatica

Polymyalgia rheumatica is a pain and stiffness syndrome that commonly affects the elderly. It may be difficult to distinguish from late-onset rheumatoid arthritis.[152] Constitutional symptoms include fever, malaise, and weight loss. Symmetrical shoulder involvement is common, sometimes expressed as synovitis, although large effusions are uncommon, with no propensity for joint destruction. Acute-phase reactant serum markers are invariably elevated and strongly support the diagnosis.[453]

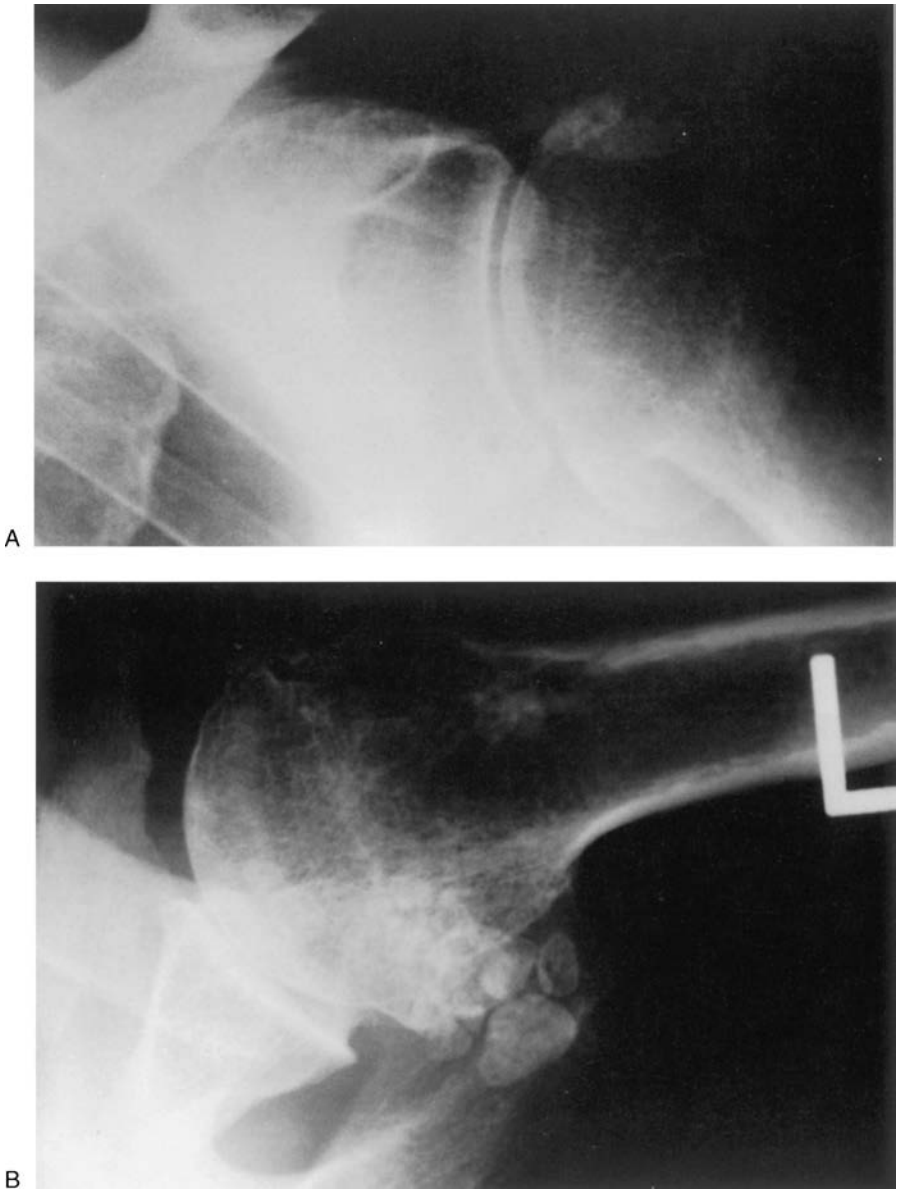


Figure 18-68 **(A,B)** Synovial osteochondromatosis with early degenerative changes of the glenohumeral articulation.

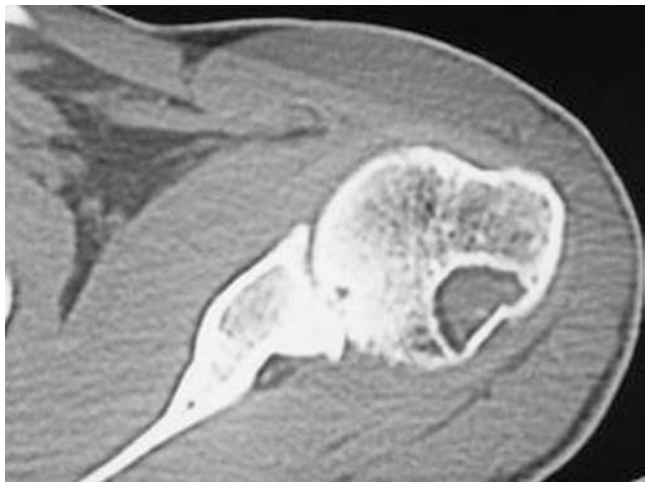
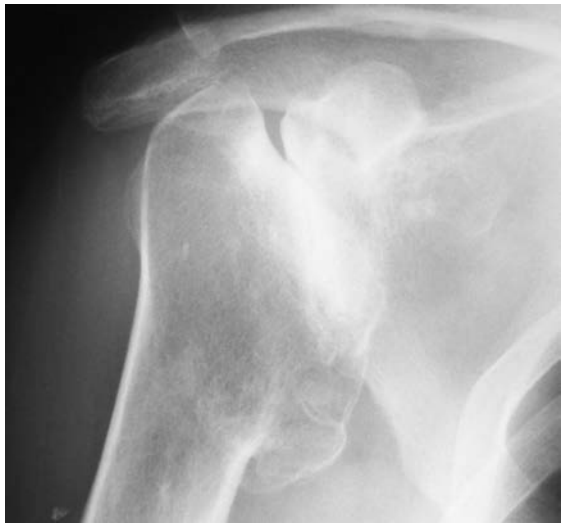


Figure 18-69 Pigmented villonodular synovitis resulting in glenohumeral arthropathy.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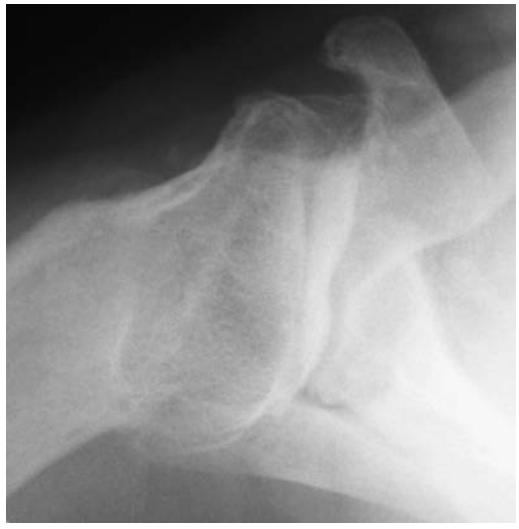
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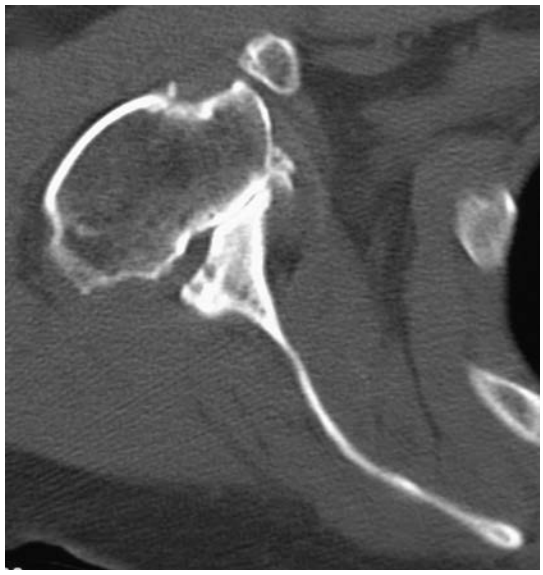
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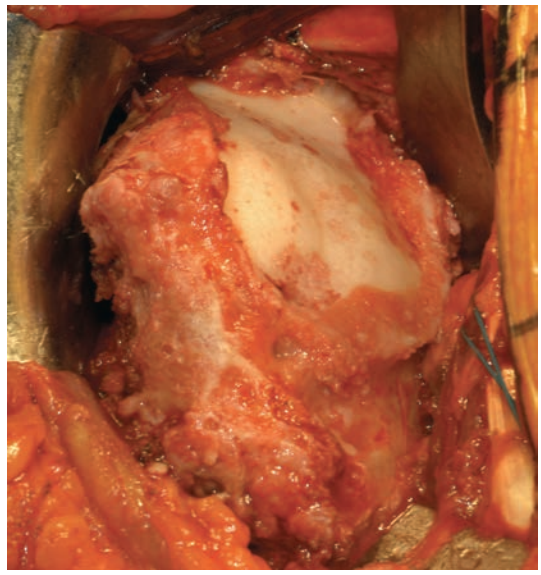
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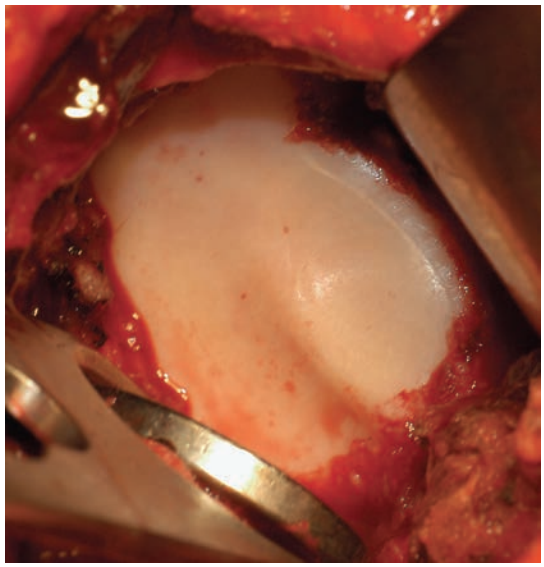
E

Figure 18-70 **(A–C)** Multiple epiphyseal dysplasia. Significant deformity has led to extreme secondary osteoarthritis. **(D,E)** Long-standing accommodation has resulted in "mated" humeral and glenoid surfaces.

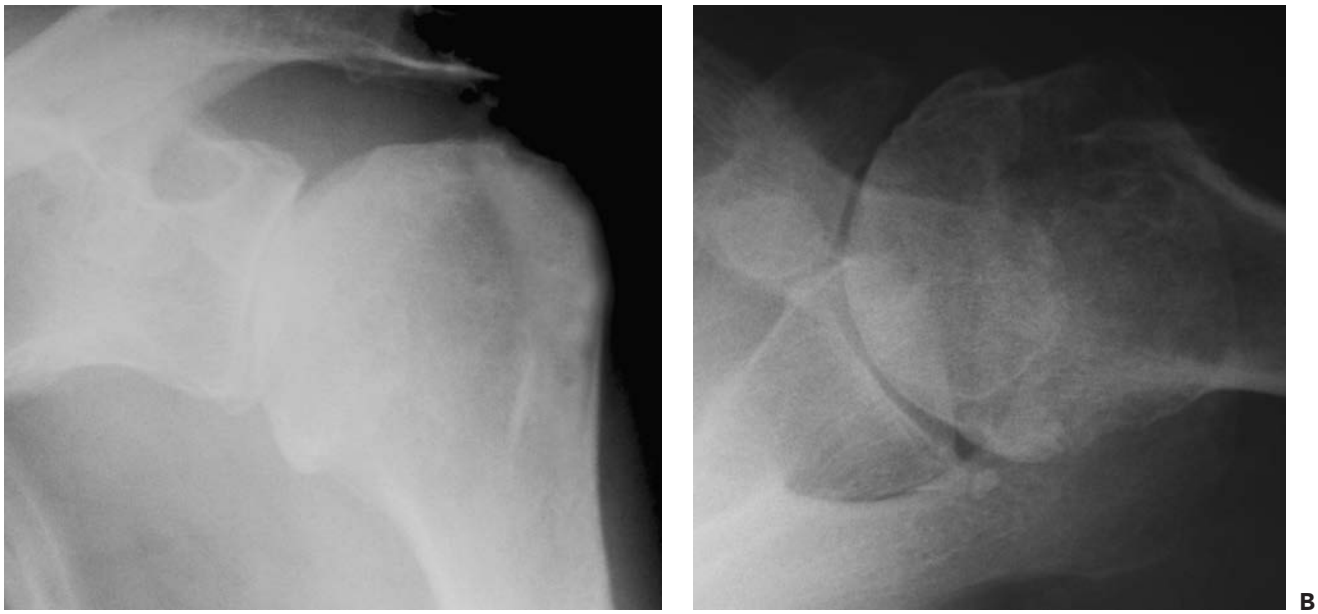

Figure 18-71 Sarcoidosis arthropathy.

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19 Biomechanics of the Glenohumeral Joint: Influence on Shoulder Arthroplasty

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INTRODUCTION

The normal glenohumeral joint is often referred to as a ball-and-socket joint. The hip joint is another well-known ball-and-socket joint whose bony socket covers two-thirds of the ball. Compared to the hip joint, however, the glenohumeral joint is much less constrained. Its osseous anatomy is more like a golf ball on a golf tee. Despite this lack of constraint, the glenohumeral joint exhibits a kinematic pattern that closely approximates the purely rotational motion seen in true ball-and-socket joints. This normal kinematic pattern requires a complex interaction between articular surface anatomy, capsular constraints, and rotator cuff, deltoid, and scapular muscle forces. The goals of unconstrained prosthetic replacement of the glenohumeral joint are to anatomically reconstruct the articular surfaces, to restore flexibility to the capsular constraints, and to repair or rehabilitate the rotator cuff, deltoid, and periscapular muscles so that pain-free, normal motion is restored. In some disease processes, such as cuff tear arthropathy, rotator cuff deficiency may be irreparable and anatomic reconstruction of the joint may not be possible. Under these circumstances, prosthetic reconstruction with nonanatomic devices (i.e., reversed prostheses) may be required.

Several anatomic and biomechanical factors have been identified that may affect prosthetic design. Important anatomic factors include humeral head size and its

orientation in three-dimensional space; humeral head shape; humeral head offset; humeral neck–shaft angle; glenoid size, shape, and orientation; glenoid offset; and lateral glenohumeral offset. Biomechanical factors that may influence prosthetic design and performance include glenohumeral articular conformity, glenohumeral articular constraint, periarticular muscular forces, and ligamentous restraints. Certain prosthetic characteristics, such as variability in humeral neck–shaft angle and version, humeral head offset, glenoid fixation and the shape of its interface with the native glenoid, glenoid metal backing, and material properties of the articulating surfaces, may influence prosthetic performance and clinical outcome. The purposes of this chapter are to review the current knowledge regarding relevant anatomic and biomechanical factors that potentially influence glenohumeral prosthetic design and to discuss the possible effects that variations in these design parameters may have on prosthetic performance.

ANATOMIC FACTORS

Humeral Head Size

Given the assumption that the humeral head is a sphere, the volume (i.e., size) of the articular segment is determined by the humeral head radius of curvature and humeral head thickness or neck length (Fig. 19-1). Humeral head radius, humeral head thickness, and humeral head size are all variable. Mean humeral head radius is approximately 24 mm, with a range of 19 to 28 mm (Fig. 19-2).[51,86] Mean humeral head thickness is approximately 19 mm, with a range of 15 to 24 mm (Fig. 19-3).[51,86] Both humeral head radius and thickness correlate strongly with humeral shaft length and patient height.[51,86] However, the ratio of humeral head thickness to humeral head radius of curvature is remarkably constant at approximately 0.7 to 0.9, regardless of patient height or humeral shaft size.[51,86] The surface arc of the humerus available for

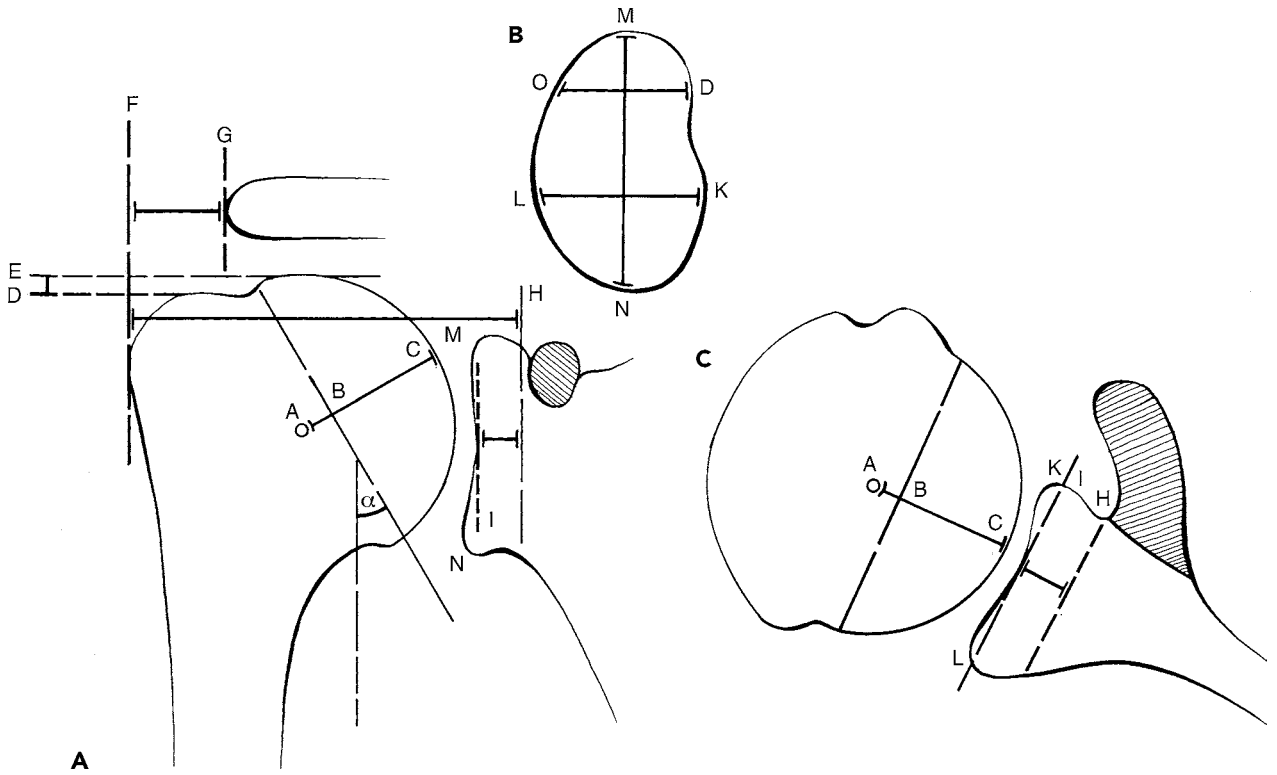

Figure 19-1 **(A)** Normal glenohumeral relationships depicted in the coronal plane include the humeral head center (*A*), humeral head radius of curvature (*AC*), humeral head thickness or neck length (*BC*), humeral neck–shaft angle (α), lateral glenohumeral offset (*HF*), greater tuberosity to acromion distance (*FG*), greater tuberosity to humeral head distance (*DE*), superior–inferior glenoid dimension (*MN*), and glenoid offset (*HI*). **(B)** The glenoid dimensions measured in the sagittal plane include the superior–inferior dimension (*MN*), the anterior–posterior dimension of the upper half (*OD*), and the anterior–posterior dimension of the lower half (*KL*). **(C)** The glenohumeral relationships in the axial plane include the humeral head center (*A*), the humeral head radius of curvature (*AC*), the humeral head thickness or neck length (*BC*), the anterior–posterior dimension of the glenoid (*KL*), and the glenoid offset (*HI*). (Reprinted with permission from Iannotti JP, Gabriel JP, Schneck SL, et al. The normal glenohumeral relationships. An anatomical study of one hundred and forty shoulders. *J Bone Joint Surg Am* 1992;74:491.)

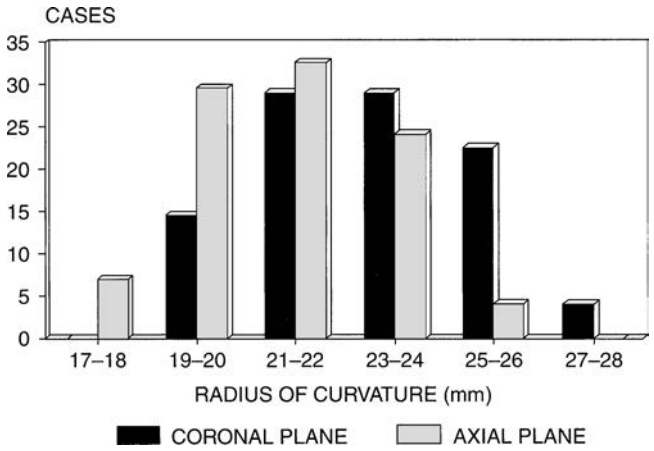

Figure 19-2 Humeral head radius of curvature fits a Gaussian distribution with a mean of 23 to 24 mm and a range of approximately 19 to 28 mm. Note that the axial plane radius is slightly smaller than the coronal plane radius. (Reprinted from Iannotti JP, Gabriel JP, Schneck SL, et al. The normal glenohumeral relationships. An anatomical study of one hundred and forty shoulders. *J Bone Joint Surg Am* 1992;74:491.)

contact with the glenoid is directly proportional to the ratio of humeral head thickness to humeral head radius and is, therefore, also relatively constant, irrespective of humeral shaft length or patient height.[54,86]

Humeral Head Shape

The assumption that the humeral head is spherical is only partially accurate. The central 80% of the articular surface is spherical, with the coronal plane and axial plane radii of curvature being equal.[1,21,51,69] However, in the peripheral 20% of the articular surface, the radius of curvature in the axial plane is, on average, 2 mm less than in the coronal plane (see Fig. 19-2). Therefore, the peripheral 20% of the humeral articular surface is elliptical, with the ratio of the axial plane radius to the coronal plane radius being 0.92.[51] The clinical significance of this anatomic finding remains unclear, as almost all current prosthetic systems utilize spherical humeral heads, and cadaver studies evaluating the range of motion and three-dimensional kinematics of natural joints and joints reconstructed with spherical humeral heads show no difference.[56]

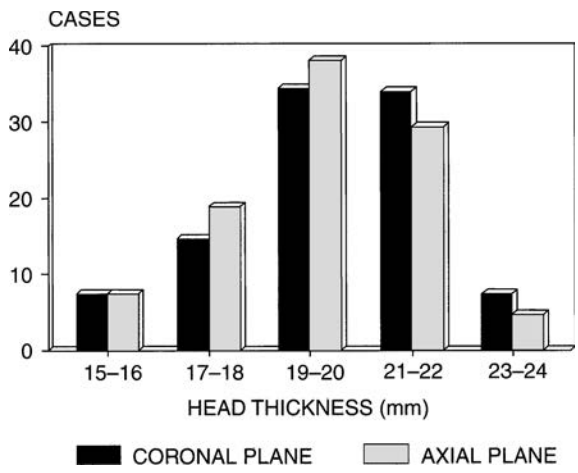

Figure 19-3 Humeral head thickness also demonstrates a bell-shaped distribution with a mean of 19 mm and a range of 15 to 24 mm. (Reprinted with permission from Iannotti JP, Gabriel JP, Schneck SL, et al. The normal glenohumeral relationships. An anatomical study of one hundred and forty shoulders. *J Bone Joint Surg Am* 1992;74:491.)

Humeral Head Offset

The center of the humeral head does not coincide with the projected center of the humeral shaft. Alternatively, the point in the proximal humeral metaphysis that corresponds to the center of the humeral head (assuming that the humeral head is a sphere) does not lie on a line projected proximally into the humeral metaphysis from the central axis of the intramedullary canal of the humeral diaphysis.[6,12,72,86,95] The distance between the center of the humeral head and the central axis of the intramedullary canal is defined as the humeral head offset.[6,12,86,95] Although humeral head offset is undoubtedly three-dimensional, it is commonly described in two planes, coronal and axial. Like most other proximal humeral anatomic parameters, reported humeral head offsets are variable.[6,12,72,86,95] In the coronal plane, the humeral head offset is approximately 7 to 9 mm medial to the central axis of the intramedullary canal; in the axial plane, the humeral head offset is 2 to 4 mm posterior to the central axis of the intramedullary canal (Fig. 19-4).[6,12,86,95]

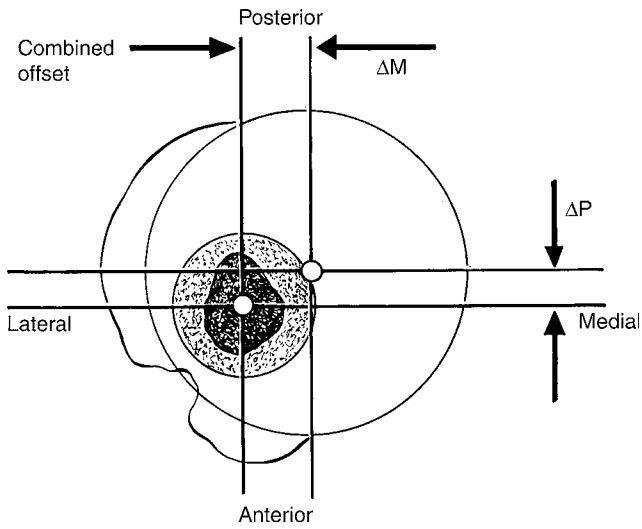

Figure 19-4 This axial plane section reveals the combined posterior and medial humeral head offset with respect to the central axis of the intramedullary canal. (Reprinted with permission from Boileau P, Walch G. The three-dimensional geometry of the proximal humerus. Implications for surgical technique and prosthetic design. *J Bone Joint Surg Br* 1997;79:857.)

Humeral head offset is correlated with humeral head radius and humeral head thickness.[86] However, for a given humeral head radius, humeral head thickness, and humeral head offset in the coronal and axial planes, the location of the humeral articular surface may vary with respect to angle of rotation about the central intramedullary axis (i.e., humeral retroversion) and superior–inferior translation along the central intramedullary axis (i.e., head to greater tuberosity height). Humeral retroversion averages 20 to 30 degrees with a wide range of approximately 20 to 55 degrees.[12,63,86,95] The vertical distance between the highest point of the humeral articular surface and the highest point of the greater tuberosity (i.e., head to greater tuberosity height) is approximately 8 mm and shows a relatively small range of interspecimen variability.[51,86]

Humeral Neck–Shaft Angle

The neck–shaft angle is defined as the angle subtended by the central intramedullary axis of the humeral shaft and the base of the articular segment (see Fig. 19-1). The average neck–shaft angle is 40 to 45 degrees.[12,51,72,86] However, more importantly, the humeral neck–shaft angle demonstrates significant individual variation with a range of 30 to 55 degrees.[12,51,72,86] One study has shown a correlation between humeral head radius and neck–shaft angle, with larger humeral heads demonstrating larger neck–shaft angles (Fig. 19-5).[51]

Glenoid Size and Shape

The size and shape of the articular surface of the glenoid can be defined by its linear superior–inferior and anterior–posterior dimensions as well as by its radius of curvature (see Fig. 19-1).[51,72] The mean superior–inferior dimension

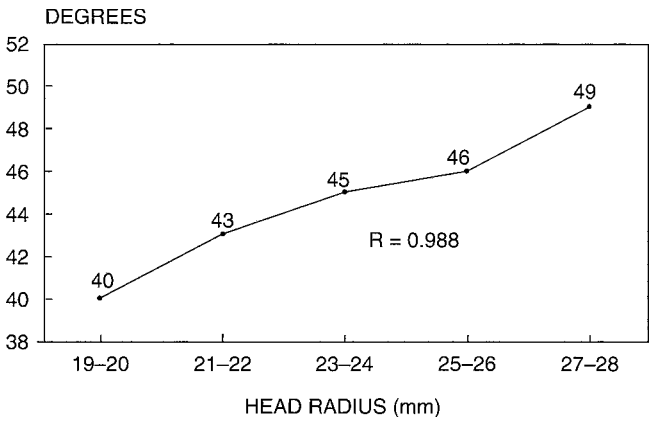

Figure 19-5 Humeral neck–shaft angle has been shown to correlate with humeral head size. (Reprinted with permission from Iannotti JP, Gabriel JP, Schneck SL, et al. The normal glenohumeral relationships. An anatomical study of one hundred and forty shoulders. *J Bone Joint Surg Am* 1992;74:491.)

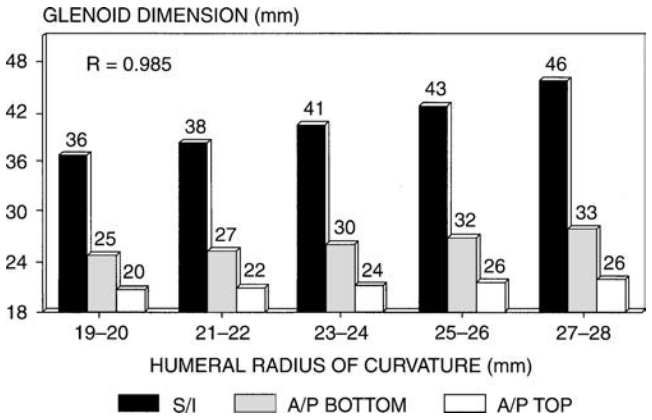

Figure 19-6 Humeral head size correlates with the superior–inferior dimension of the glenoid, the anterior–posterior dimension of the upper half of the glenoid, and the anterior–posterior dimension of the inferior half of the glenoid. A/P = anteroposterior; S/I = Superoinferior. (Reprinted with permission from Iannotti JP, Gabriel JP, Schneck SL, et al. The normal glenohumeral relationships. An anatomical study of one hundred and forty shoulders. *J Bone Joint Surg Am* 1992;74:491.)

of the glenoid (excluding the labrum) is approximately 39 mm (range 30 to 48 mm).[51] The anterior–posterior dimension of the superior half of the glenoid is shorter than the inferior half of the glenoid, resulting in a pear-shaped or comma-like appearance. The mean anterior–posterior dimension of the superior half of the glenoid (excluding the labrum) is approximately 23 mm (range 18 to 30 mm), and the mean anterior–posterior dimension of the inferior half of the glenoid (excluding the labrum) is approximately 29 mm (range 21 to 35 mm).[51] The ratio of the superior–inferior dimension to the anterior–posterior dimension of the larger, inferior half of an average glenoid is 1:0.7.[51] The humeral head radius correlates with the size of the glenoid in both the superior–inferior and anterior–posterior dimensions (Fig. 19-6).[51]

The glenoid vault has a complex geometry with a regular shape that can be approximated by a model of stacked triangles.[9] The glenoid vault shape is consistent among a wide patient population and is independent of glenoid size or patient gender (Fig. 19-7).[9]

Controversy exists with regard to the relationship between the glenoid and humeral articular radius of curvature.[51,59,72,101] This controversy exists because of differences in measuring techniques, differences in sample sizes, and large individual variations in anatomy. The thickness of the articular cartilage of the glenoid increases toward the periphery of the articular surface and must be included when measuring the glenoid radius of curvature.[101] However, even when the articular cartilage is included in the measurement, the radius of curvature of the glenoid articular surface does not equal the radius of the humeral articular surface in all specimens.[51,59,72,101] Iannotti and colleagues[51] observed that, on average, the glenoid radius of curvature in the coronal plane was 2.3 mm larger than the

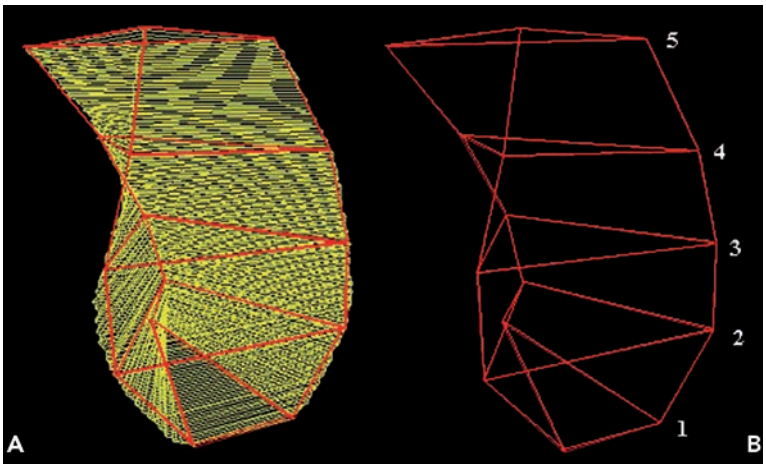

Figure 19-7 The shape of the glenoid vault can be approximated using a model of stacked triangles. (Reprinted with permission from Bennetts C, Gordiev K, Powell K, et al. Quantitative image analysis of glenoid vault shape. J Shoulder Elbow Surg (in review).

coronal plane humeral radius of curvature in the same specimen. Soslowsky and coworkers[101] reported a difference between humerus and glenoid radii of curvature of less than 2 mm in 88% of specimens and less than 3 mm in all specimens. Kelkar and associates[59,60] reported a mean humeral radius of curvature that was 2 mm less than the mean glenoid radius of curvature. In addition, as the humerus is abducted from a neutral position, the percentage of glenoid contact increases significantly, indicating that the functional conformity may increase during active elevation.[115]

Glenoid Offset

The glenoid offset is defined as the perpendicular distance between the base of the coracoid process and the deepest portion of the glenoid articular surface (see Fig. 19-1). This distance determines the location of the glenohumeral joint line and is not correlated with humeral or patient size.[51] The average glenoid offset is approximately 2 mm (range −5.0 to +5.0 mm).[51]

Lateral Glenohumeral Offset

The lateral glenohumeral offset can be defined as the perpendicular distance from the base of the coracoid process to the most lateral extent of the greater tuberosity (Fig. 19-1).[51] The distance from the most lateral extent of the greater tuberosity to the lateral edge of the acromion process correlates with the lateral glenohumeral offset and is easily measured intraoperatively (Fig. 19-1). Lateral glenohumeral offset is important in shoulder function because it determines capsular tension, resting length of the rotator cuff muscles, and the moment arm for the deltoid muscle. Lateral glenohumeral offset averages approximately 54 to 57 mm (range 43 to 68 mm), and the distance from the greater tuberosity to the lateral margin of the acromion process averages 17 mm (range 15 to 21 mm).[51] Since the glenoid offset is less than 5 mm in all normal shoulders, it has little effect on lateral glenohumeral offset.[51] However, both lateral glenohumeral offset and greater tuberosity to acromion distance correlate with humeral head size, which in turn correlates with patient height (Fig. 19-8).[51]

BIOMECHANICAL FACTORS

Glenohumeral Articular Conformity

Conformity of the articular surfaces refers to the difference between the radii of curvature of the humerus and glenoid. Congruent or conforming articulations are characterized by glenoid and humeral surfaces with equal radii of curvature. Incongruent or nonconforming articulations exist when the

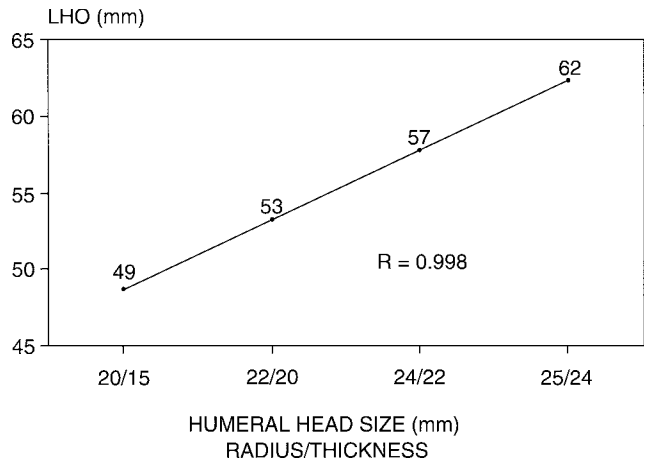

Figure 19-8 Lateral glenohumeral offset is defined as the distance from the base of the coracoid process to the lateral aspect of the greater tuberosity and is correlated with humeral head size. LHO = lateral humeral offset. (Reprinted with permission from Iannotti JP, Gabriel JP, Schneck SL, et al. The normal glenohumeral relationships. An anatomical study of one hundred and forty shoulders. *J Bone Joint Surg Am* 1992;74:491.)

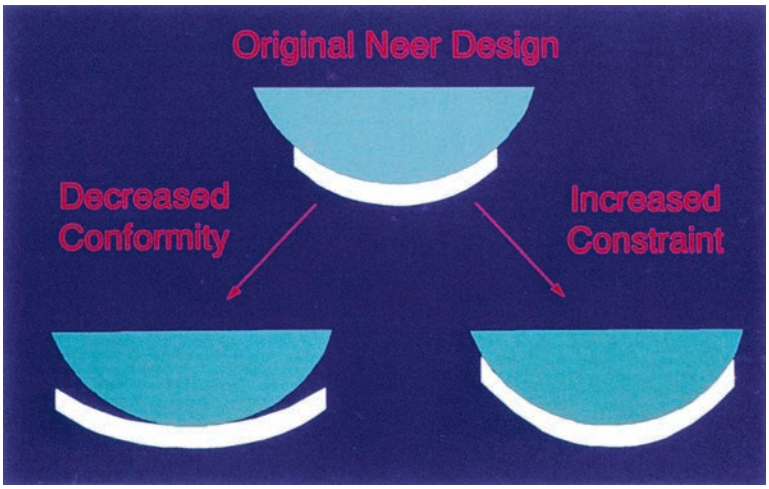

Figure 19-9 Conformity refers to the difference between the humeral head radius and the glenoid radius. Constraint is determined by glenoid wall height and is independent of conformity. Two articulations may have identical conformity and different constraints and vice versa.

humerus and glenoid radii of curvature are not equal. In the normal shoulder, the components of the articulating surfaces include the humeral articular cartilage, glenoid articular cartilage, and glenoid labrum. The humeral articular cartilage is uniform in thickness throughout the entire articulating surface. As mentioned previously, the glenoid articular cartilage is thicker at the periphery than in the center.[101] The glenoid labrum extends the arc of the articular cartilage and deepens the glenoid socket, but does not change the radius of curvature of the glenoid surface.[49] The articular cartilage and glenoid labrum are reversibly deformable tissues. Therefore, for practical purposes, normal glenohumeral joints can be considered to be conforming if the humeral head radius of curvature is equal to or larger than the glenoid radius of curvature.[58] Normal glenohumeral joints are considered to be nonconforming when the humeral head radius is less than the glenoid radius.[58]

Conformity or congruence of the normal glenohumeral articulation is variable, as mentioned above in the section on glenoid size and shape.[35,51,58,59,72] In the majority of specimens reported, the humeral head radius is within 2 to 3 mm of the glenoid radius.[51,58,59,72] However, even small amounts of articular incongruence or nonconformity have important implications with regard to glenohumeral kinematics during active motion.[58]

Glenohumeral Articular Constraint

Constraint is defined as the ability of the articular surfaces to resist translational motion of the humerus on the glenoid. The most extreme example of a constrained articulation is one in which the glenoid and humeral components are mechanically coupled to one another. In normal shoulders, where the glenoid and humerus are obviously not mechanically coupled, articular constraint is a function of the percentage of the humeral head covered by the glenoid. Articular constraint is correlated with glenoid wall height or, alternatively, socket depth, and is independent of articular conformity. In other words, different articulations may have equal conformities but different constraints, and vice versa (Fig. 19-9).

The normal glenoid is more constrained (i.e., has greater depth) in the superior–inferior direction than in the anterior–posterior direction.[49,72] Additionally, the depth of the normal glenoid is contributed to equally by the articular cartilage and the glenoid labrum.[49] In the superior–inferior and anterior–posterior dimensions, the glenoid articular cartilage and labrum combine to yield a socket that is 9 mm and 5 mm deep, respectively.[49] Glenoid depth can also be thought of in terms of coverage of the humeral head.[72] In the superior–inferior direction, the glenoid covers approximately a 65-degree arc of the humeral surface, compared to a 50-degree arc of coverage in the anterior–posterior direction.[72]

Periarticular Muscular Forces

The interactions between the many muscles that control motion of the shoulder girdle are extremely complex and beyond the scope of this chapter. However, in a simplified sense, the deltoid muscle provides power movement to the glenohumeral joint while the rotator cuff muscles compress the humeral head into the glenoid socket to provide a steering and stabilizing function.[7,37,87–89,100,114] In a normal shoulder, the sheer force imparted by the deltoid and other large muscles is counterbalanced by the axial or compressive force imparted by the rotator cuff. This results in decreased humeral translation and increased joint stability.[68] Pathologic conditions that result in altered rotator cuff function, such as cuff tear arthropathy, may exhibit altered glenohumeral kinematics because of muscular imbalance between the rotator cuff and deltoid. In the worst-case scenario, the deficient rotator cuff may be unable to provide a sufficient enough fulcrum to allow

elevation of the arm. Under these circumstances, anatomic prosthetic designs that depend on soft tissue integrity for stability and function will not restore overhead function.

Ligamentous Restraints

The glenohumeral ligaments are thickenings of the joint capsule that are somewhat variable in their appearance.[20,32,82,110,116] They act as checkreins to excessive rotation or translation of the humeral head at the extremes of glenohumeral motion.[20,82,116] Although various regions of the capsule act reciprocally to help stabilize the shoulder at extremes of motion, the ability of various ligaments to control glenohumeral motion depends upon their location within the capsule as well as the arm position. The rotator interval capsule occupies the gap between the upper portion of the subscapularis and the anterior border of the supraspinatus. It consists of the superior glenohumeral ligament and coracohumeral ligament.[27,43,117,118] The rotator interval capsule is primarily responsible for limiting external rotation and inferior subluxation of the humerus with the arm at the side.[27,43,116,117] The middle glenohumeral ligament limits external rotation and anterior subluxation of the humerus with the arm in midabduction.[82]

The inferior glenohumeral ligament consists of an anterior band, an axillary pouch, and a posterior band.[81,110] It is taut in abduction and therefore limits the degree of abduction as well as inferior translation of the humeral head in full abduction. The anterior band tightens in combined abduction and external rotation and therefore limits the amount of abduction and external rotation possible as well as anterior translation with the arm in this position.[81,110,118] The posterior band is taut in abduction and internal rotation. Therefore, it limits the amount of simultaneous abduction and internal rotation possible and also limits posterior humeral translation with the arm in this position.[81,110,118] The anterior and posterior bands can be made even more taut by passively positioning the abducted humerus posterior and anterior to the scapular plane, respectively.

Tension in any of the glenohumeral ligaments will cause obligate translation in the opposite direction. For example, tension in the anterior band of the inferior glenohumeral ligament will result in posterior translation of the humeral head.[43,45,58] Pathologic conditions that result in capsular contracture, such as glenohumeral arthritis, may lead to altered humeral rotation and translation because of premature capsular tightening. One of the most important goals of prosthetic reconstruction is to release capsular contractures to restore joint flexibility and, presumably, improve kinematics.

Normal Kinematics

Glenohumeral motion is composed of translation and rotation of the humerus on the glenoid. When the humerus is moved from one point in space to another, the motion occurs as a combination of rotation and translation. The percentage of that motion that occurs as rotation is variable and depends on the rotator cuff forces, capsular tension, and joint conformity.[58] If the motion were purely rotation, the shoulder would be considered strictly a "ball-in-socket" joint. Conversely, if the motion were entirely translation, the shoulder would be considered a planar joint.

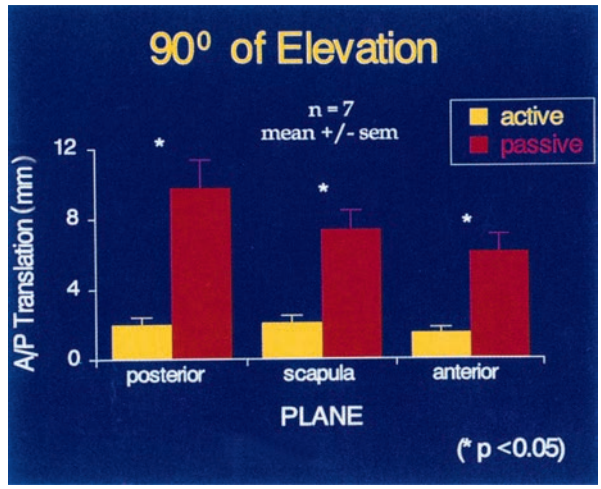

Figure 19-10 This figure depicts anterior–posterior humeral translation during both active and passive positioning from maximum internal to maximum external rotation at 90 degrees of total elevation (i.e., 60 degrees glenohumeral and 30 degrees scapular) in 30 degrees anterior to and 30 degrees posterior to the scapular plane. Note that anterior–posterior humeral translation is smaller (2 mm) during active joint positioning and larger (8 mm) during passive joint positioning. A/P = anteroposterior.

Humeral head translation during active joint positioning with a normal rotator cuff and no capsular contracture is variable but small.[10,58,61] Under these conditions, glenohumeral motion approaches "ball-in-socket" kinematics. Anterior–posterior translation and superior–inferior translation are approximately 2 mm (Fig. 19-10).[58] Rotation occurs about a relatively fixed point that is located within 1 to 3 mm of the geometric center of the humeral head.[10,59,61] Although the translations are small, they are correlated with conformity of the articular surfaces. Nonconforming joints exhibit more translation during active motion than conforming joints (Fig. 19-11).[58]

Humeral head translation during passive joint positioning with a nominal centering force is much larger than during active joint positioning with normal rotator cuff forces and is independent of joint conformity (see Figs. 19-10 and 19-11).[45,46,58] Anterior–posterior passive translation is approximately 8 mm, fourfold greater than active translations.[58] Passive superior–inferior translation is approximately 4 mm, twofold greater than active translations (see Fig. 19-10).[58]

The range of motion achievable during passive joint positioning, with only a nominal centering force applied, is much larger than during active joint positioning.[58] The

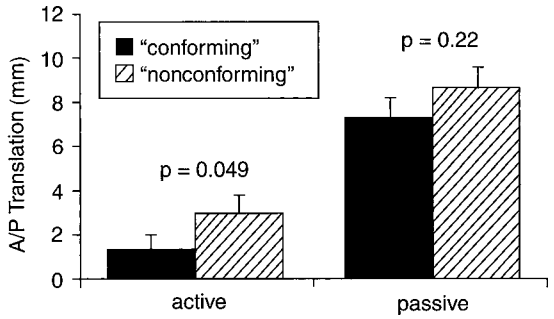

Figure 19-11 Nonconforming joints have significantly greater translation during active joint positioning than conforming joints. A/P = anteroposterior. (Reprinted with permission from Karduna AR, Williams GR, Williams JL, et al. Kinematics of the glenohumeral joint: influences of muscle forces, ligamentous constraints, and articular geometry. *J Orthop Res* 1996;14:986.)

increased humeral translation observed during passive joint positioning occurs at the extremes of rotational motion not achievable during active joint positioning. In fact, if passive and active humeral translations are compared over the same range of motion (i.e., midrange), the difference is insignificant (Fig. 19-12).[58] Moreover, the increased passive translations that occur at the extremes of motion correlate with increased ligament length (i.e., tension) on the side of the joint opposite the translation.[58] For example, anterior translation with the humerus in 90 degrees of elevation in the scapular plane occurs with increasing internal rotation and is associated with increasing length of the posterior band of the inferior glenohumeral ligament.[58]

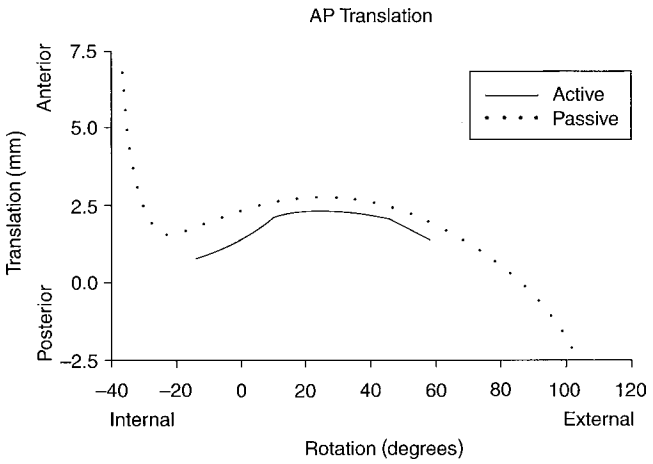

Figure 19-12 This representative plot of translation versus range of motion demonstrates that the increased translations seen during passive joint positioning occur at the extremes of rotation not achievable during active joint positioning. Humeral head translations during active and passive joint positioning over the same range of rotation are not significantly different. AP = anteroposterior. (Reprinted with permission from Karduna AR, Williams GR, Williams JL, et al. Kinematics of the glenohumeral joint: influences of muscle forces, ligamentous constraints, and articular geometry. *J Orthop Res* 1996;14:986.)

Prosthetic Kinematics

In general, the relationships between active translations, passive translations, joint conformity, and ligament length observed in the normal shoulder are also present in the shoulder with a normally functioning rotator cuff and restored joint flexibility that has been reconstructed with anatomic, nonconstrained prostheses.[56] Humeral head translations observed during active joint positioning are small (0.3 to 1.7 mm) and strongly correlated with prosthetic component conformity (Fig. 19-13).[56] Humeral head translations during passive joint positioning are large (5 to 9 mm) and independent of prosthetic component conformity.[56] The increased passive translations that occur at the extremes of motion are correlated with increasing length of the capsule (i.e., ligament) on the opposite side of the joint to the translation.[56] The degree of prosthetic radial mismatch that produces a kinematic pattern of active motion that most closely resembles the natural joint is unknown and likely to vary from individual to individual. However, in one study of seven cadaver shoulders, prosthetic radial mismatch of approximately 4 mm produced active translations that most closely resembled the specimen mean prior to prosthetic arthroplasty (see Fig. 19-13).[56]

IMPLICATIONS FOR NONCONSTRAINED PROSTHETIC DESIGN

The ability of a particular prosthetic design to achieve anatomic reconstruction of the glenoid and humeral articular surfaces and to restore normal glenohumeral kinematics is obviously limited by the ability of the surgeon to restore soft tissue balance and correct any bony deficiencies. Certainly, prosthetic design alone cannot overcome pathology that is too severe to be corrected. Moreover, the choice of

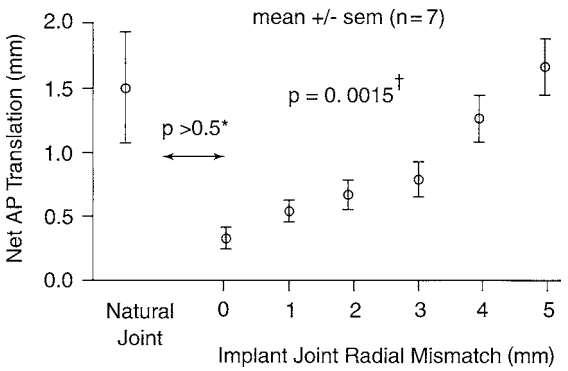

Figure 19-13 Humeral head translation during active joint positioning following prosthetic arthroplasty is correlated strongly with component conformity. AP = anteroposterior. (Reprinted with permission from Karduna AR, Williams GR, Williams JL, et al. Glenohumeral joint translations before and after total shoulder arthroplasty. A study in cadavera. *J Bone Joint Surg Am* 1997;79:1166.)

Radius of Curvature *(mm)*	Thickness *(mm)*		
	15–17	18–20	21–24
19–20	10	3	2
21–22	7	18	3
23–24	0	9	18
25–26	0	8	14
27–28	0	0	4

Figure 19-14 Eighty-five percent of all humeral head sizes are defined by eight combinations of humeral head radius and thickness. Note that humeral head sizes with large radii but small thicknesses, and vice versa, are not frequently encountered in normal shoulders. (Reprinted with permission from Iannotti JP, Gabriel JP, Schneck SL, et al. The normal glenohumeral relationships. An anatomical study of one hundred and forty shoulders. *J Bone Joint Surg Am* 1992;74:491.)

implant cannot substitute for skillful surgical technique. However, assuming that all other variables are equal, the implant that is able to place a humeral head of the same size as the normal head, in exactly the same position on the humeral metaphysis as the normal head and in articulation with a glenoid component that anatomically restores both the location and inclination of the joint line, has the best opportunity for restoring normal glenohumeral kinematics.

Anatomic Factors

Theoretically, 85% of all humeral heads can be reconstructed to within 2 mm of their natural dimensions with eight head sizes (Fig. 19-14).[51] Maintaining the ratio of humeral head radius to humeral head thickness to a narrow range will allow maximization of humeral surface area available for contact.[51,86] Except in unusual situations with extraordinary bone loss or soft tissue deficiency, humeral heads with small radii of curvature and large thicknesses (or vice versa) are undesirable. Heads with inappropriately large neck lengths for their radii of curvature have a larger volume than the natural head that was removed and will result in overstuffing of the joint, increased soft tissue tension, and decreased range of motion or subscapularis rupture.[44]

Modular humeral systems offer some advantages over one-piece designs. The ability to place any humeral head on any humeral body results in less inventory. In addition, if revision is required, the head can be removed without removing the body.[99] However, modularity is also accompanied by some potential disadvantages. First, the head and body can become disassembled.[28] Second, in some designs, there is a large gap between the body of the implant and the head. This results in loss of available articular surface and potential contact between nonarticular portions of the proximal humerus and the glenoid component (Fig. 19-15).[54,86] If this gap is combined with beveling of the edge of the humeral head, thus creating a nonspherical implant, loss of available articular surface can be substantial.[5]

The normal variability of the humeral neck–shaft angle creates some difficult choices with respect to prosthetic design. An implant with a fixed neck–shaft angle can result in an anatomic reconstruction of the articular surface only if its neck–shaft angle matches the neck–shaft angle of the natural humerus in which it is being implanted. Differences in neck–shaft angle between the implant and the

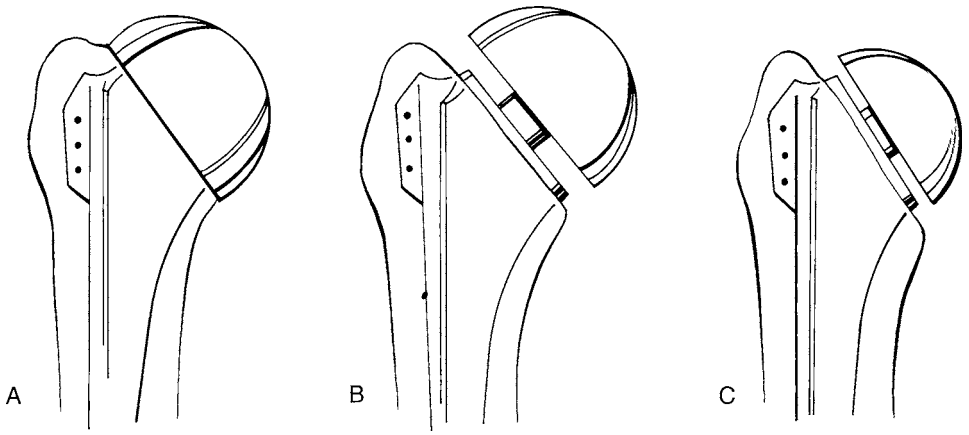

Figure 19-15 **(A)** A prosthesis whose neck–shaft angle matches the neck–shaft angle of the native humerus and whose articular surface rests flush against the cut surface of the humerus offers the most anatomic reconstruction with maximal surface area for contact with the glenoid. **(B)** A modular prosthesis that has a large gap between the head and the collar can overstuff the joint and also lead to internal component impingement because of diminished surface area for contact, even if the neck–shaft angle and head size are anatomic. **(C)** If the prosthesis is undersized to account for the gap between the head and the body, the tuberosity to head distance is altered (i.e., the greater tuberosity is prominent) and the surface area available for glenoid contact is diminished.

natural humerus cannot be corrected by changing humeral head thickness, radius, or offset without placing the articular surface of the head in a nonanatomic location or changing head volume.[86] If the neck–shaft angle of the implant is less than the natural humerus' (i.e., varus cut), the position of the prosthetic humeral head will be too low on the humeral metaphysis and the greater tuberosity will be too high (Fig. 19-16). If the prosthetic neck–shaft angle is more than the natural humerus' (i.e., valgus cut), the articular surface will be located superior and lateral to the anatomic location. Furthermore, the joint will contain the volume of the remaining natural head as well as the added volume of the prosthetic head. This overstuffing cannot be avoided by decreasing the humeral head size without also decreasing the surface area available for contact.[86]

Ideally, the prosthetic humeral head should be placed in the center of the cut surface of the humeral metaphysis. Since the center of the humeral head often does not coincide with the central axis of the intramedullary canal of the humeral diaphysis, the prosthetic humeral head must also be offset with respect to the intramedullary canal. This requires that the prosthetic humeral head be offset with respect to the prosthetic humeral body or that the prosthetic head and body together be offset with respect to the intramedullary canal. The latter scenario can be accomplished by undersizing the prosthetic humeral body and cementing it in the intramedullary canal so that the head and body are centered on the cut surface of the metaphysis rather than within the intramedullary canal. The former situation can be accomplished through prosthetic design by providing a coupling mechanism, such as an offset taper, between the humeral head and body that allows the surgeon multiple placement options of the head on the body.

The relationship between prosthetic anatomic variability (i.e., variable neck–shaft angle, variable humeral head offsets, etc.) and accuracy of humeral reconstruction with press-fit humeral stems has been highlighted by Pearl and colleagues.[84,85] They used a computerized optimization algorithm and cadaveric anatomic data to show that four commonly used press-fit humeral stems with fixed neck–shaft angles and one medial–lateral offset per humeral head size were unable to accurately reconstruct the humeral articular surface.[84] More specifically, despite optimized stem and head placement, the humeral head center was displaced 14.7 mm (range, 3.3 to 31.4 mm) from its original position and resulted in a decrease in the size of the humeral articular surface arc of 26 degrees (range, 11 to 41 degrees). In comparison to the position of the native humeral head, the optimized prosthetic head was smaller and shifted superiorly up the slope of the humeral osteotomy.[84] The addition of greater prosthetic anatomic variability in the form of variable neck–shaft angles and humeral head offsets resulted in more accurate recreation of the humeral articular surface, with center of

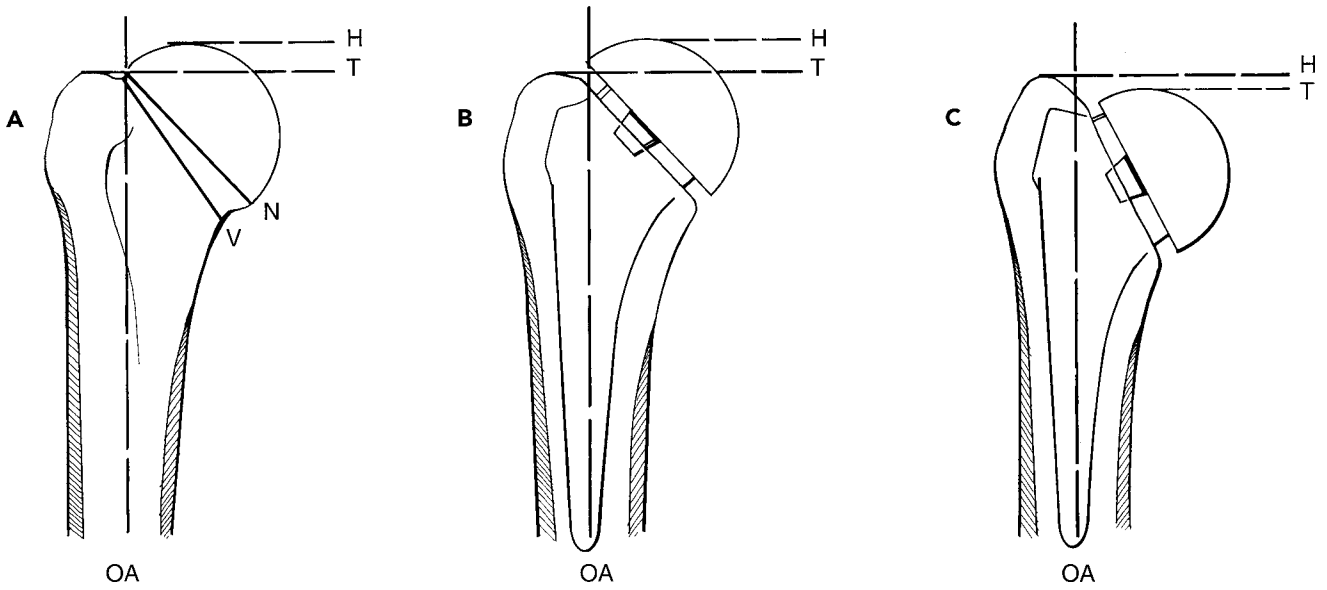

Figure 19-16 The greater tuberosity to head distance (*HT*) can be altered by the type of humeral osteotomy performed—normal (*N*) or varus (*V*) **(A).** If the neck–shaft angle of the prosthesis matches the neck–shaft angle of the native humerus, the humerus undergoes a normal cut (*N*), and the prosthetic humeral head is the appropriate size, the greater tuberosity to head distance will be anatomically reconstructed **(B).** However, if the prosthesis has a neck–shaft angle that is less than the neck–shaft angle of the native humerus and calls for a varus cut (*V*), the greater tuberosity to head distance will be less and may even become negative, even if the head is anatomically sized **(C).**

rotation displacement of 2.1 mm and decrease in surface arc of 12 degrees.[85]

The consequences of nonanatomic placement of the humeral head with respect to the humeral diaphysis are unknown. However, several potential problems can be identified. First, if the prosthetic humeral head is malpositioned anteriorly on the humeral metaphysis, the edge of the implant could overhang the anterior cortex of the humeral metaphysis and produce excessive tension on the subscapularis repair (Fig. 19-17). In addition, the anteriorly displaced humeral head will leave the posterior portion of the metaphysis uncovered. When the humerus is placed in abduction and external rotation, this uncovered humeral metaphysis may impinge against the posterosuperior glenoid and interfere with further motion or cause damage to the glenoid component (see Fig. 19-17).[54] Inferior malposition of the humeral head results in a decreased or reversed greater tuberosity to humeral head distance. When the humeral head is then centered within the glenoid, the greater tuberosity may impinge against the acromion or coracoacromial ligament (Fig. 19-18).

If one assumes that anatomic reconstruction of the humerus is a desirable goal, the question of exactly how anatomic the reconstruction needs to be still remains. In one cadaver study, as little as 4 mm of inferior displacement of the humeral head resulted in abnormal subacromial contact.[122] In addition, articular malposition of 4 mm in any direction resulted in small changes in range of motion and translation of the humeral head during both active and passive joint positioning.[122] The authors recommended reconstruction of the humeral articular surface to within 4 mm of the native humerus to minimize subacromial contact and maximize range of motion.[122]

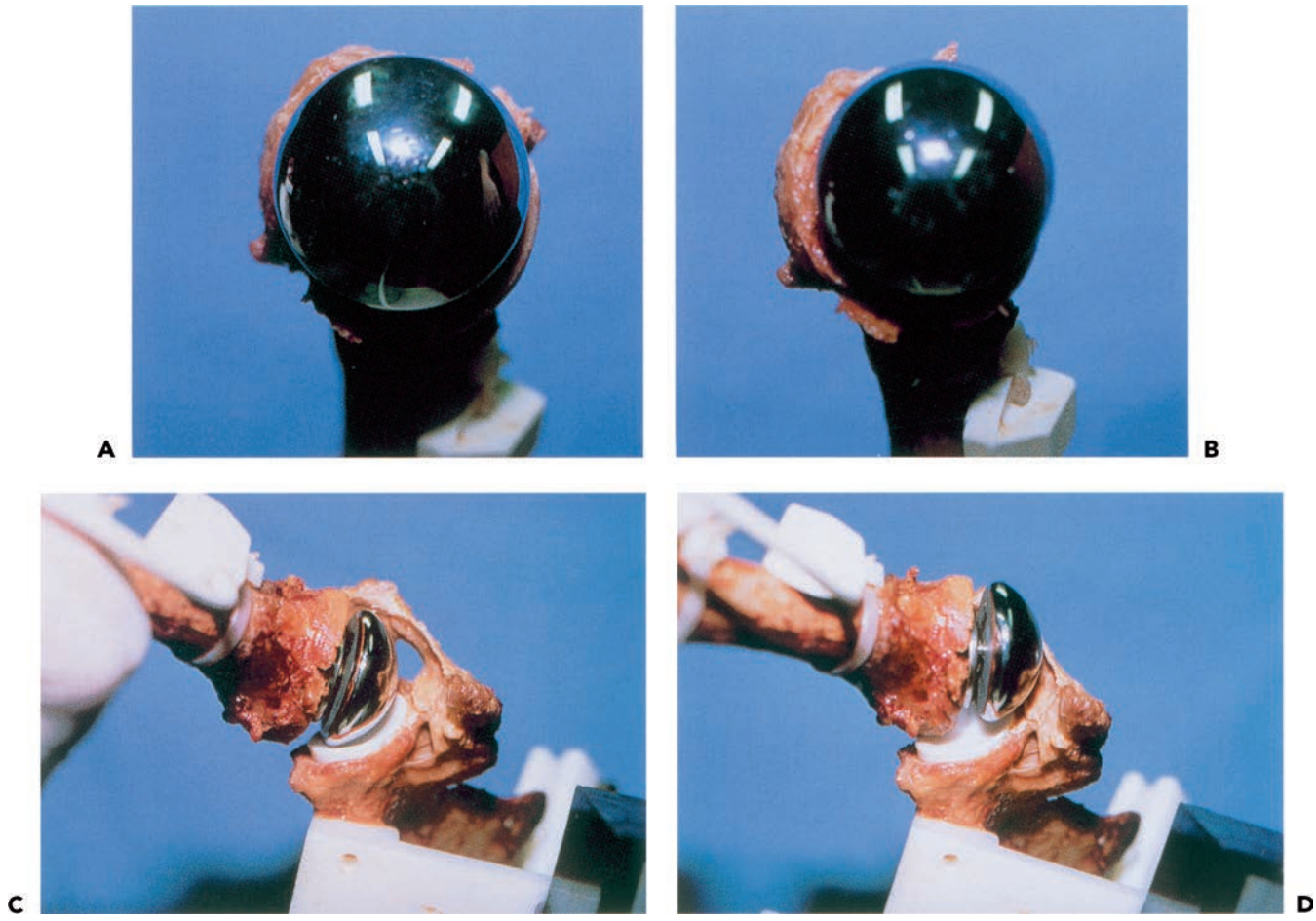

Figure 19-17 **(A)** Ideally, the prosthetic humeral head should be positioned in the center of the cut surface of the humeral metaphysis. **(B)** If the humeral head is offset anteriorly with respect to the metaphyseal center, it may overhang the anterior cortex of the humeral metaphysis and place tension on the subscapularis repair. **(C)** A head that is centered on the metaphysis allows full abduction and external rotation without impingement of the nonarticular portion of the humerus against the glenoid component. **(D)** An anteriorly offset humeral head leaves the posterior portion of the metaphysis uncovered, which can impinge against the glenoid component when the arm is positioned in abduction and external rotation.

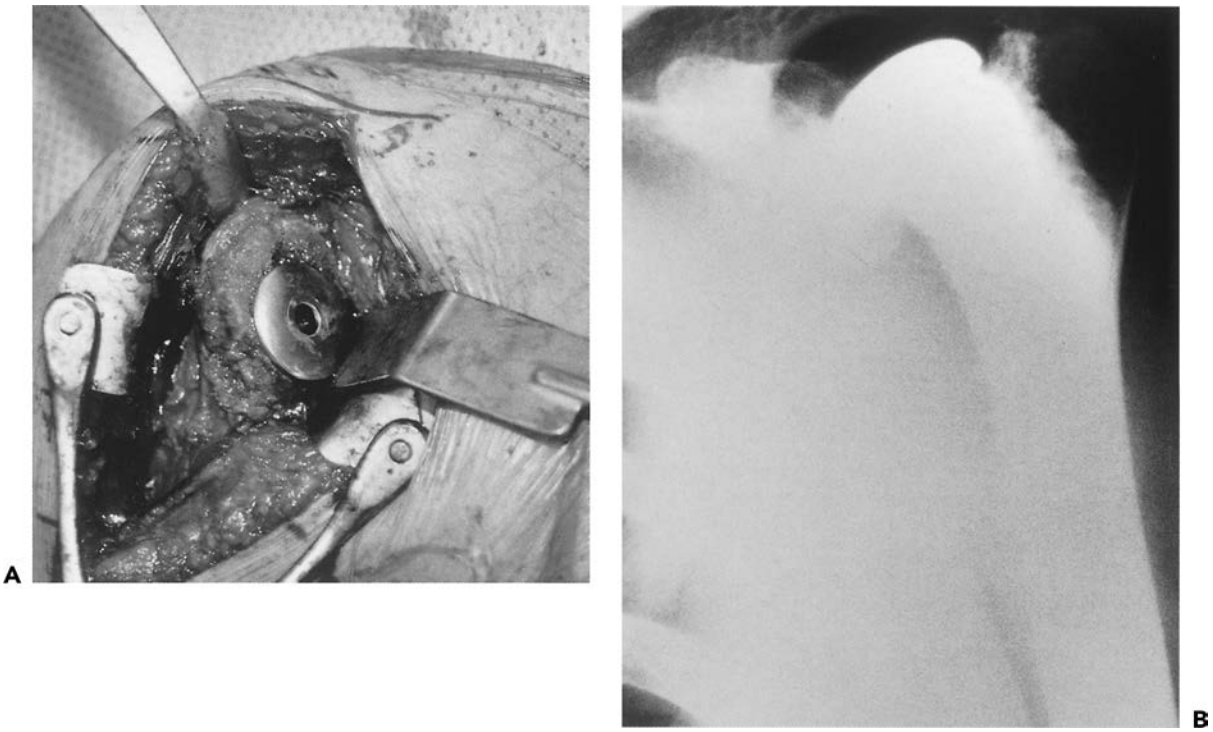

Figure 19-18 **(A)** Inferior placement of the humeral stem intraoperatively may occur from a number of causes, most commonly a varus cut. **(B)** This will lead to a reversed greater tuberosity to head distance and potential subacromial impingement.

The number of glenoid sizes required to fit all glenoids is probably less than the number of humeral head sizes required to fit all humeri. However, if articular mismatch is considered to be important and is to remain constant, a matching glenoid must be available for every humeral head. If articular mismatch were to remain within a narrow range, rather than constant, fewer glenoids would be necessary.

The bone of the natural glenoid should support the prosthetic glenoid component in its entirety. A pear-shaped component would seem to offer the best opportunity to provide the largest amount of glenoid articular surface without overhanging the anterior or posterior margins of the superior portion of the natural glenoid. If the anterior–posterior dimension of the superior and inferior portions of the glenoid component is equal (i.e., the component is oval), the humeral head may articulate against the unsupported anterosuperior and posterosuperior portions of the component. This may result in bending moments at the component rim, which could stress the component anchoring point or deform the rim. In addition, the increased anterior–posterior dimension of the superior portion of an oval glenoid component may facilitate contact between the edges of the component and nonarticular portions of the proximal humerus. However, many, if not most, arthritic glenoids have lost their natural pear shape and are more oval. For these reasons, there is no clear consensus with regard to the use of either pear-shaped or oval glenoid components.

The concept of nonarticular portions of the prosthetic humerus contacting the glenoid component has been mentioned as a potential consequence of nonanatomic reconstruction. In the nonprosthetic shoulder, this phenomenon has been termed internal glenoid impingement.[53,54] Nonanatomic prosthetic designs may result in component impingement.[53,54] Nonanatomic features that may predispose to the development of internal component impingement include decreased humeral surface area, increased glenoid anterior–posterior size, and nonanatomic humeral head placement. These potentially predisposing factors are especially relevant when glenohumeral motion following prosthetic replacement is normal or near normal. In the presence of diminished glenohumeral motion, internal component impingement may only occur with large deviations in prosthetic sizing or placement.

Biomechanical Factors

Elastic deformation of the glenoid articular cartilage and labrum in the normal shoulder can accommodate the small humeral head translations observed during active motion with a normal rotator cuff and no capsular contracture. Polyethylene does not have the same loading characteristics as articular cartilage and labrum. In a completely conforming prosthetic articulation, humeral translation can only occur with subluxation of the humeral

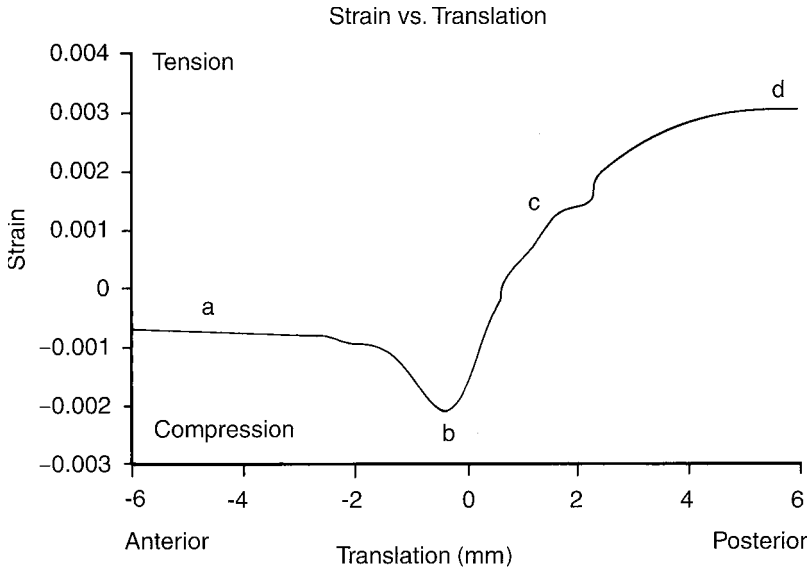

Figure 19-19 As the humeral head is translated from the anterior glenoid rim to the posterior glenoid rim, strain on the anterior surface of the keel changes. The strain begins as a compressive strain, which increases to a maximum when the humeral head is positioned over the deepest portion of the glenoid component. The strain reverses and becomes tensile and reaches a maximum tensile strain when the humeral head reaches the posterior glenoid rim. Increasing component conformity is associated with an increase in maximum tensile strain. Decreasing component conformity is associated with increasing maximum compressive strain. (Reprinted with permission from Karduna AR, Williams GR, Iannotti JP, Williams JL. Total shoulder arthroplasty biomechanics: a study of the forces and strains at the glenoid component. *J Biomech Eng*, 1998;120(1):92–99)

component and subsequent rim loading. This rim loading may result in permanent deformation of the glenoid component, unusual wear of the glenoid component, or rocking of the glenoid anchoring points.

Strain patterns on the surface of the keel of a cemented glenoid component are dependent upon articular conformity.[55] As a simulated humeral head is translated across the glenoid surface, from the anterior rim to the posterior rim, strain along the anterior surface of the keel starts out as mildly compressive and reaches a maximum compressive strain when the humeral head reaches the midpoint of the glenoid. As the humeral head continues to translate posteriorly, the strain becomes increasingly tensile until it reaches a maximum at the posterior glenoid rim (Fig. 19-19). The maximum tensile strain increases with increasing conformity, indicating the presence of an increasing bending moment. However, the maximum compressive strain observed when the humeral head is at the midpoint of the glenoid increases with decreasing conformity.[55] The clinical significance of these alterations in component strain is not currently known.

The force required for dislocation of a prosthetic glenohumeral joint with a constant joint reaction force is a function of component constraint (i.e., wall height), not component conformity.[57] If wall height and joint reaction force remain constant, the force required for dislocation of the prosthetic components remains the same, regardless of conformity. As articular conformity decreases, the amount of humeral translation permitted before the glenoid wall is encountered increases. However, continued translation to the top of the glenoid rim requires the same force, irrespective of decreasing component conformity (Fig. 19-20).[57]

Articular nonconformity of prosthetic components seems desirable if reproduction of normal glenohumeral kinematics is a goal of prosthetic reconstruction. However, the ideal amount of articular mismatch is not known. There is probably an acceptable range of articular nonconformity. Too much nonconformity may decrease contact area and increase contact stresses to a level that could threaten the integrity of polyethylene. In addition, given the small dimensions of the natural glenoid, a glenoid component that is both excessively flat and small enough to fit on the surface of the natural glenoid will have a diminutive wall height and may be prone to instability. Conversely, too little nonconformity limits humeral translation and encourages glenoid component rim loading. This results in increased tensile strain at the anchoring

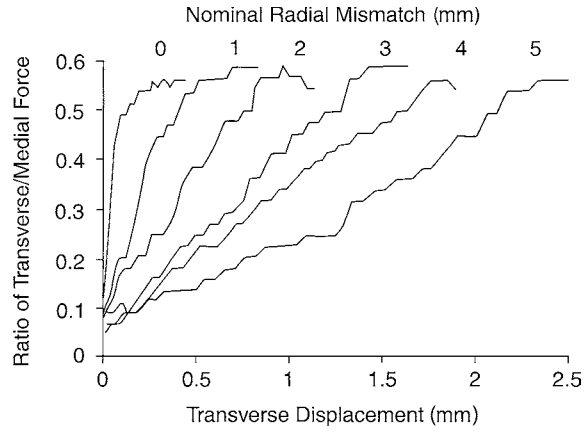

Figure 19-20 This graph plots force versus transverse displacement as a function of conformity. As component conformity decreases, there is more humeral head translation permitted for the same normalized displacement force ratio. However, the force required for maximum displacement (i.e., dislocation force) does not change appreciably. (Reprinted with permission from Karduna AR, Williams GR, Williams JL, et al. Joint stability after total shoulder arthroplasty in a cadaver model. *J Shoulder Elbow Surg* 1997;6:506.)

point of the glenoid component and may lead to increased component wear, peripheral component deformation, or early loosening.

Walch and colleagues studied the effect of prosthetic articular mismatch on the presence of glenoid radiolucent line formation postoperatively.[113] In this retrospective study of 319 total shoulder arthroplasties, a significant linear relationship was found between articular conformity and radiolucent line scores. Increasing nonconformity was associated with lower (i.e., better) radiolucent line scores, with the best scores associated with implants with radial mismatches of greater than 5.5 mm.[113]

Component Positioning and Fixation

As mentioned above, glenoid version shows substantial individual variation. However, on average, normal glenoid version is 0 to 2 degrees of retroversion.[19] In osteoarthritis, glenoid retroversion is increased, presumably as a result of posterior glenoid wear or congenital hypoplasia.[38,112] This increase in glenoid retroversion results in increased stresses to the posterior glenoid and posterior subluxation during external rotation.[18] In addition, increased glenoid retroversion may be a predisposing factor for anterior rotator cuff tears.[108] Therefore, except in the most severe cases of congenital hypoplasia, the goal of shoulder arthroplasty is to return glenoid version to normal so that the stresses on the glenoid and rotator cuff are normalized. This is accomplished most often through asymmetric glenoid reaming, glenoid bone grafting, or a combination of the two. Although the possibility of using a built-up glenoid component exists, currently there has been little experience with this technology.

In cases where the glenoid version cannot be normalized without glenoid bone grafting, altering humeral version has been suggested as an alternative. Theoretically, altering humeral version may alter the lines of action of the rotator cuff and improve function. However, since the prosthetic humeral head is a sphere with approximately 5 mm of offset from the stem axis in most arthroplasty systems, modest changes (i.e., 15 to 20 degrees) in humeral version will not change stability (Fig. 19-21).[102] Therefore, if correction of posterior instability in the presence of increased glenoid retroversion is desired, it must be accomplished by normalizing glenoid version.

Humeral component fixation can be accomplished through cemented or cementless means. In general, aseptic humeral loosening is an uncommon problem, especially with cemented components.[78,96] However, there have been reports of humeral component subsidence with press-fit cylindrical stems with proximal low profiles.[23,97] The use of implants with larger proximal profiles and the addition of surface coatings to encourage biologic in-growth have been shown to improve the rate of humeral lucency.[70,97,103] One study documented better performance using press-fit stems with a metaphysis-filling implant.[70] In this prospective study of 131 shoulder arthroplasties, no component subsidence was identified, 50 shoulders showed no radiolucency, 75 shoulders showed radiolucency around the distal tip, and two shoulders showed radiolucency around the metaphyseal region of the prosthesis. Only 11 radiolucencies were greater than 1 mm. Neutral stem alignment was less likely to be associated with radiolucency. The prevalence of radiolucency was unrelated to length of follow-up and the presence of a glenoid component.[70] Conversely, other studies have documented increased humeral radiolucency with longer follow-up and in total shoulder arthroplasty as compared to hemiarthroplasty.[97,103,104]

The prevalence of radiolucent line formation around the glenoid component is much higher than the humeral component and has been reported to occur in 30% to 96% of components.[11,22,33,47,65,73,105,109,113] Although the rate of clinical glenoid loosening (0% to 12.5%) is much lower than the rate of lucent line formation, a loose glenoid component is the most common prosthesis-related cause for revision surgery.[4,14,16,25,48,75,119,121,124] Certain aspects of glenoid preparation and fixation may influence the rate of lucent line formation and loosening. Initial stability of the glenoid component, which may be related to lucent line formation and loosening, is enhanced by concentric reaming of the glenoid bone surface so that its shape matches the back of the glenoid component.[26] Moreover, components with a curved back have been shown in one study to have better loosening performance than components with flat backs.[2] Finite element analysis comparing flat-back and curved-back glenoid components reveals that curved-back components have less shear stress and less slip than flat-back components and that these differences are increased if the component is malpositioned in retroversion (Fig. 19-22).[50] Consequently, most current arthroplasty systems provide specialized glenoid reamers to create such matching concave surfaces.

Methods of glenoid component fixation have evolved over time and will probably continue to evolve as long as glenoid loosening remains a prominent cause of failure. Neer's original glenoid component was a cemented, all polyethylene design with an axisymmetric keel with respect to the frontal plane.[76,77] It was introduced in 1973 and became the most popular type of glenoid component for many years.[77] Changes in keel geometry, specifically placing the keel in an offset position with respect to the central axis of the articular surface, could potentially improve loading characteristics.[74,83] The rationale for an offset keel design is that the deepest portion of the glenoid vault lies anterior to the central axis of the articular surface.[74] When the keel is centered to the articular surface, it may contact the posterior cortical bone of the vault.[74] A stress analysis of an anterior offset keel design using finite element modeling showed that, under a variable joint load, the stresses in the offset keel design were lower than

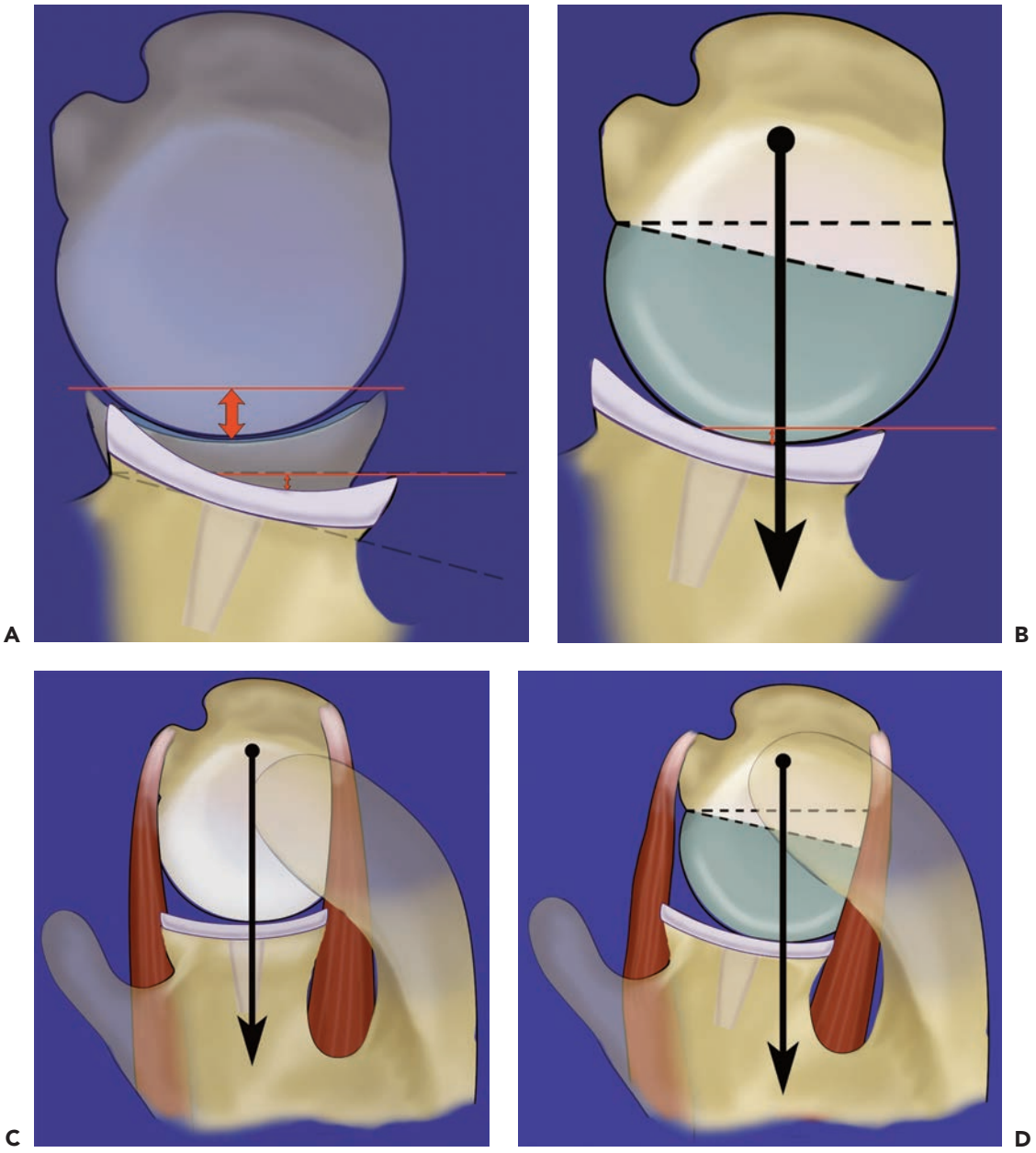

Figure 19-21 When the glenoid is retroverted, stability is not increased by decreasing humeral retroversion by 15 degrees. This is likely due to the fact that the functional wall height of the glenoid component is decreased with glenoid retroversion as the humeral head shifts posteriorly with the glenoid component **(A,B).** This shift is unchanged with changes in humeral component version. In addition, the force vectors of the rotator cuff muscles are not affected by changes in humeral component version as only the head is rotated and the position of the tuberosities to the scapula remain the same, resulting in a more posteriorly directed vector **(C,D).** (Reprinted with permission from Spencer EE Jr, Valdevit A, Kambic H, et al. The effect of humeral component anteversion on shoulder stability with glenoid component retroversion. *J Bone Joint Surg Am* 2005;87:808.)

those in a conventional center-keel design.[74] This is thought to be due to the anterior placement of the keel resulting in the keel being positioned more directly under the line of action of the maximum glenohumeral force (90 degrees of abduction). This ultimately results in less bending of the component. The offset keel has lower cement stresses than the center keel, as the cancellous bone in the posterior region of the glenoid has been shown to have a higher stiffness when compared with the anterior region. Also, insertion of the offset keel prosthesis results in removal of low stiffness bone, leaving the higher stiffness bone of the posterior region to support the prosthesis.[74]

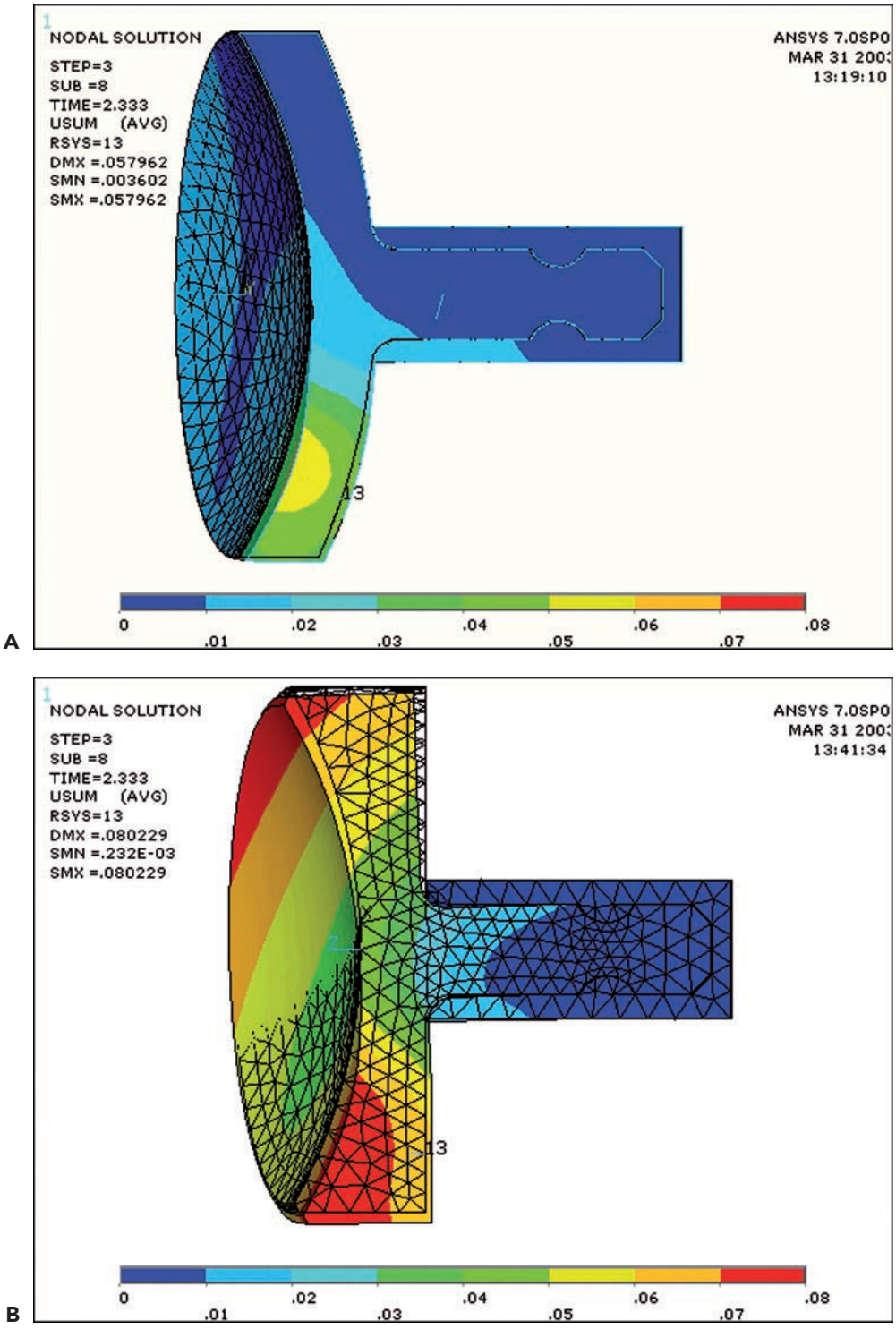

Figure 19-22 Finite element analysis reveals increased component shift in flat-back **(A)** versus curved-back **(B)** glenoid components. The increased amount of green, yellow, and red in A indicates greater amount of shift. These differences are increased when the component is malpositioned in retroversion. (Reprinted with permission from Iannotti J, Spencer EE Jr, Winter U, et al. Prosthetic positioning in total shoulder arthroplasty. *J Shoulder Elbow Surg* 2004;14:111S.)

Despite these potential advantages, offset keel designs have not gained in popularity.

More recently, glenoid component fixation using multiple pegs—rather than a keel—has been introduced. The peg-shaped anchorage systems provide greater stability against shear forces. As long as they are spaced far enough apart, pegs in any implant act individually to resist shear.[41] Anglin and colleagues documented better loosening performance in pegged versus keeled designs.[2,3] Lazarus and colleagues evaluated the radiographic outcomes of cemented pegged versus cemented keeled glenoid components in total shoulder arthroplasty.[65] They concluded that radiolucencies at the cement–bone interface and incomplete component seating occurred more frequently with keeled components versus pegged components.[65]

Wirth and colleagues also compared a pegged component with a central specialized cementless peg versus cemented keeled components in a canine model.[123] They found that the histology and the radiographs for the keel design supported the conclusion that the implant was not well integrated into the bone. The peg design allowed a "biologic" fixation between the implant's central peg and bone (Fig. 19-23). The fixation strength of the pegged design more than doubled by 3 months after surgery. The pegged glenoids demonstrated excellent osseous integration, adaptation, and increased bone density.

Parametric studies have evaluated the influence of fixation peg design on implant stability.[41] The parameters included were number and size of the fixation pegs as well as the aspect ratio (length/diameter). Five peg geometries of various shapes and sizes were tested for sheer stability. The results suggested that components with multiple small pegs create a uniform stress distribution in the anchoring material and provide more sheer stability per unit volume than implants with fewer, but larger pegs.[41] In addition, pegs with a rough surface rather than a smooth one have better fixation strength.[2] Because of these potential advantages, most current arthroplasty systems offer both keeled and pegged glenoid component designs.

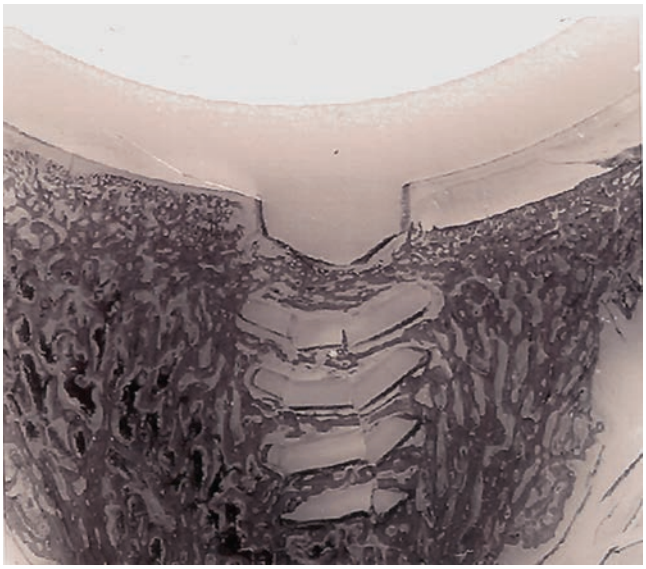

Figure 19-23 Histology reveals bone that has grown between the flutes of the central peg, presumably yielding improved fixation over time. (Reprinted with permission from Wirth MA, Korvick DL, Basamania CJ, et al. Radiologic, mechanical, and histologic evaluation of 2 glenoid prosthesis designs in a canine model. *J Shoulder Elbow Surg* 2001;10:140.)

Cementless glenoid component fixation offers the theoretical possibilities of preservation of glenoid bone and biologic fixation that improves over time. Except for the component described by Wirth and colleagues above,[123] most cementless designs utilize metal backing—often with textured surfaces to encourage biologic in-growth.[11,23,24,39,71,121] The obvious advantage of cementless, biologic in-growth technology is permanent fixation of the glenoid implant to bone. However, some potential disadvantages of metal backing also exist. Given the difference between the modulus of elasticity of glenoid bone and metal, the potential for stress shielding and loss of glenoid bone stock exists,[106,107] although one study suggested the possibility of improved stress transfer to bone with metal backing.[83] The most important problem, which has yet to be solved, is the effect on articular surface (i.e., polyethylene) wear of metal backing. Metal backing causes an increase in contact stresses and potentially a higher wear rate.[106,107] In addition, many metal-back designs utilize a metal tray fixed to the glenoid bone, into which a polyethylene component is placed. This two-piece design may result in significant additional wear between the polyethylene component and the metal (i.e., backside wear), resulting in the generation of wear debris that may lead to early loosening or polyethylene failure. Metal-back components with a one-piece design, in which the articular surface is permanently bonded to the metal, may eliminate backside wear. However, when the articular surface wears out, retrieving the permanently fixed metal backing may be difficult. Research into biologic fixation options will undoubtedly continue because of the potential advantages.

The vast majority, if not all, of currently available nonconstrained total shoulder prostheses have articular surfaces consisting of a metal (usually cobalt-chrome alloys) humeral head and a polyethylene glenoid. Since the ultimate cause of failure of properly implanted total shoulder prostheses is articular surface (i.e., polyethylene) wear, interest in alternative articular surfaces with superior wear characteristics has surfaced. Alternative bearing surface materials include cross-linked polyethylene, ceramics, and highly polished metals. The wear characteristics of these materials exceed traditional polyethylene. Glenoid components made from cross-linked polyethylene will soon be commercially available. However, further work is required to determine the exact role of alternate bearing surfaces in total shoulder arthroplasty. Some of the anatomic

requirements of shoulder prosthetic components, such as the relative thinness of the glenoid component, make the designing of alternative bearing surfaces difficult. The interest in alternative bearing surfaces will undoubtedly continue with the goal of identifying articular surface materials that will last the patient's lifetime and lend themselves to biologic fixation options.

IMPLICATIONS FOR CONSTRAINED PROSTHETIC DESIGN

Nonconstrained implants, as described in the sections above, are indicated when the rotator cuff is intact, when small tears can be reconstructed, or when there is enough rotator cuff remaining, so that the proximal pull of the deltoid can be converted into a rotational moment through the establishment of a fulcrum between the humeral head and glenoid. In the normal shoulder, this is done very effectively, since glenohumeral motion approaches ball-and-socket kinematics. In some cases of rotator cuff deficiency, there is more than the 2 mm of translational motion seen in normal shoulders but a fulcrum is eventually established and the arm can be raised. In other cases, particularly if there is coracoacromial arch insufficiency, deltoid contraction results in almost no rotational motion and the humeral head translates anterosuperiorly, out of the glenoid fossa. When arthroplasty is required under these circumstances, replacement with nonconstrained implants, even with tendon transfers and partial rotator cuff repairs, does not predictably result in improved function. The ability to create a glenohumeral fulcrum is irretrievably lost. Humeral hemiarthroplasty may result in some pain relief but function will remain severely compromised.

Renewed interest in implants with a fixed-fulcrum design has occurred because of the failure of nonconstrained devices to produce improvement in function in patients with arthritis and massive rotator cuff deficiency.[13,15,30,31,36,40,42,52,64,94,98,111,120] Constrained arthroplasties of various designs were performed decades ago with high rates of loosening and component failure.[17,29,34,62,66,67,90–93] It is thought that one potential factor important in component failure was the placement of the center of rotation markedly lateral to the glenoid, thus imparting substantial bending moments to the glenoid anchoring points. The more recent designs have used a reverse ball-and-socket design, with the ball on the glenoid side and the socket on the humeral side.[36,42] This renewed interest was initially driven by Paul Grammont, a French orthopaedic surgeon.[42] The important features of his prosthesis are a relatively large, low-profile (i.e., no neck) ball whose center of rotation lies within the glenoid vault and a relatively horizontal (155 degrees) inclination of the humeral cup compared to the normal neck shaft angle (Fig. 19-24).[42] These features result in shifting the center of rotation distally and medially, thereby improving the moment arm and strength of the deltoid, on which elevation and function of the arm depends. The relative valgus inclination of the humeral cup resists dislocation of the implant during early elevation.[13] However, this combined with the relatively low profile of the ball results in impingement of the polyethylene liner of the humeral cup against the inferior glenoid.[80] This contact between the polyethylene liner and the scapula may result in premature, localized polyethylene wear and notching. This impingement can be theoretically improved by lateralizing the instant center of rotation slightly[36] or by moving the ball inferiorly.[79]

Although the relative experience with these implants is short term, the initial results are very promising in a difficult group of patients that have few, if any, other options. There is reliable improvement in function and pain. The complication rate is higher than with nonconstrained implants, but not nearly as high as previous constrained devices.[8,15,36,42,52,94,98,111,120] Further follow-up and research is required to determine the durability of these implants, the significance of inferior notching in the medialized design,[13,42] any differences between the medialized[42] and lateralized[36] designs with respect to results and complica-

Figure 19-24 Grammont's reverse arthroplasty (Delta Prosthesis, Depuy, a Johnson and Johnson company, Warsaw, IN) consists of a relatively large glenoid sphere, whose center of rotation lies within the glenoid vault, and a relatively horizontal (i.e., neck–shaft angle 155 degrees) humeral cup.

tions, and the need for any additional design changes. However, the early results of these modern reverse prostheses justify continued work, since they provide improved elevation and strength in a way that replacement with anatomic, nonconstrained devices and soft tissue reconstruction cannot.

SUMMARY

Glenohumeral component design should be based on known anatomic and biomechanical relationships of the normal shoulder. The goals of prosthetic reconstruction of the glenohumeral joint should continue to be anatomic reconstruction of the articular surfaces with restoration of normal glenohumeral kinematics. Given the severe soft tissue and occasional osseous abnormalities that are encountered in patients with glenohumeral arthritis, this goal may not be attainable in all patients. However, so long as bone stock and soft tissue quality are adequate, prosthetic designs that most closely mimic normal anatomy and allow the most intraoperative flexibility with regard to prosthetic sizing and placement are likely to improve our chances of attaining these goals. The important features of most current anatomic implant systems are modular, anatomically correct humeral heads with offset capabilities; press-fit stems with porous coating and/or proximally filling geometries; low-profile, smooth, distally tapered stems for use with cement; and all polyethylene glenoids with both keel and peg configurations. Intense investigation continues with regard to cementless glenoid fixation and alternative bearing surfaces. For shoulders with such severe soft tissue destruction that anatomic reconstruction will not result in improved function, reverse arthroplasty shows good early promise. However, it should be remembered that shoulder replacement is and always will be a technique-dependent process. Therefore, no prosthetic implant can substitute for good surgical technique.

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20 Alternatives to Replacement Arthroplasty for Glenohumeral Arthritis

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INTRODUCTION

The last century has witnessed the evolution of shoulder arthroplasty from simply a novel surgical technique to the current standard for restoring function in patients with debilitating end-stage glenohumeral arthritis. Unfortunately, as with any artificial prosthetic device, results are not perfect and there also exists a substantial population that may not be ideal candidates for prosthetic replacement of the glenohumeral joint. Such patients include those with coexisting medical morbidities that would prevent arthroplasty, and those young and active patients in whom a prosthetic replacement may be subject to excessive risk of failure. Even as the search for the ideal prosthetic arthroplasty has continued, it is this challenging group of patients that has

driven the exploration of nonprosthetic treatments for glenohumeral arthritis. Furthermore, until shoulder replacements reach the utopian ideal of perfect functional restoration with essentially no risk of prosthetic complication, surgeons should explore nonprosthetic alternatives in an attempt to provide both longitudinal pain relief and durable functional restoration (see Treatment Algorithms I and II).

Causes of Pain in Glenohumeral Arthritis

The results of nonprosthetic management of shoulder arthritis are quite variable depending on the cause of the arthritis. Because of this, it is important to understand the causes of pain in both osteoarthritis and rheumatoid/inflammatory arthritis.

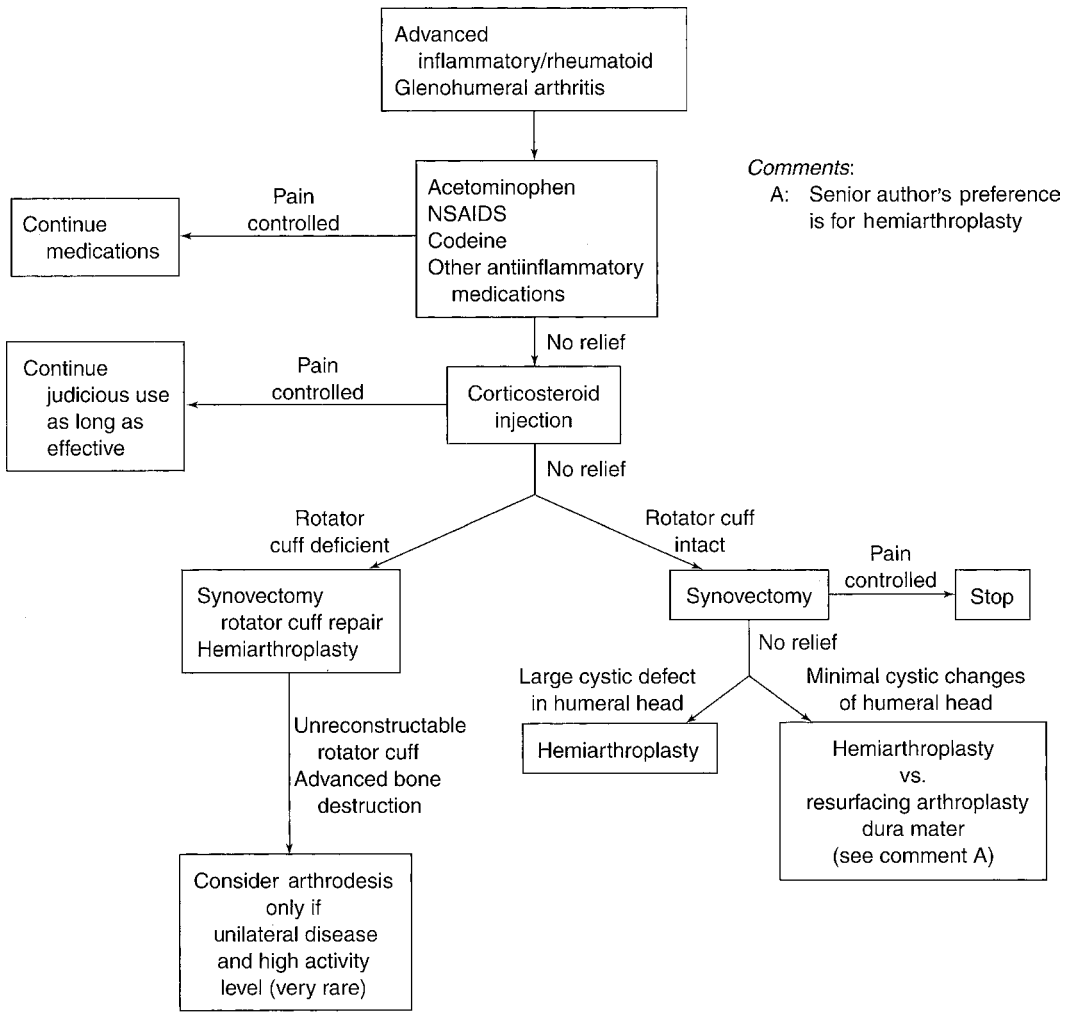
Osteoarthritis

The relative importance of factors that control the development of symptoms in osteoarthritis (OA) is still disputed.[24,25,27] Although roentgenographic features seen in this disease primarily result from the loss of articular cartilage and changes in the adjacent bone,[96] this disease process affects all periarticular tissues.[12,53,86] To some extent this may explain the often poor correlation between clinical symptoms and roentgenographic changes in various arthritic joints.[14,53] Many patients with this disease have mechanical, inflammatory, and psychological components to their pain.[22,107]

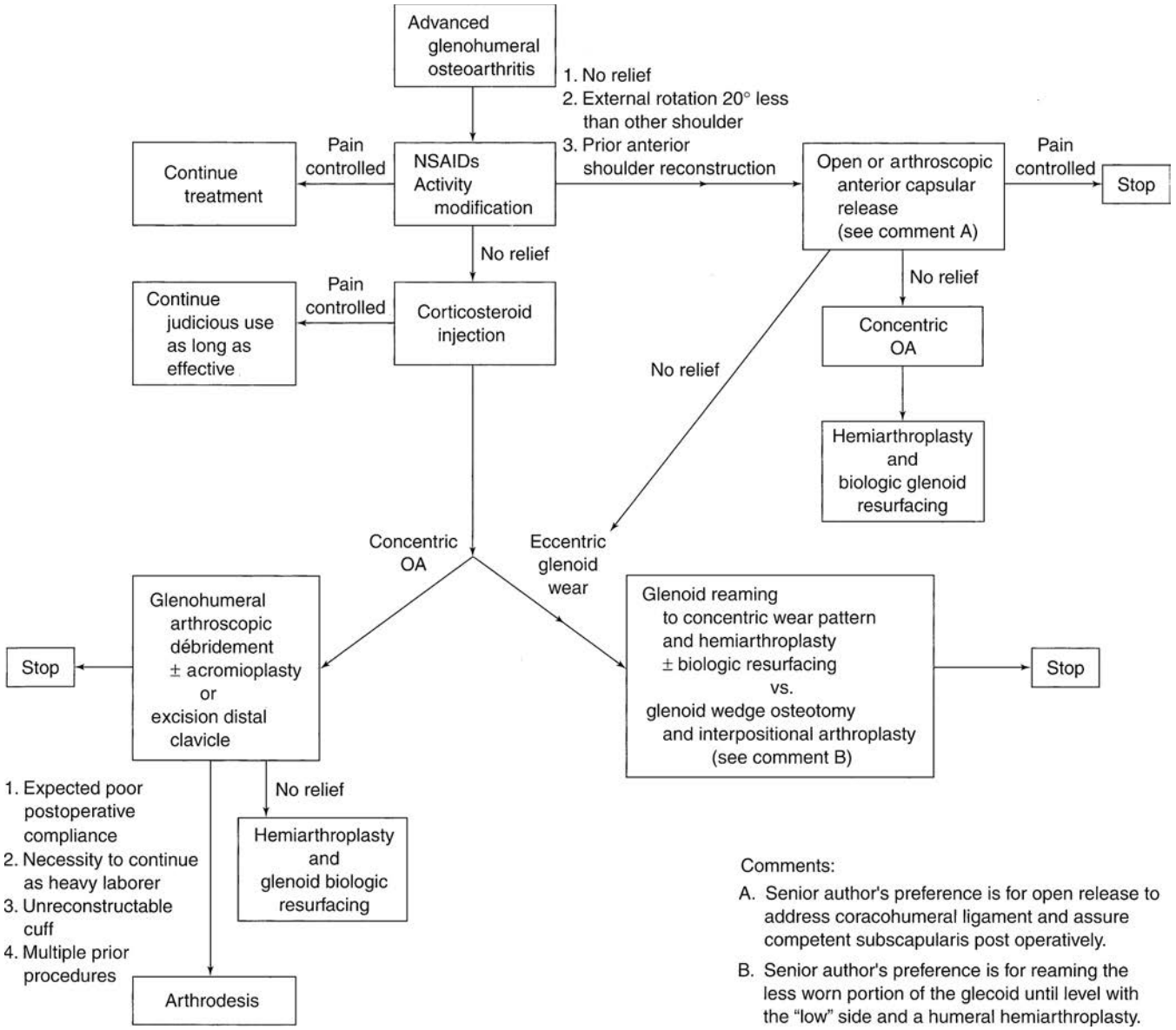
The shoulder joint is an uncommon site of primary OA, except in elderly women.[20] However, in relatively young individuals, OA of the shoulder may be seen as a consequence of trauma.[84] Specifics of the pathoanatomy of this posttraumatic diagnosis have been detailed by Neer.[72]

Rheumatoid and Other Inflammatory Arthritides

Glenohumeral joint involvement is common in patients with rheumatoid arthritis (RA), and is usually part of a polyarthropathy.[83] Laine et al.[99] defined the spectrum of



ALGORITHM 20-I
Algorithm for treatment of inflammatory arthritides. NSAIDs = nonsteroidal antiinflammatory drugs.



ALGORITHM 20-II
Algorithm for treatment of glenohumeral osteoarthritis. NSAIDs = nonsteroidal antiinflammatory drugs; OA = osteoarthritis.

shoulder disease in 277 hospitalized patients with RA.[52] Glenohumeral arthritis was detected in 47% of patients and many also had symptoms arising from the coracoacromial arch. Of note, 16 (6%) patients, with a mean age of 31 years, had shoulder arthralgia without demonstrable roentgenographic changes. Petersson similarly documented that 91% of patients with RA reported shoulder problems.[83] Thirty-one percent of these patients had such severe shoulder disability that they considered it to be their main problem.

Patients with symptomatic RA of the shoulder are typically rheumatoid-factor–positive women between 35 and 55 years of age. The destructive process may be quite advanced before significant symptoms are noted.[18] In addition to loss of motion, two common complaints in patients with RA are fatigue and muscle weakness. Rotator cuff defects occur in approximately 25% of these patients.[30,88] Many symptomatic patients with early stages of arthritis, regardless of cause, appear to benefit from noninvasive forms of therapy.

NONOPERATIVE TREATMENT

Education

An initial explanation of the arthritic process and discussion of its probable future implications is an important

first step in treating all patients. This helps ensure that patients' expectations are consistent with their prognosis. Education and encouragement also promote compliance with appropriate exercise and avoidance of detrimental activities. The goals of treatment and anticipated improvements should also be discussed. Typically this means that patients may need to adjust expectations since they may not be able to regain previous levels of sporting and other physical activities. If a patient is a manual laborer, the early realization that he or she may be unable to continue this occupation will facilitate early retraining.

Patients with arthritis must be taught to interpret their own symptoms to provide information important in determining appropriate therapy. They should both contribute to the exercise plan and assume responsibility for its implementation. The patient's active contribution to treatment fosters a sense of control over the effects of the disease. An insightful essay by R. E. Jones[46] discusses how patient satisfaction is strongly influenced by the personality and manner of the physician, as well as the quality of the physician–patient relationship. Applying the basic principles discussed by Jones will help physicians teach these patients how to manage and best enjoy the highest quality of life that is possible through the technologic achievements of present-day operative and nonoperative interventions for their arthritic shoulders.

Physical Therapy

General Principles

Although nearly all patients with glenohumeral arthritis will benefit from physical therapy, those who benefit most begin with decreased range of motion and weakness in the presence of minor roentgenographic findings.[62] In some patients, especially those with painful inflammatory arthropathies, an initial period of rest may be beneficial. However, an excessive period of rest can contribute to muscle atrophy, joint contractures, and worsening functional abilities. The aim of physical therapy is to increase range of motion and strength.[55] Nevertheless, therapy should not be considered a failure if the result is only the maintenance of the existing range of motion. Exercises are designed specifically for the needs of each patient—this includes gentle passive motion and isometric strengthening.[55] Preferential strengthening of specific muscle groups is especially beneficial for patients with mild OA whose symptoms are exacerbated by instability.[23,36]

Although acting in nearly diametric ways, heat and cold are therapeutically useful when employed at appropriate times in chronic and acute phases of inflammatory pain.[54,56] Cold treatment is useful in treatment of acute inflammatory flare-ups; the analgesic effects have been attributed to the ability of cold to depress the excitability of nerve fibers and muscle spindles, thereby increasing the pain threshold.[51,98] Cutaneous vasoconstriction and resulting reduction in blood flow reduce edema even in deeper tissues.[110]

When inflammatory pain is chronic, then the application of heat, usually in the dry form, is useful in temporizing pain and enhancing joint motion by increasing tissue elasticity.[3,50,52,57,98] Increased tissue metabolic activity also accompanies the increased blood flow resulting from vasodilation. Heat can be delivered superficially by using hot packs, hot water, or convective fluid therapy, while therapeutic ultrasound can be used for deeper penetration (see later section).

Hydrotherapy

The therapeutic efficacy of water therapy cannot be overstated. This remains a mainstay in the rehabilitation of any glenohumeral joint, and especially in the arthritic one. Buoyancy effects of water help to reduce stress exerted on muscles and joints during therapeutic shoulder exercises.[87] The patient's exertion can also be regulated because of the direct relation between the speed of movement and the resistance encountered. Adjustments in water temperature and agitation enhance the beneficial effects of hydrotherapy.

Therapeutic Ultrasound

The clinical usefulness of therapeutic ultrasound is based primarily on its capacity to increase blood circulation and temperature in deep tissues. Tissue temperature can be elevated at depths up to 5 cm from the point of application on the patient's skin, and peak temperatures occur in bone.[48,49] When the treatment objective is to heat muscle tissue, the most effective modality currently available is shortwave diathermy.[98]

Application of heat and cold, hydrotherapy, ultrasound, and range of motion and strengthening exercises are commonly used modalities for helping restore mobility to the stiff shoulder.[45,55,87] These and other nonoperative therapeutic interventions are contraindicated in patients who have severe pain and a rapidly deteriorating condition.[98] This scenario could imply concomitant occult sepsis, especially in the rheumatoid patient.

Occupational Therapy

Functional limitations are second only to pain in frequency of complaints of patients with glenohumeral arthritis. Impaired ability to dress and perform personal hygiene are particular problems. Occupational therapists can assist these patients by providing assistive devices and by teaching alternative ways to accomplish these tasks. For patients who desire to continue their gainful employment, analysis of job requirements is helpful in identifying problematic work practices.

Antiinflammatory and Pain Medications

Oral analgesics, such as salicylates, acetaminophen, and codeine, can be very effective in treating arthritic pain. The role of nonsteroidal antiinflammatory drugs (NSAIDs) in treating patients with symptomatic inflammatory arthropathies is well established.[44] However, for use in OA, it is controversial whether NSAIDs are any better than simple analgesia.[24] NSAIDs may have deleterious effects on articular cartilage in addition to the risk of untoward effects on gastric, renal, and liver function, which are particular problems in elderly patients.[24,44] Nevertheless, in a randomized trial examining pain relief in patients with OA of the knee treated with NSAIDs versus simple analgesics, NSAIDs were clearly superior in providing better quality of life and pain relief at the time of true inflammation.[71] Recently introduced COX-2–specific inhibitors have produced controversy both with regard to efficacy as well as potential for exacerbating medical comorbidities. At this time, Food and Drug Administration (FDA) approval for such medications remains in force, though physicians should seek to utilize any NSAID or other medication both judiciously and appropriately.

Nonoperative medical management of patients with intractable arthritic shoulder pain can be difficult. They may be best treated initially with a trial of interventions managed by a team of specialists trained in treating chronic pain. These specialists can manage depression, manipulate medication intake, inject trigger points, perform nerve blocks, and administer other modalities including low-dose narcotic medication. Such low-dose narcotics have demonstrated symptomatic efficacy in treating elderly patients with chronic pain. A neurologic workup is also often necessary to rule out cervical radiculopathy, brachial plexopathy, reflex sympathetic dystrophy, or other disorders.

Corticosteroids

Local anesthetic injections are of great value in diagnosing shoulder conditions by localizing the anatomic site of the pain generators. Appropriate steroid injections have also been documented to have significant therapeutic value.[8,35] Although the use of intraarticular steroids can be effective in abating symptoms in joints afflicted with RA, they are generally of comparatively limited value in OA, typically providing relatively short-term relief of symptoms.

Dacre et al.[23] studied the prospective, randomized, blinded results of local steroid injections or physiotherapy in treating patients with painful or stiff rheumatoid shoulders. Sixty consecutive patients of similar age, sex, diagnosis, and disease severity were allocated into three groups to receive either local steroids, 6 weeks of physiotherapy, or both. Results showed that physiotherapy alone was just as effective as local steroid injections or a combination of these two methods. In the uncomplicated case, a local steroid injection was the most cost-effective treatment.

Similarly, recently published meta-analyses of the peer-reviewed literature regarding the use of corticosteroid injections used for periarticular and intraarticular shoulder pain document variable success.[8,11] Patients with subacromial pathology (such as rotator cuff disease) and patients with varying degrees of stiffness/capsulitis statistically benefited from use of corticosteroids. Results for treatment of OA remained difficult to interpret. Anecdotally, we have found that, if a single intraarticular water-soluble corticosteroid injection is helpful, then another can be performed at a 6-month interval (no more often than two injections per year). If the first injection provides minimal relief (assuming a truly intraarticular position of the injection—if there is any doubt, fluoroscopic imaging should be considered), then further corticosteroid injections are likely to have little potential benefit.

Viscosupplementation

Peer-reviewed publications over the past decade have documented the apparent statistical benefit of intraarticular injections of hyaluronic acid in the treatment of knee and hip osteoarthritis for pain relief.[53,109] Unfortunately, neither in vitro nor in vivo studies have demonstrated a chondroprotective effect of either low- or high-molecular-weight hyaluronic acid, and the likely effect of these substances on the human joint (if any) appears to be symptomatic relief of joint pain. Further work is ongoing regarding the use of intraarticular glucosamine as both a chondroprotective and pain-relieving agent.

Viscosupplementation of the glenohumeral joint has demonstrated much more variable clinical results. There remains no peer-reviewed, double-blinded, prospective, randomized clinical trial documenting any benefit of such injections when compared to simple corticosteroid injections in the shoulder. Nevertheless, as with any other treatment modality, there may still be a benefit in certain patients who seek to avoid operative treatment for their glenohumeral arthritis. These indications as yet have not been clarified.

Nutritional Supplements

The past 5 years have seen the introduction of many adjuvant nutritional supplements, or "nutraceuticals," for the amelioration of all musculoskeletal complaints including arthritis. Animal work has demonstrated a reduction in the clinical, inflammatory, and histologic components of arthritis with the use of both glucosamine and chondroitin sulfates in combination. The apparent mechanism of glucosamine/chondroitin is to favorably shift cartilage biology toward matrix synthesis instead of degradation.

Human clinical trials with regard to knee arthritis demonstrated little radiographic change, but symptomatic improvement of pain and function has been documented.

However, randomized controlled in vivo human studies are clearly necessary to evaluate the efficacy, long-term effects, and quality of these substances. No peer-reviewed report has documented the efficacy of these substances in patients with isolated glenohumeral arthritis, though patients with concomitant arthritis in other joints have anecdotally reported an overall relief.

OPERATIVE TREATMENT: ARTHROSCOPIC PROCEDURES

Arthroscopic Débridement

Arthroscopic débridement or lavage for arthritis has been successfully used in the weight-bearing joints of the lower limb, particularly the knee.[38,41,70] In many cases, this may be the result of a strong placebo effect.[63] In contrast to the knee, the benefits of, and indications for, arthroscopic débridement in the shoulder are not as clear, although most orthopedic surgeons know of anecdotal evidence suggesting that this can benefit some patients.[74,76] In some cases, arthroscopy will reveal previously unrecognized grade 2 to 4 osteochondral lesions.[37] Cofield reported[18] that in eight patients with glenohumeral arthritis, the use of arthroscopy confirmed or modified the diagnosis, or altered the course of treatment, in all cases. Ellman et al. reported[29] on 18 patients who underwent shoulder arthroscopy for impingement syndrome and, at surgery, were found to have coexisting glenohumeral degenerative joint disease, which was not apparent during preoperative, clinical, and roentgenographic evaluation.

Indications

While extrapolation of ideal indications remains difficult, arthroscopic débridement may be considered for patients with mild to moderate glenohumeral joint disease without structural alteration of the joint.[111] Those patient with mechanical symptoms secondary to loose bodies, or small lesions of the humeral head secondary to avascular necrosis, may also benefit. It is unlikely that any patient with motion loss or progressive joint alteration (such as biconcave glenoid erosion from OA) will benefit from simple joint débridement alone. There are also several reported cases in whom synovial chondromatosis of the shoulder, associated with arthritic changes, has been adequately treated by arthroscopic débridement.[19,81,103]

Surgical Technique

Standard glenohumeral anterior and posterior arthroscopic portals are utilized in either the beach-chair or lateral decubitus positions. While standard diagnostic glenohumeral arthroscopy is performed, special attention must be paid to the humeral and glenoid articular surfaces. Cartilaginous "lesions" or "flaps" should be gently débrided (often the use of a blunt-tip probe is enough) to prevent mechanical engagement. Exposed bony lesions should be identified and possibly considered for procedures such as microfracture or abrasion arthroplasty. The recess behind the subscapularis tendon and the axillary pouch (both anterior and posterior) should be carefully examined for loose bodies. Osteophytes can be removed from the inferior portion of the humeral head.

Postoperative Management

Immediate motion is paramount, and a sling should be used for no longer than 1 night (if used at all). Immediate physical therapy exercises emphasizing motion in external rotation and forward flexion are undertaken. Dietary modification to include glucosamine/chondroitin substances can be discussed.

Results of Arthroscopic Débridement

Generally, results of arthroscopic débridement for OA of the shoulder depend on the extent of degenerative changes.[40,66–68] Ogilvie-Harris and Wiley[75] reviewed 54 patients with OA of the shoulder who were followed for 3 years. When degenerative changes were mild, successful outcome occurred in two-thirds of cases; when changes were severe, successful outcome occurred in only one-third of cases. An additional group of patients who did well were those who had débridements of degenerative labral tears.

In another study, a group of 27 patients, with a primary diagnosis of degenerative joint disease of the glenohumeral joint, underwent arthroscopic débridement.[98] Average follow-up was 30 months (range 9 to 63), and their average age was 42 years (range 27 to 72). These patients presented with moderate to severe pain and had failed conservative treatment. The average time from onset of symptoms to surgical treatment was 24 months (range 3 to 60). Arthroscopic treatment included simple joint lavage, loose body removal, débridement of degenerative cartilage, débridement of labral or soft tissue, and subacromial space bursectomy. Although there were no significant changes in range of motion, a significant improvement in pain relief and function was obtained. Overall, there were 78% satisfactory results (excellent and good) and 22% unsatisfactory (fair and poor). There were no surgical complications. Sixty-seven percent of the patients involved in recreational sports (unspecified) were able to return to their previous activities. Of the unsatisfactory results, some pain relief was obtained in all patients for a minimum of 8 months before deterioration. Additional surgical treatment had been recommended for 11% of the patients. These authors concluded that in patients with mild glenohumeral osteoarthritis, with concentricity maintained, arthroscopic débridement is a useful

procedure. However, a subacromial procedure was included in all of these patients.

In contrast, Norris and Green[73] were less enthusiastic about the usefulness of arthroscopic débridement in the glenohumeral joint. In their series of patients, only a small percentage obtained any long-lasting pain relief. They concluded that arthroscopic débridement did not alter the natural history of glenohumeral arthritis. However, no subacromial procedures were performed in this series.

Capsular Releases

In 1990, Hawkins and Angelo[35] recognized OA in 10 patients (11 shoulders) who had had Putti-Platt capsulorrhaphies for anterior shoulder instability. They hypothesized that this resulted from excessive stress imparted to the cartilage during the abnormal glenohumeral motion caused by the excessively tight anterior capsule imbrication. In seven shoulders the complication was treated successfully with periodic administration of NSAIDs, supplemented by gentle range-of-motion exercises and occasional mild analgesic medications. The remaining four shoulders were treated operatively; two of these received nonconstrained total shoulder replacements. All four of these shoulders received anterior capsular releases for improving motion and decreasing abnormal forces on the glenohumeral joint. Hawkins and Angelo expected that this procedure would improve pain and retard the degenerative process in the two cases where it was done without resurfacing arthroplasty. With this rationale, it has been suggested that in similar cases in whom mild to moderate OA is associated with limited motion, especially external rotation, capsular releases should be considered.

Indications

Any patient with mild to moderate shoulder arthritis and restriction of motion, especially external rotation, relative to the contralateral side should be considered for arthroscopic capsular releases. Those patients with concomitant metabolic abnormalities (such as diabetes or thyroid abnormalities) and glenohumeral arthritis may be particularly responsive to capsular procedures that reduce the pain from the adhesive capsulitis component of their arthritic shoulders.

Surgical Technique

After routine diagnostic glenohumeral arthroscopy as described above, an ablation device is inserted into the anterior cannula and used to release the tight rotator interval capsule from the anterior edge of the supraspinatus to the superior edge of the subscapularis tendon until the fibers of the coracoacromial ligament can be seen. The coracohumeral ligament is released above the biceps tendon. The middle glenohumeral ligament is released from the edge of the labrum, and the anterior capsule is released adjacent to the labrum to expose the fibers of the subscapularis to approximately the 5 o'clock position to increase the amount of external rotation. The inferior capsule is left alone to protect the axillary nerve, as rupture of the inferior capsule will occur with the postarthroscopy manipulation in passive forward flexion. The arthroscope is then switched to the anterior portal for release of the posterior capsule if there is no evidence of posterior subluxation of the humerus on the glenoid (both radiographically and/or arthroscopically) and if there is a lack of internal rotation compared to the contralateral side. An angled incisor blade is then placed into the posterior portal and used to resect approximately 1 cm of posterior capsule adjacent to the labrum. The ablation device is then placed into the posterior portal and the release is completed to the 9 o'clock position. The arthroscopic instruments are then withdrawn, and the glenohumeral joint is passively manipulated into full forward flexion, full external rotation at side, full external rotation in the 90-degree abducted position, and full internal rotation in the 90-degree abducted position.

Postoperative Management

As this procedure is designed to increase motion, patients should be considered for a long-acting interscalene block or catheter (minimum 18 to 24 hours postoperatively) so that continuous passive motion (CPM) can be performed in the hospital. A CPM chair is utilized in the hospital and for 8 hours per day for the first postoperative week. Immediate physical therapy is also instituted, emphasizing external rotation, and no arm sling is used.

Results

Similar to what Hawkins reported, Ogilvie-Harris and Wiley[66] reported that in 54 patients with OA of the shoulder who were treated with arthroscopic débridement, 14 had an associated frozen shoulder. In these cases severely restricted motion was restored either by cutting or removing adhesions, or by manipulation. In general, if there was more than 20 degrees loss of passive motion compared with the opposite shoulder, then an arthroscopic capsular release was performed with symptomatic benefit.

We have reported on the use of capsular releases as a combined procedure for glenohumeral arthritis.[50] Thirty-three consecutive patients with a mean age of 62 years and minimum 2-year follow-up were evaluated. Excellent results were reported in 15 patients (46%) and good results in 10 patients (31%). Overall, 77% achieved a satisfactory outcome (excellent or good result). Five patients (15%) reported fair results and three patients (8%) reported poor results. Three patients required revision to arthroplasty at an average of 28 months (range 12 to 48) after arthroscopy. All three cases initially presented with a biconcave glenoid

as diagnosed by axillary radiographs. The mean patient pain score (0 = no pain; 10 = worst pain) improved from 6.3 preoperatively to 2.8 at final follow-up, with 80% of patients indicating that they would have the surgery again. Average postoperative motion increased for forward elevation by 42 degrees and for external rotation by 32 degrees. Worker's compensation patients obtained inferior subjective results overall; however, improvements in pain and function were found in all patients.

Subacromial Procedures

While a small percentage of patients with glenohumeral OA will have full-thickness tears of the rotator cuff, peer-reviewed reports in the medical literature have documented the efficacy of subacromial injections in providing pain relief for these patients. It does appear that there is a component of pain in the arthritic shoulder that can be ascribed to the subacromial bursa and space, and decompression of the acromiohumeral articulation can be beneficial for glenohumeral pain relief.

Indications

Any patient undergoing arthroscopic débridement of the arthritic shoulder, without a structurally important rotator cuff tear or cuff tear arthropathy, is a candidate for decompression of the subacromial space. Relatively osteopenic patients with inflammatory arthropathies should be judiciously evaluated prior to recommending this as part of the arthroscopic procedure.

Surgical Technique

After glenohumeral arthroscopy, the arthroscope is placed into the subacromial space and an anterolateral working portal (2 to 2.5 cm lateral and distal to the anterolateral corner of the acromion) is established. The ablation device is used to débride the coracoacromial ligament and periosteum off of the anterior acromion. An arthroscopic burr is used to flatten the undersurface of the acromion. We prefer not to resect the anterior acromion, but rather to simply create a flat acromion with an inferior acromioplasty. The arthroscope is transferred to the anterolateral portal and the burr to the posterior portal. The remaining inferomedial spur on the acromion is visualized and resected with the burr. An arthroscopic shaver is then placed into the posterior portal and a complete bursectomy (paying special emphasis to the "posterolateral gutter" between the infraspinatus muscle belly and the subdeltoid shelf) is performed.

Postoperative Management

Patients begin immediate motion as described previously. Special emphasis should be paid to heat and cold modalities in an attempt to reduce the subacromial space postoperative inflammation.

Results

The efficacy of subacromial decompression in providing pain relief in the face of established glenohumeral arthritis has been demonstrated in two recent studies. Simpson and Kelley[99] performed an acromioplasty and bursectomy in 24 patients with rheumatoid arthritis with radiographic evidence of advanced glenohumeral disease. They were able to achieve good pain relief and improved range of motion in 19 of these patients. Ellowitz et al.[31] also evaluated the results of subacromial decompression in a group of 21 patients who were noted to have Outerbridge grade IV osteoarthritic changes in the glenohumeral joint on preliminary arthroscopic evaluation. They reported uniformly good results and concluded that subacromial decompression provided adequate pain relief in this group of patients.

In our combined series, as reported above, all patients underwent a concomitant subacromial decompression with a positive impact on the functional results.

Surgical and "Medical" Synovectomy

Beneficial effects of synovectomy in the treatment of inflammatory arthropathies of some joints are well established.[18,46,100,101,113] The knee has historically been considered one of the most appropriate joints for this procedure, and open synovectomy was the means by which this was accomplished.[46,70,90] An early reported case of synovectomy in the shoulder was in 1965 when Wilkinson and Lowry reported on a series of 69 synovectomies, one of which was in a shoulder and was done without the arthroscope.[112]

In proliferative synovitis of the shoulder, surgical synovectomy gives good pain relief and increased mobility and function.[106,115]

Indications

Open synovectomy of the shoulder is no longer considered absolutely necessary unless carried out in conjunction with débridement of cystic bone lesions,[91] extensive disease,[73] rotator cuff repair,[66] or osteotomy (see later discussion); otherwise, arthroscopic synovectomy is adequate.[13]

Synovectomy done early in the inflammatory disease process will slow its progression; however, the procedure may need to be repeated several times, but it is associated with low morbidity. Although abnormal synovium may regrow after synovectomy for rheumatoid arthritis,[72] patients typically enjoy a relatively pain-free period with improved function.

In the early effusive stages of inflammatory shoulder disease or in OA with synovitis, synovectomy can also be accomplished medically by the use of sclerosing agents or

radiocolloids such as yttrium 90.[59,89] Yet these methods have an overall success rate of only about 50% and, similar to surgical synovectomy, often require multiple treatments. Radiocolloid synovectomy should be considered in patients who are not medically stable for arthroscopic shoulder synovectomy.

Surgical Technique

As described previously, routine glenohumeral arthroscopy is performed. An ablation device is utilized to remove synovitis from the glenohumeral capsule. Special care should be taken to avoid iatrogenic damage to either cartilage or tendon.

Postoperative Management

Management should follow guidelines previously indicated. For patients with aggressive and proliferative synovitis, consultation with a rheumatologist for medical management should be considered.

Results

In an attempt to better evaluate the results of open surgical synovectomy in late stages of rheumatoid disease, Tressel et al.[106] reviewed 75 cases in 53 patients with average follow-up of 6 years. Only 4% of shoulders had early-stage disease. Swelling and motion improved in approximately 70% and pain was diminished in approximately 50%, irrespective of the site and degree of joint destruction (Table 20-1). Overall, 75% of patients were satisfied with their results. The best outcomes were obtained when the shoulder joint alone was involved and the poorest outcome occurred with more extensive soft tissue involvement (e.g., adjacent bursa and rotator cuff).

In a study using yttrium 90 on various joints, Stucki et al.[100] reported poor results in the shoulder and suggested that this may be due to established pathologic involvement of the all-important rotator cuff, which would not be influenced by synovectomy.

TABLE 20-1
RESULTS OF OPEN SYNOVECTOMY IN LATE-STAGE RHEUMATOID DISEASE

Direction of Motion	Stages I and II (*n*=20)	Stage III (*n*=44)	Stage IV (*n*=11)
Flexion beyond 90°	85%	66%	45%
Abduction beyond 90°	70%	52%	66%
Internal rotation beyond 20°	75%	66%	80%

Authors' Preference for Arthroscopic Management

After failure of nonoperative measures, we prefer to attempt an arthroscopic management of glenohumeral arthritis if patients meet the following criteria:

1. Preservation of joint space on axillary lateral radiograph
2. No or minimal biconcave posterior erosion of the glenoid
3. Preservation of acromiohumeral distance greater than 6 mm
4. External rotation beyond the neutral (0-degree) position

Patients with inflammatory arthritis who have proliferative synovitis are also considered for arthroscopic management.

We prefer to perform four procedures with any arthroscopic débridement for glenohumeral arthritis:

1. Intraarticular débridement and/or synovectomy as necessary
2. Capsular releases
3. Subacromial decompression
4. Distal clavicle resection

The addition of the distal clavicle resection (5 mm in females and 7 mm in males) has been demonstrated by the senior author (WZB) to decompress the motion interface between the scapulothoracic articulations and the glenohumeral articulations, and may improve not only pain relief but also overhead motion in these arthritic patients through compensatory scapulothoracic motion.

OPERATIVE TREATMENT: OPEN SURGICAL PROCEDURES

Periarticular Osteotomy

Indications

Although uncommonly performed in the United States, periarticular osteotomy is a well-established treatment for symptomatic degenerative joint disease associated with biomechanically abnormal articulations.[114] In addition to abnormal transarticular force transmission, subchondral venous hypertension may be one of the causal factors in the symptoms associated with OA in these patients.[10] This hypothesis resulted in the development of osteotomies for decreasing venous pressure in joints afflicted with OA. Other suggested benefits of such osteotomies may be the enhancement of local perfusion that, presumably, accompanies healing of the osteotomy.

In general, periarticular osteotomy is not commonly performed and has not been reported by other surgeons in peer-reviewed publications. This is probably because endoprosthetic replacement provides more consistent pain relief and improvement in motion, and has a low reoperation rate.[18]

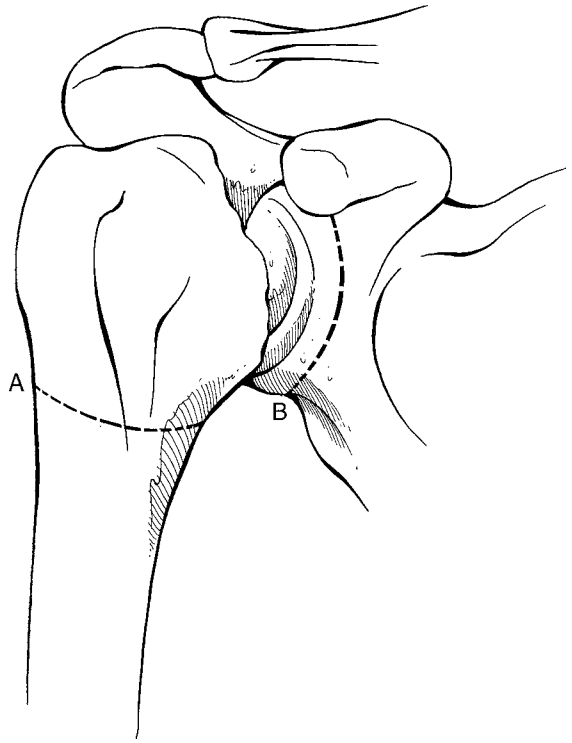


Figure 20-1 Schematic representation of the Benjamin double osteotomy. **(A)** Humeral osteotomy. **(B)** Glenoid osteotomy. (Reproduced with permission from Jaffe R, Learmonth ID. Benjamin double osteotomy for arthritis of the glenohumeral joint. In: Lettin AFW, Petersson C, eds. *Rheumatoid arthritis surgery of the shoulder. Rheumatology.* Basel: Karger, 1989;12:52–59.)

Surgical Technique

On the basis of these principles and noted benefit in arthritic knees, Benjamin et al. described a double osteotomy for the arthritic shoulder.[6] An anterior approach is used, and the subscapularis is incised medial to its insertion, exposing the neck of the glenoid. The glenoid is osteotomized 5 to 10 mm medial to the articular surface. The posterior cortex is not cut, but it is manually cracked so that the posterior periosteum maintains position and stability. Transverse osteotomy of the humeral neck is then performed just distal to the capsule; again the posterior cortex is cracked to preserve the posterior periosteal hinge (Fig. 20-1). The osteotomies are not fixed.

Postoperative Management

Passive shoulder motion is begun with 1 week, and activity is gently progressed.

Results

In Benjamin's series of 16 patients, the procedure was performed for OA in four cases, adult RA in 10 cases, and adult or juvenile RA in two cases.[7] Average patient age was 51 years; average time to evaluation was 2 years and 11 months. All of these patients reported substantial pain relief and had an average of 50 degrees' increase (range 10 to 150 degrees) in active abduction. The increased motion was from both scapulothoracic and glenohumeral joints.

When using this double osteotomy for arthritic shoulders, Jaffe and Learmonth[42] reported similar improvement in pain and active motion in 32 shoulders of 28 patients followed up for 8 to 72 months (mean 35 months). Twenty-seven patients had rheumatoid arthritis, four had degenerative arthritis, and one had avascular necrosis.

Tillmann and Braatz, however, achieved less improvement in their series of 24 surgeries carried out entirely for RA.[103,105] This discrepancy may be the consequence of the mixed diagnoses or the lower mean age (about 9 years) of patients in the study of Jaffe and Learmonth.[42]

With this procedure, long-term maintenance of pain relief and motion has been less than ideal.[61,94] Most authors suggest that this procedure should be reserved for relatively young patients with severe RA, and for patients with limited functional goals, poor rehabilitative capacity, or poor compliance. In contrast, interpositional arthroplasty (see later discussion) is recommended for patients with good bone stock, an intact rotator cuff, and an ability to comply with the postoperative regimen.[94]

Corrective Osteotomy for Dysplasias and Acquired Deformities

Humeral osteotomy has been described for correcting rotational deficits and abnormal shoulder biomechanics associated with malunited fractures and glenohumeral instability. In theory, this procedure abates the early development of degenerative joint disease in these conditions. External derotation osteotomy of the humerus is also effective in relieving pain in selective cases of shoulder rheumatoid arthritis associated with internal rotation contractures.[1]

It has been suggested that posterior glenohumeral instability may, occasionally, result from increased retroversion of the glenoid.[9,26,36] However, there is no documented evidence clearly showing that this leads to the development of degenerative disease of the glenohumeral joint. Radiographic projection-effect error may also errantly demonstrate significant (more than 7 degrees) glenoid retroversion. In some symptomatic individuals with significant retroversion or fixed subluxation and early eccentric glenohumeral arthritis, osteotomy of the scapular neck may be an effective treatment by correcting the abnormal biomechanics of the joint (Fig. 20-2).[37] Complications of these osteotomies include infection, nonunion, and arthritis secondary to glenoid penetration by metal hardware.

Destructive osteoarthritic changes in the glenohumeral joint have rarely been described in patients who have glenoid hypoplasia.[75,77] Although glenoid hypoplasia is very uncommon, progressive degenerative joint disease, which can be quite symptomatic, develops in a small percentage of

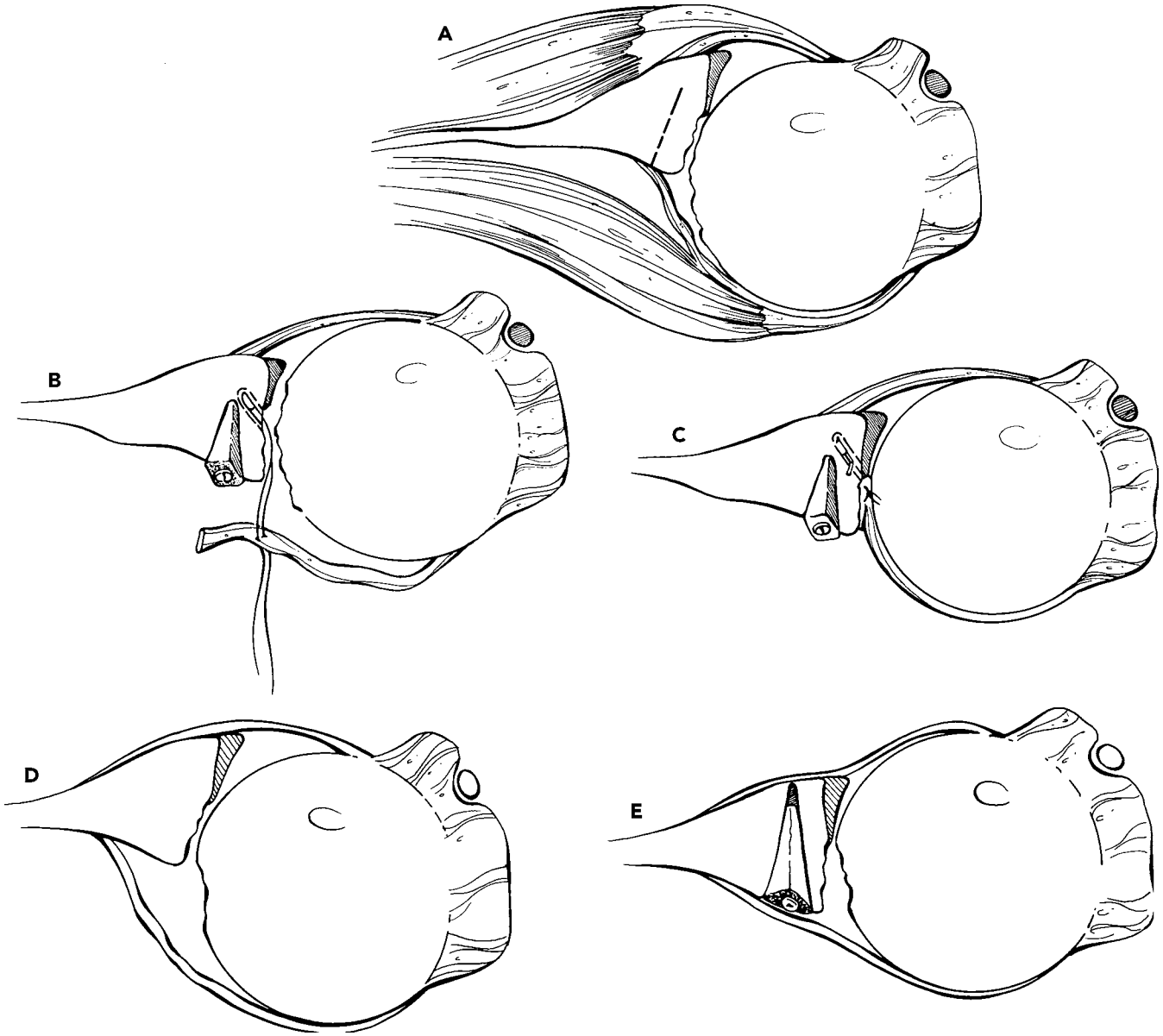


Figure 20-2 Interposition arthroplasty with wedge osteotomy for eccentric glenohumeral wear. **(A)** Posterior subluxation: The *dotted line* demonstrates depth and direction of osteotomy. **(B)** Proper position of iliac crest wedge graft and placement of suture anchor in glenoid. **(C)** Final construct with interposed posterior capsule in area of eccentric wear. **(D)** Symmetrical posterior eccentric wear with posterior subluxation of the humeral head and "relative" redundancy of the posterior capsule. **(E)** The osteotomy is carried out to the anterior cortex, and the anterior periostomy capsule sleeve is kept intact to improve stability of the glenoid fossa. The wedge is opened and bone is grafted with an opening posterior wedge. The capsule is tightened and the head is recentered. (From Hertling D. Autobilization techniques of the extremity. In: Kessler RM, Hertling D, eds. *Management of common musculoskeletal disorders.* Philadelphia: Harper & Row, 1983:178–191, with permission.)

patients.[102] Nearly all patients can be managed with a specific rehabilitation program for the shoulder.[102]

Resection Arthroplasty

The reluctance of most surgeons to undertake shoulder arthroplasty for glenohumeral arthritis in younger high-demand patients resulted in the development of techniques for resecting, débriding, or reshaping the articular surfaces of the glenohumeral joint. Resection of the humeral head has been advocated in the past as a treatment for severe fracture or sepsis, but only occasionally for degenerative or inflammatory arthropathies.[18] After humeral head resection, chondroid tissue forms between the remaining humeral head and the glenoid. The pseudarthrosis formed is important in achieving a good functional result.

Rather than resecting the humeral head, some authors have reduced its diameter.[92] If stability is a concern, then adjustments in the version of the newly fashioned head can also be made. For example, in situations of rotator cuff deficiency, increasing retroversion by some 10 degrees can reduce the chance of dislocation. However, the results of this procedure have not been reported in peer-reviewed publications by these or other surgeons.

Glenoidectomy has also been advocated for treatment of the painful arthritic shoulder.[31,97,108] Gariepy described resecting 7 to 8 mm of the glenoid surface, leaving a new flat surface.[34] More recently, this has been carried out, albeit with more limited bone resection, in conjunction with a humeral hemiarthroplasty and biologic interpositional arthroplasty over the glenoid fossa (Fig. 20-3).[11] Glenoidectomy is probably performed relatively infrequently because of both the limited indications for its use and the greater predictability of prosthetic replacement.

Interposition Arthroplasty and Biologic Glenoid Resurfacing

The use of biologic tissue to form an interpositional arthroplasty has been successful in treating some patients with arthritic joints. In the shoulder this technique was first described in 1918 by Baer, who used a pig's bladder.[4] Tillmann and associates[103,104] described an interpositional arthroplasty using lyophilized dura mater to cover the surfaces of the glenohumeral articulation (Fig. 20-4).

Biologic glenoid resurfacing was developed in 1988 as an alternative to total shoulder arthroplasty in selected younger patients with primary, posttraumatic, or postreconstruction glenohumeral arthritis as a method to improve the results of humeral head replacement. A variety of biologic surfaces have been combined with an uncemented hemiarthroplasty including anterior capsule, autogenous fascia lata, tendo Achilles allograft, and recently human dermal collagen allografts as well as meniscal allografts.

Indications for Interposition Arthroplasty

The main indication for modern techniques of shoulder interpositional arthroplasty is in the rheumatoid patient who has late-stage disease and a well-preserved rotator cuff, and who is capable of complying with postoperative rehabilitation. Contraindications include the presence of large cysts in the humeral head or major defects in the rotator cuff.

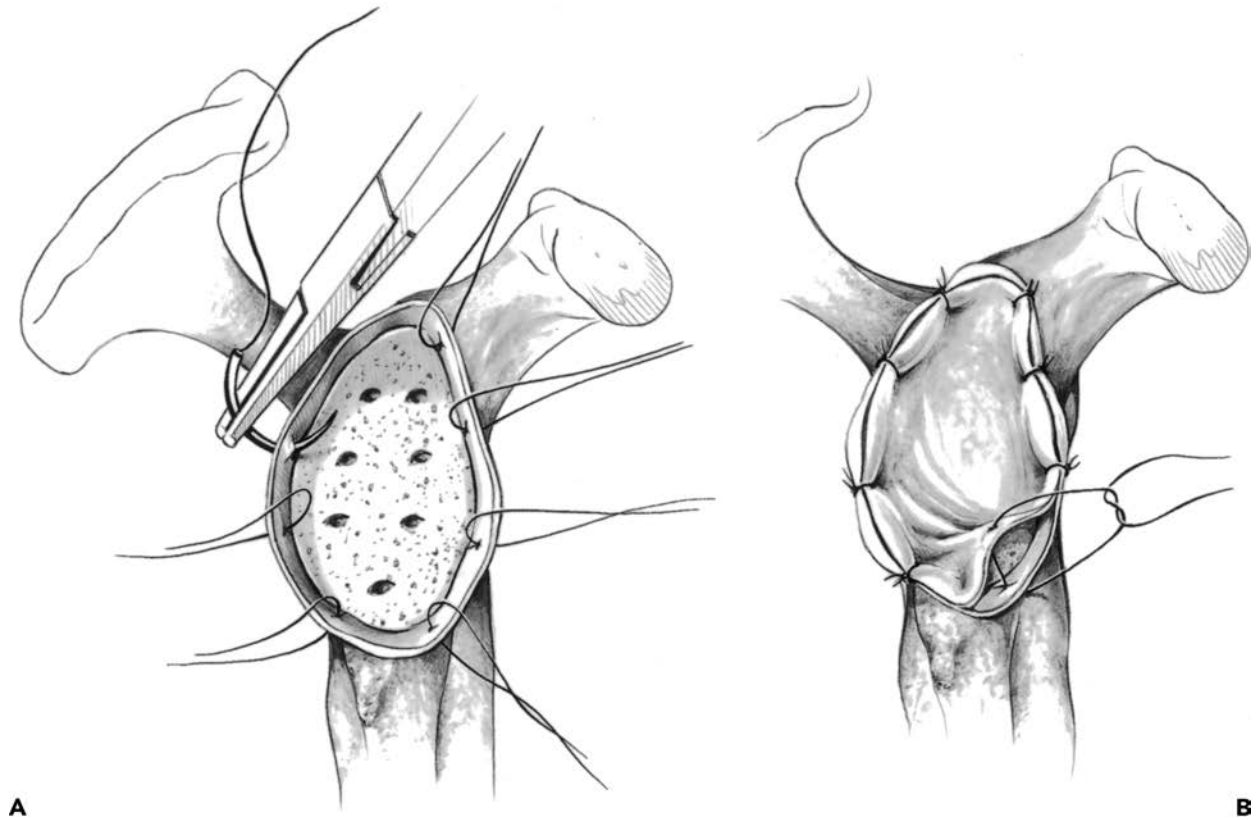


Figure 20-3 **(A)** Preparation of the glenoid, showing marginal suture placement and central drill holes to enhance incorporation of the graft. **(B)** Final glenoid construct demonstrating graft material sutured to the glenoid surface. A suture anchor placed in the central glenoid helps to firmly appose the graft. (From Buchbinder R, Green S, Youd JM. Corticosteroid injections for shoulder pain. *Cochrane Database Syst Rev* 2003;1:CD004016, with permission.)

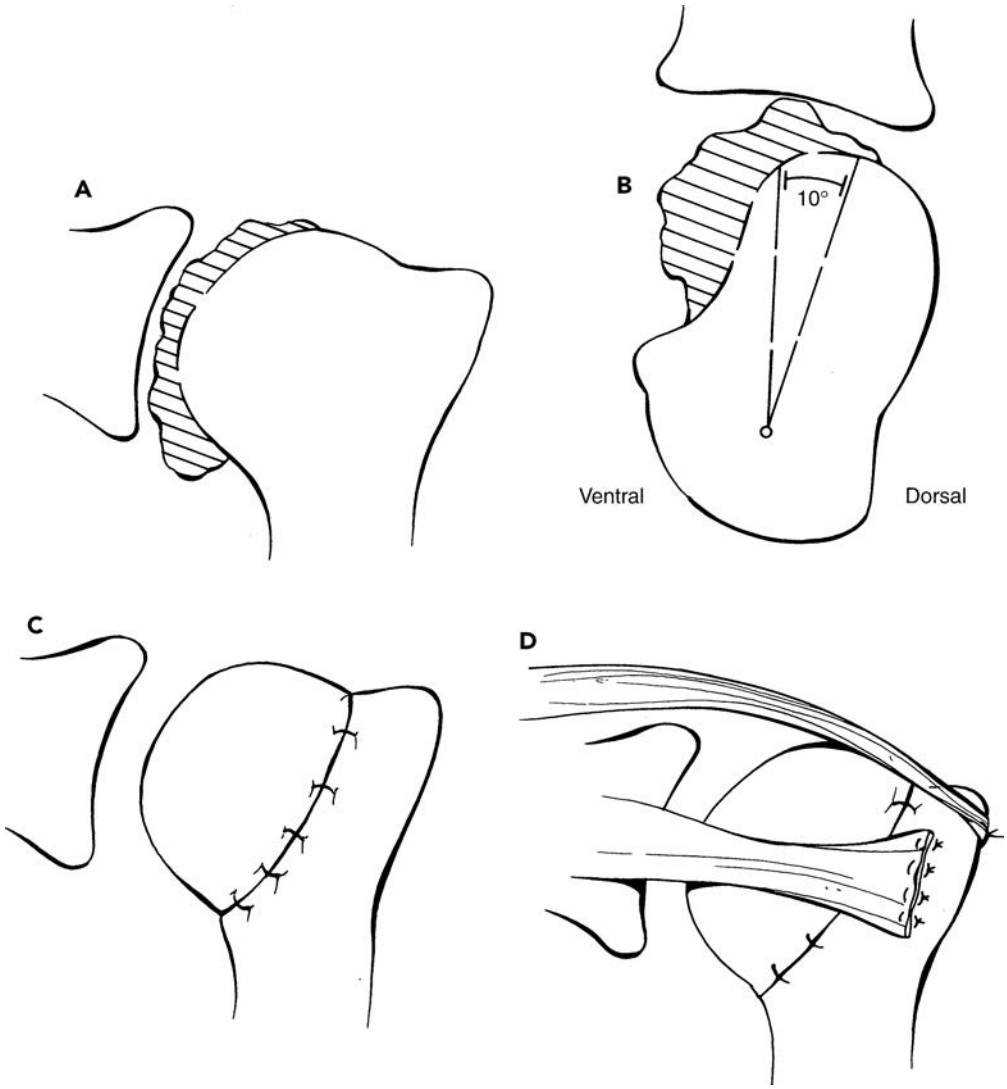


Figure 20-4 Interpositional arthroplasty of the shoulder: **(A)** bony deformity of the humeral head seen in anteroposterior view; **(B)** axillary lateral view; **(C)** newly shaped humeral head covered by lyophilized dura mater; **(D)** refixation of the rotator cuff tendons. (From Samilson RL, Prieto V. Dislocation arthropathy of the shoulder. *J Bone Joint Surg Am* 1983;65:456–460, with permission.)

Indications for Biologic Glenoid Resurfacing

Biologic glenoid resurfacing is indicated in the following patients: young, active patients who may loosen a standard glenoid implant; rheumatoid arthritic patients with irreparable cuffs and/or poor glenoid bone stock; patients with either rotator cuff tear arthropathy or irreparable cuffs and eccentric glenoid wear; and revision arthroplasties with glenoid removal and poor glenoid bone stock.

Graft Choices

Anterior capsule or autogenous fascia lata have been used extensively in the early reports documenting the nonprosthetic biologic resurfacing of the glenohumeral joint. Unpredictable outcomes and concerns regarding the durability of those tissues have led to a predominance of allograft usage for either interposition arthroplasty or biologic resurfacing.

Tendo Achilles allograft remains the more durable, lower cost, and readily available graft choice. The collagen makeup of the Achilles tendon lends itself very well to beneficial wear characteristics on the glenoid surface. Our recent work has demonstrated the significant intermediate-term and longer-term wear characteristics of the tendo Achilles allograft in resisting glenoid erosion and maintaining glenohumeral joint space.

Human dermal collagen allografts have recently been utilized. While these grafts incur a larger expense, the potential gain is a durable surface much like the tendo Achilles graft that is easier both to handle and work with in the surgical setting. The reduced "bulk" of this type of graft

choice, when compared with the tendo Achilles allograft, may promote a more anatomic restoration of glenohumeral anatomy. Nevertheless, the durability of this graft over time is still unknown.

Similarly, recent reports have documented success with the use of a human meniscal allograft as a bearing glenoid surface. The attractive nature of this graft is the noted durability of its natural capacity in the human knee. Unfortunately, this graft choice is more expensive and less readily available, and peer-reviewed reports still document only short-term outcomes with regard to progressive glenoid erosion.

Surgical Technique for Interposition Arthroplasty

Tillman and associates describe a transacromial approach to expose the glenohumeral joint. The anterior two-thirds of the rotator cuff, including the cranial half of the infraspinatus insertion, are dissected off the bone. After a complete synovectomy, the humeral head is reshaped to a smaller radius of curvature that facilitates reconstruction of the usually deficient rotator cuff; retroversion is also increased by 10 degrees to reduce the chance of dislocation. Lyophilized dura mater is then sutured around the circumference of the reshaped head. The rotator cuff is then reattached under appropriate tension.

Milbrink and Wigren[69] have described a modification of the procedure of Tillman in which the glenoid and humeral head of rheumatoid shoulders are covered with a biologic membrane. If necessary, the surface of the glenoid may be smoothed. A sheet of lyophilized dura mater is folded and sutured at the fold to the posterior wall of the glenohumeral joint capsule. The remaining portions of the graft are then sutured, one to the rim of the glenoid and the other over the humeral head.

Surgical Technique for Biologic Glenoid Resurfacing (Authors' Preferred Technique Using Tendo Achilles Allograft Combined with Prosthetic Humeral Hemiarthroplasty)

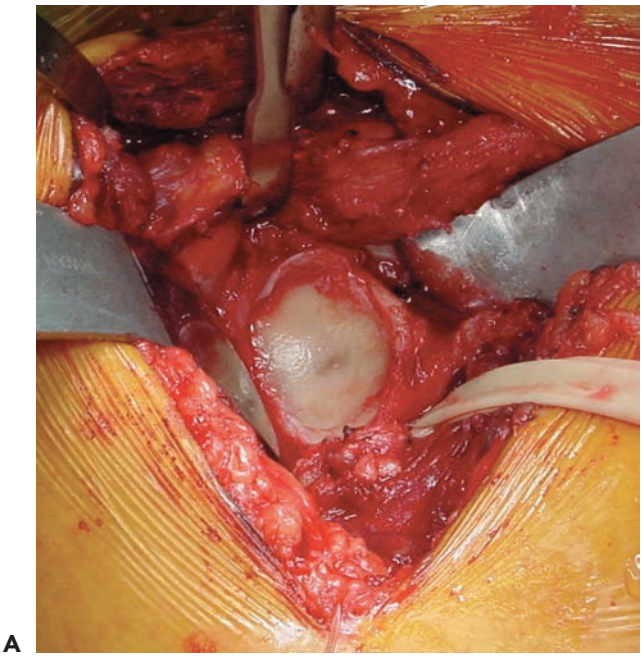
We prefer to expose the glenohumeral joint through a modified 2.5- to 3-in deltopectoral approach, although an anterosuperior transacromial approach may be used. The deltopectoral interval is identified and the cephalic vein is retracted medially with the pectoralis major. We do not "nick" the pectoralis major tendon. The clavipectoral fascia is opened just lateral to the conjoined tendon musculature, and the axillary nerve is identified and protected throughout the entirety of the procedure. The tendon of the long head of the biceps (if still present) is released at the supraglenoid tubercle systematically and will be tenodesed at the end of the procedure.

Once the subscapularis is exposed on its three borders, the anterior humeral circumflex vessels ("three sisters") are ligated. The subscapularis is examined for both integrity and quality and then is released from the lesser tuberosity with a small "flake" of bone. This bone flake aids in reattachment at the end of the procedure with transosseous sutures and also provides a postoperative radiographic marker for indicating whether the subscapularis has healed or has torn. At this point, the anterior and inferior capsular tissue is divided while protecting the axillary nerve. This step is critically important for adequate glenoid exposure.

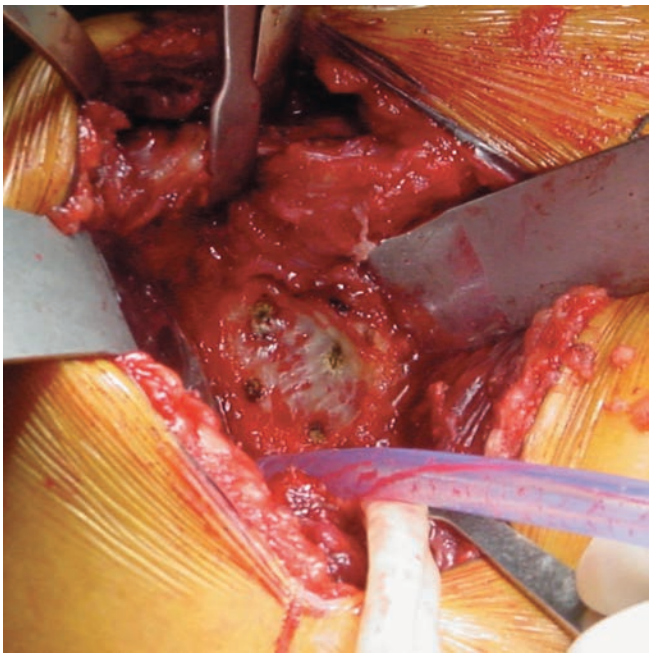
The glenoid is exposed with retractors on its posterior, anterior, and superior rims, and the axillary nerve is protected inferiorly (Fig. 20-5A). Once the glenoid is exposed, we use a standard glenoid reamer to remove the cartilaginous surface and also to "contour" the glenoid to a bleeding subchondral bony surface (Fig. 20-5B). We also attempt to "normalize" the version of the glenoid perpendicular to the scapular spine. This can also be done in a "free hand" technique using a small (3- or 4-mm) hand bur. If the glenoid has previously been replaced and this is a revision operation, the previous component is removed and the glenoid vault is packed with cancellous chips and then reamed or burred to create a relatively smooth bleeding surface. At this point, four bioabsorbable anchors (Mitek Panalok 3.5 Anchors, Mitek USA, a Johnson and Johnson Company, Norwood, MA) with #2 Ethibond suture (Ethicon USA, a Johnson and Johnson Company, Boston, MA) are placed. Four anchors are placed at the "cruciate corners" of the glenoid—the positions on the glenoid that correspond to the positions on a clock face of 12, 3, 6, and 9 o'clock. If desired, a fifth anchor is used in the direct center of the glenoid face. If the glenoid center is deficient (such as after removal of a loose glenoid component), the central anchor is not used. These anchors provide the "anchor" fixation of the biologic resurfacing (Fig. 20-5C).

The tendo Achilles allograft is prepared by removing the calcaneal bone first in a subperiosteal manner with a scalpel (Fig. 20-6). The graft is then folded on itself two or three times ("doubled" or "tripled," depending on the thickness of the Achilles tendon) to form a cushion. We size the superior–inferior dimension of the graft to the native glenoid, and this is easily performed by using a prosthetic glenoid trial as a "sizer." The graft is then "whipstitched" in a mattress fashion with a heavy nonabsorbable suture around the entire periphery of the folded portion (Fig. 20-7)—this performs three important functions:

1. It maintains the graft in a folded position.
2. The mattress peripheral whipstitch "rolls" the edges of the graft into a bumper (similar to a Bankart labral repair that restores the labral bumper).
3. It allows for a central "softer" portion of the folded graft, so that the graft becomes concave in the center and convex on the periphery.



A



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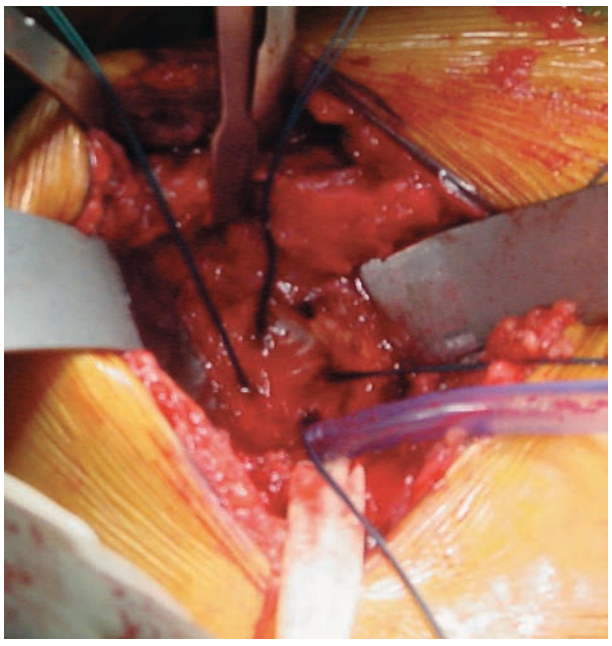

C

Figure 20-5 Biologic resurfacing of the glenoid: **(A)** exposure of the glenoid surface; **(B)** glenoid surface prepared to bleeding bone; **(C)** suture anchors placed for graft fixation.

Using the sutures from the suture anchors in a horizontal mattress fashion, all sutures are brought directly through the graft and the graft is reduced to the bleeding glenoid surface (Fig. 20-8). The sutures from the suture anchors are now tied, producing four points of fixation for the graft to the glenoid. The periphery of the graft is then sutured directly to the glenoid rim using a minimum of four transosseous Bankart type sutures of #2 Ethibond (Ethicon USA, a Johnson and Johnson company, Boston, MA) (Fig. 20-9). (Note that this operative technique is combined with standard humeral hemiarthroplasty using a prosthetic component.)

Postoperative Management

A simple Velpeau sling or shoulder immobilizer is worn for 4 weeks postoperatively. Passive range-of-motion exercises are begun immediately, with the parameters of full anterior elevation and restriction of external rotation based on final postoperative external rotation after subscapularis repair. No active motion is allowed until the sling is removed.

Once the sling is removed, active-motion exercises are begun, allowing for a full range of glenohumeral motion. Resistance exercises are begun at 10 weeks after surgery.

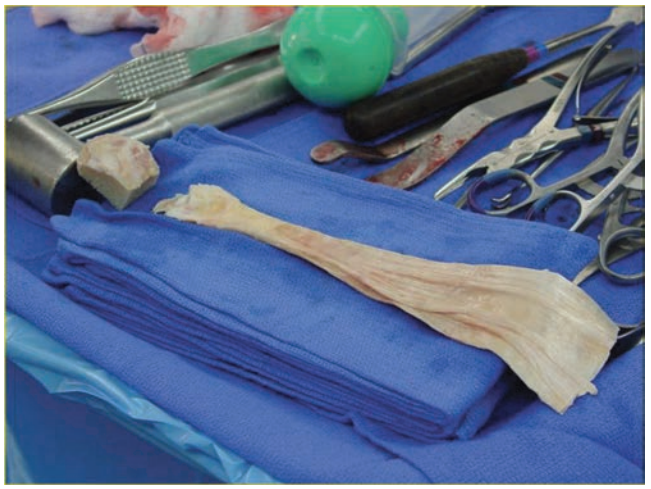

Figure 20-6 Tendo Achilles allograft. Calcaneal bone block is removed from the tendofascial portion of the graft.

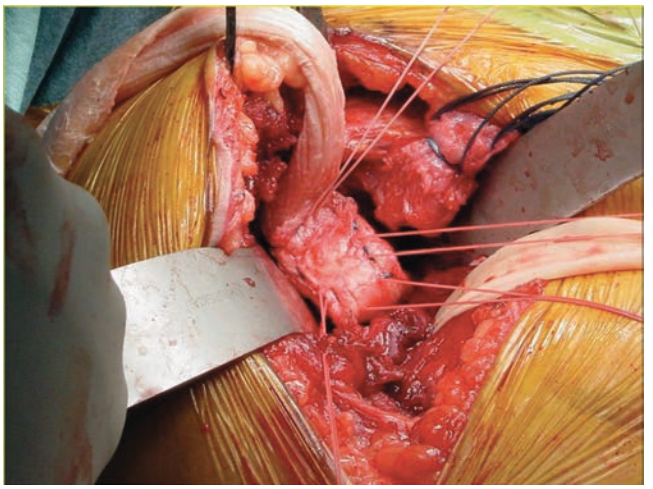

Figure 20-8 Sutures from suture anchors passed through graft prior to final "inset" of the graft.

Results of Interposition Arthroplasty

In Tillman's series,[104] a 4- to 10-year follow-up in 29 patients showed good pain relief and approximately 80-degree increased range of movement in combined flexion and abduction. During the course of follow-up there was no deterioration in either pain relief or range of motion.

Miehlke and Thabe[68] evaluated the results of interpositional arthroplasty performed on 32 shoulders and evaluated at an average of a 20-month follow-up. The diagnosis was rheumatoid arthritis in 29 of the 32 cases. Twenty-seven shoulders had mild or no pain, three had moderate pain, and two had severe pain. Range of motion improved in all cases. In 23 of the 32 cases, a rotator cuff tear was present and subsequently repaired. Complications included three cases of dislocation of the lateral part of the acromion. (The acromion was osteotomized in the surgical approach.) One case was revised, and the other remained asymptomatic and was not treated. The third case had an associated rotator cuff tear in addition to acromial dislocation; ultimately, arthrodesis was performed for biomechanical instability resulting from severe muscular deficit.

In Milbrink and Wigren's series,[69] results reported in 10 patients with late-stage shoulder RA at 6 months to 1 year of follow-up showed very good pain relief and improvement in range of motion. There was also significant improvement in strength by the end of the first postoperative year. However,

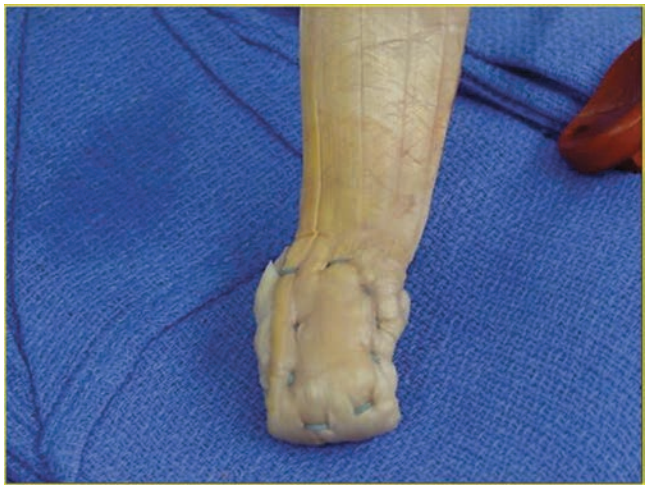

Figure 20-7 Prepared tendo Achilles allograft. Graft is "whip-stitched" around its periphery to form the size of the native glenoid.

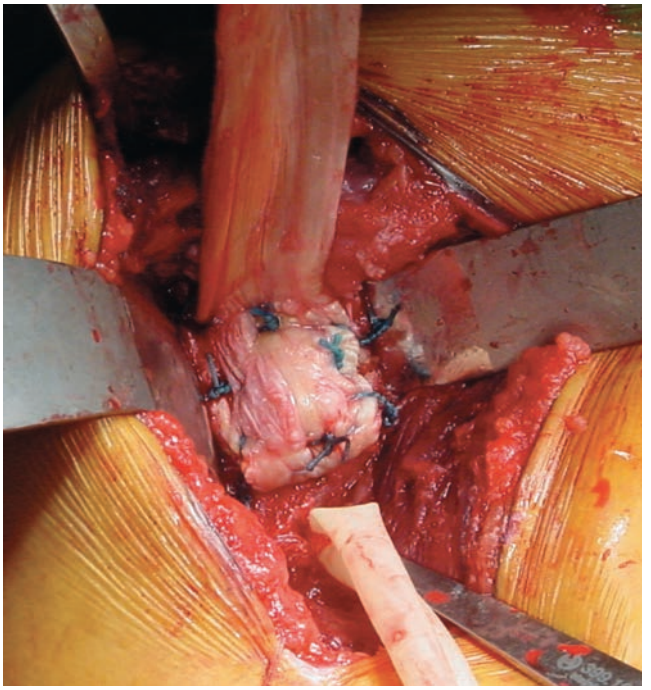

Figure 20-9 Final biologic resurfacing of the glenoid. Graft is secured by mattress sutures from the suture anchors and simple peripheral transosseous sutures.

it is important to emphasize that the follow-up in this study averaged only approximately 6 months.

Results of Biologic Glenoid Resurfacing

In our series, from November 1988 to November 2001, 24 patients (26 shoulders) were followed prospectively. There were 22 males and two females, with an average age of 52 years (range 30 to 75). Fourteen of 26 shoulders had undergone previous surgery. The diagnosis was primary glenohumeral osteoarthritis (9 of 26), posttraumatic arthritis (4 of 26), avascular necrosis (1 of 26), and postreconstructive arthritis (12 of 26). Anterior capsule was used for seven cases, autogenous fascia lata for 12 cases, and tendo Achilles allograft for eight cases. Five- to 13-year results demonstrated 12 of 26 excellent (46%), 9 of 26 satisfactory (35%), and 5 of 26 unsatisfactory results (19%). Glenoid erosion averaged 7.2 mm, stabilizing at approximately 5 years. Joint space averaged 1.3 mm. There were no revisions for humeral loosening. Complications included infection (2 of 26), instability (3 of 26), brachial plexitis (1 of 26), and deep vein thrombosis (1 of 26). Factors associated with unsatisfactory results were use of anterior capsule, infection, and early reinjury. Three of five poor results were revised to total shoulders with conventional polyethylene glenoid implants (two with excellent results and one with poor results). Based on this midterm review, we recommend the use of tendo Achilles allograft as the bearing surface and do not use either anterior capsule or autogenous fascia lata. Other bearing surfaces (human dermal collagen allograft and meniscal allograft) are currently under review.

Arthrodesis

Although advances in the development of shoulder prosthetic arthroplasty have greatly reduced the indications and frequency of shoulder arthrodesis as a primary procedure, arthrodesis remains an excellent salvage procedure in a small percentage of patients with glenohumeral arthritis.[5,78,80,82] Indications for glenohumeral arthrodesis include recurrent or indolent infection, severe soft tissue deficiency including massive rotator cuff tear and coracoacromial deficiencies, poor function of the deltoid, brachial plexus palsy, or persistent symptomatic instability. It is also a viable option in patients operated on multiple times. Patients with both neurogenic pain (e.g., owing to brachial plexus injury) and glenohumeral pain (caused by arthritis) will not have relief of neurogenic pain with a shoulder arthrodesis. However, if function is improved, then the neurogenic pain is generally better tolerated by the patient.[79,80]

In times past, shoulder arthrodesis, coupled with spica immobilization, did not predictably produce solid fusion and was not tolerated well by individuals at risk for surgical complications.[2,15,17,18] However, with the use of internal fixation, autogenic and allogenic bone graft material, and aggressive medical management, glenohumeral fusion is more predictable in these patients.[2] The procedure is contraindicated for patients who cannot cooperate with the program of rehabilitation.[80] Sufficient motion of the scapulothoracic muscles and strength of the trapezius and serratus anterior muscles are important for good function of the arthrodesed shoulder.[58,79]

In a review of a series of 71 shoulder arthrodeses performed for a variety of conditions, Cofield and Briggs[17] reported a pseudarthrosis rate of 4%. Other potential complications include reflex sympathetic dystrophy, acromioclavicular joint arthritis, infection, and failure of the internal fixation. In a review of a series of 41 arthrodeses carried out solely for rheumatoid arthritis, Rybka et al.[92] reported that 90% of shoulders had solid bony fusion at an average of 6 years of follow-up (range 6 months to 20 years). The remaining shoulders had not yet achieved fusion or had fibrous ankylosis. The range of scapulothoracic movement improved by an average of 60% (includes active abduction plus sagittal flexion). Results were rated as excellent or good in 68%, and fair in the remaining 32%.

Current techniques for shoulder arthrodesis are described elsewhere in this textbook.

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Glenohumeral Arthrodesis

21

Robin R. Richards

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OPTIONS

The advent of total shoulder arthroplasty and the refinement of other reconstructive procedures have narrowed the indications for glenohumeral arthrodesis.[39] Nevertheless, arthrodesis of the glenohumeral joint continues to provide a valuable method of shoulder reconstruction for specific indications.[15,22,61] Although the procedure is infrequently performed, it reliably provides patients with a stable, strong shoulder. Albert first attempted glenohumeral arthrodesis in 1881. Since then a voluminous literature has evolved outlining different indications for the procedure and a variety of surgical techniques for performing glenohumeral arthrodesis. Controversies have developed in the literature about the indications for the procedure and the optimum position for glenohumeral arthrodesis.[1,52] More recently, discussion has arisen of the functional results that can be achieved with the procedure.[11] In this chapter I will address the complex and revision problems and then discuss the complications and the results of glenohumeral arthrodesis.

Arthrodesis is an important method of shoulder reconstruction. The procedure has stood the test of time and continues to deserve a place in the shoulder surgeon's armamentarium. For certain specific indications it provides the best method of restoring function to the shoulder.

INDICATIONS

Glenohumeral arthrodesis can effectively restore shoulder function to highly selected patients. Successful glenohumeral arthrodesis reliably results in a strong and stable

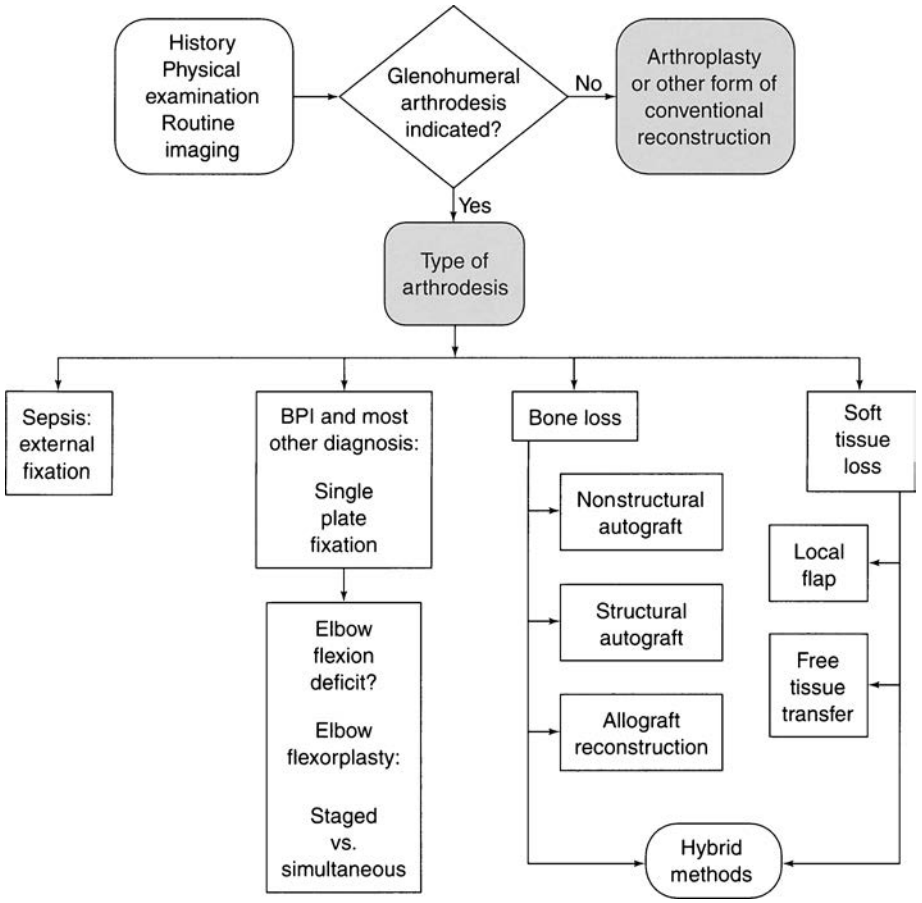

Figure 21-1 Algorithm for evaluation and management of patients presenting to be considered for glenohumeral arthrodesis. BPI, brachial plexus injury.

shoulder. However, the procedure sacrifices all rotation through the glenohumeral joint. Wherever possible, shoulder arthroplasty is preferable to glenohumeral arthrodesis, if for the particular patient there is a choice between the two procedures (Fig. 21-1). Shoulders can be fused if an arthroplasty fails, although fusion in this situation is a technical challenge. The indications for surgery in the author's personal series are illustrated in Fig. 21-2.

Paralysis

All authors agree that the presence of a flail shoulder is an indication for glenohumeral arthrodesis. Patients with anterior poliomyelitis, severe proximal root, and irreparable upper trunk brachial plexus lesions and some patients with isolated axillary nerve paralysis are candidates for glenohumeral arthrodesis.[49,51] These patients have good function in their elbows and hands, but are unable to optimize their upper extremity function because of their inability to place their hand in space. If such a patient has good function in the periscapular musculature, particularly the trapezius, levator scapula, and serratus anterior, glenohumeral arthrodesis stabilizes the extremity and allows effective hand function.[46] Such patients can then fully utilize their upper extremity potential and can work effectively at bench level. In addition, many patients with flail shoulders develop inferior subluxation of the glenohumeral joint owing to periarticular paralysis (Fig. 21-3). This condition is uncomfortable and, in some patients, frankly painful. Such patients often find that they must keep their arm in a sling to avoid injuring it. Painful inferior subluxation of the shoulder provides another indication for stabilization of the glenohumeral joint.

Patients who have the combination of a flail shoulder and flail elbow need both shoulder and elbow reconstruction. In this situation, glenohumeral arthrodesis combined with elbow flexorplasty improves the result of the elbow flexorplasty. Without shoulder stabilization elbow flexion tends to drive the humerus posteriorly, resulting in shoulder extension, rather than elbow flexion. Arthrodesis of the shoulder in some flexion and abduction helps prevent this phenomenon and optimizes the result that can be achieved with the elbow flexorplasty. Patients with flail shoulders often have a tendency to internally rotate their upper extremity to their chest when some function remains in the powerful internal rotators of the shoulder (pectoralis major, latissimus dorsi) and no function remains in the external rotators. Shoulder stabilization either in the form of arthrodesis or L'Episcopo tendon transfer reduces this undesirable tendency.[46]

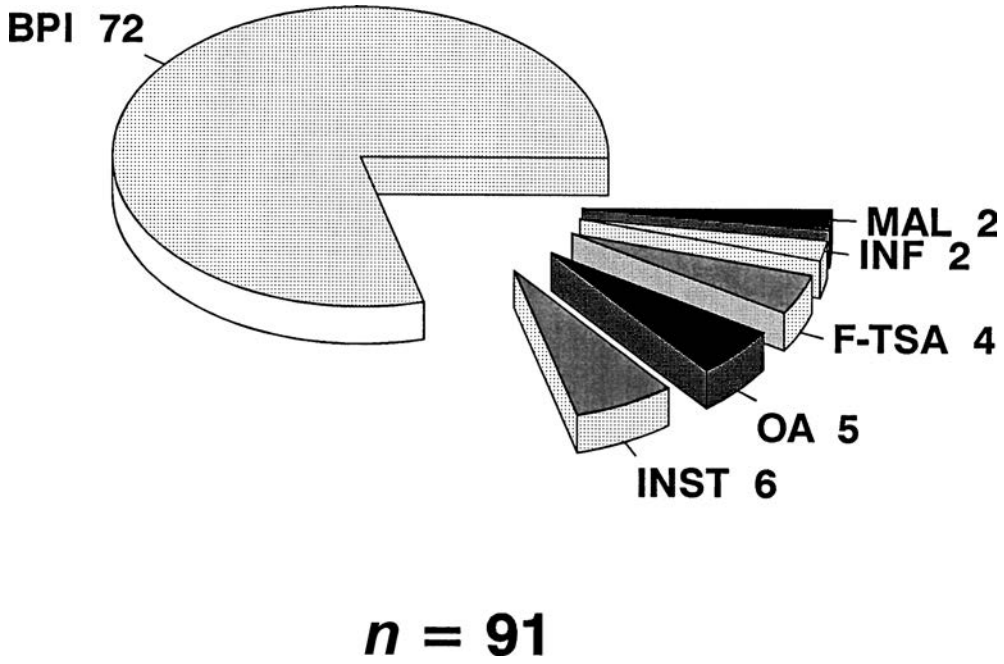

Figure 21-2 Indications for glenohumeral arthrodesis in the author's personal series. BPI, brachial plexus injury; F-TSA, failed total shoulder arthroplasty; INF, infection; INST, instability; MAL, malunion; OA, osteoarthritis.

There is a wide degree of variability in the disability caused by axillary nerve paralysis, and I have seen many patients who have virtually full motion following paralysis of the axillary nerve, providing the rotator cuff musculature is undisturbed (endurance is never normal). Patients with isolated paralysis of the axillary nerve can be treated by either muscle or tendon transfer or glenohumeral arthrodesis. Numerous reports exist in the literature on the value of muscle and muscle tendon transfer to restore shoulder function following paralysis of the axillary nerve.[20] It is generally agreed that multiple transfers are necessary to restore deltoid function and that significant problems can occur with gliding of transfers over the acromion. It is often necessary to harvest autogenous tissue, such as fascia lata, to prolong the transfers, and the process of rehabilitation is challenging following such procedures. In my experience such transfers are indicated primarily for pediatric patients and adult patients who have only partial paralysis of the axillary nerve.[20] However, pediatric patients do well with glenohumeral arthrodesis, and some authors feel they are better able to adapt to the procedure.[31] If total paralysis of the axillary nerve is present and significant limitation of shoulder function ensues, I would recommend glenohumeral arthrodesis, recognizing that the alternative of muscle transfers may be available in carefully selected patients. Glenohumeral arthrodesis is useful in such patients, provided their symptoms justify the procedure.

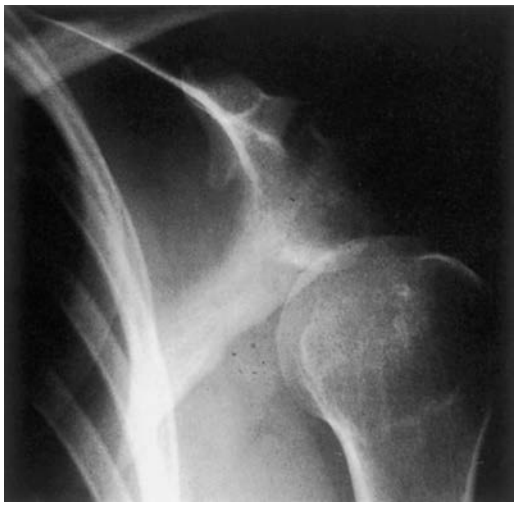

Figure 21-3 Severe inferior subluxation of the glenohumeral joint in a patient with brachial plexus palsy. The patient is severely disabled, in spite of good hand and wrist function, because he is unable to position the hand in space.

Reconstruction Following Tumor Resection

En bloc resection of periarticular malignant tumors often requires sacrifice of the rotator cuff or the deltoid, or both. If the resection requires sacrifice of these tissues, reconstruction of the shoulder with an arthroplasty is inadvisable owing to the high risk of instability if an unconstrained prosthesis is used and to the certainty of loosening if a constrained prosthesis is used. Glenohumeral arthrodesis is

the procedure of choice to reconstruct the shoulder following wide resection of periarticular malignancies. Specific techniques have been recommended for glenohumeral arthrodesis following tumor resection.[30] These include the use of specialized fixation devices and bone-grafting techniques. Vascularized bone grafts and massive allografts are sometimes necessary because of the large defects created by tumor resection.[28] These techniques will be discussed later.

Shoulder Joint Destruction by Infection

Destruction of the shoulder joint by septic arthritis remains an indication for glenohumeral arthrodesis. In the past, tuberculous arthritis was a common indication for glenohumeral arthrodesis. Worldwide, this condition remains prevalent, although in the Western world it has become extremely uncommon. Septic arthritis continues to occur, and when it does, the shoulder joint can be destroyed with resultant pain and limitation of function. Most surgeons would agree that in a young patient with shoulder dysfunction, for this reason, arthrodesis of the shoulder, as opposed to total shoulder arthroplasty, would be indicated. Although total shoulder arthroplasty can be performed in patients with a remote history of sepsis, if there is a recent history of sepsis or if the patient is young, arthrodesis provides a more satisfactory alternative.

Failed Total Shoulder Arthroplasty

I have seen several patients who have had multiple unsuccessful shoulder arthroplasties. Although the results of shoulder arthroplasty are generally good, there are some patients in whom loosening, sepsis, and implant breakage occur. These patients often have severe loss of humeral and glenoid bone stock (Fig. 21-4). In this situation the surgeon must choose between attempting a repeat revision of the shoulder arthroplasty and reconstruction of the shoulder joint by arthrodesis. The results of revision total shoulder arthroplasty are suboptimal when compared with the results of primary arthroplasty for other reasons. The decision between these two alternatives must be made on the basis of the patient's age, the presence or absence of active sepsis, the bone stock that remains in the proximal humerus and glenoid, the symptoms the patient is experiencing, the quality and function of the rotator cuff and the deltoid, and the technical experience and expertise of the surgeon. In several patients who were significantly disabled I have carried out glenohumeral arthrodesis following failed total shoulder arthroplasty and found it to be a helpful procedure for the patient. It is my belief that glenohumeral arthrodesis must be considered when the reconstructive surgeon is confronted with a patient with a history of multiple failed total shoulder arthroplasties.

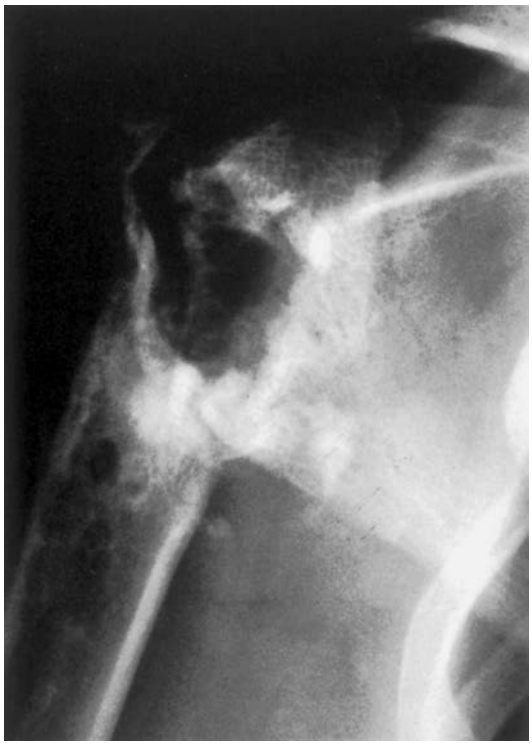

Figure 21-4 Excision arthroplasty of the glenohumeral joint after several failed total shoulder arthroplasties. The patient is disabled by severe unremitting pain with any activity. Glenohumeral arthrodesis is indicated to provide the patient with a stable, relatively painless shoulder.

Shoulder Instability

Virtually all patients with shoulder instability can be treated by soft tissue or bony reconstructive procedures to stabilize the glenohumeral joint. Rarely, a patient will present with chronic shoulder instability after multiple attempts at surgical stabilization. If every surgical therapeutic alternative has been exhausted, the patient's shoulder remains symptomatically unstable, and the patient does not wish to wear a thoracobrachial support, arthrodesis can be indicated to restore shoulder stability. In this situation careful assessment of the patient's psychologic makeup must be carried out and one must be certain the patient has a full understanding of the implications of the procedure. Much has been written about the difficulties in managing such patients and further elaboration here is not necessary.[47,53]

Rotator Cuff Tear

Severe shoulder dysfunction can result from massive rotator cuff tears.[17] Most rotator cuff tears can be managed by coracoacromial decompression and repair of the cuff. In patients who have massive rotator cuff tears that cannot be repaired, some authors have reported good results with débridement of the cuff tear and coracoacromial decompression.[50]

Long-standing rotator cuff tears can lead to cuff tear arthropathy as reported by Neer et al.[40] This difficult pathologic entity can be treated by prosthetic arthroplasty with limited goals. Glenohumeral arthrodesis should be kept in mind as a possible alternative form of reconstruction in such patients when the technical skill of the surgeon or the available tissues do not permit cuff reconstruction and the patient is sufficiently symptomatic to exchange the loss of glenohumeral motion for the relief of pain. In my experience, this situation rarely, if ever, develops.

Malunion

Glenohumeral arthrodesis is rarely indicated for posttraumatic deformity. Most patients with posttraumatic deformities, such as osteonecrosis of the humeral head, chronic fracture dislocations, tuberosity impingement, or a malunion of the proximal humerus, are best treated by shoulder reconstruction using either osteotomy, arthroplasty, or a combination of both. If these procedures are not possible, then glenohumeral arthrodesis can be considered.

Osteoarthritis

The presence of glenohumeral osteoarthritis in an otherwise normal shoulder is an indication for total shoulder arthroplasty. However, almost all patients with glenohumeral osteoarthritis are of sufficient age that they are excellent candidates for total shoulder arthroplasty. If the patient were to develop osteoarthritis at a relatively young age, then glenohumeral arthrodesis might be considered,[2] particularly if the patient was a laborer and modified work was not available to him or her. The results of total shoulder arthroplasty are so much superior that arthrodesis is rarely indicated for patients with this diagnosis.

Rheumatoid Arthritis

Patients with rheumatoid arthritis affecting the glenohumeral joint commonly have multiple problems in their upper extremity. Glenohumeral arthrodesis is recognized as a procedure that can decrease pain arising in the glenohumeral joint.[24,43,56] Obliteration of glenohumeral motion has a negative influence on the upper extremity function. Frequently both upper extremities are involved. It is my belief that patients with rheumatoid arthritis are much more effectively treated by total shoulder arthroplasty.[18] Favorable reports of the combination of shoulder and elbow arthroplasty have appeared in the literature, and I would advocate this method of reconstruction for such patients.

CONTRAINDICATIONS TO GLENOHUMERAL ARTHRODESIS

Glenohumeral arthrodesis should not be performed if an alternative method of shoulder reconstruction is available. Many patients are amenable to arthroplastic reconstruction that preserves glenohumeral motion and has greater potential to restore function. Glenohumeral arthrodesis places a significant functional demand on the patient, requiring a major effort on the part of the patient to rehabilitate the shoulder following surgery and to strengthen the thoracoscapular musculature. The procedure is contraindicated in a patient who cannot cooperate with such a program of rehabilitation. Similarly, the procedure is contraindicated in any patient with a progressive neurologic disorder who may experience paralysis or weakness of the trapezius, levator scapula, or serratus anterior. Glenohumeral arthrodesis relies on these muscles to motor the extremity, and significant weakness will grossly impair shoulder function following the procedure.

RESULTS

Review of Literature

Many techniques for glenohumeral arthrodesis have been reported. Some authors have used extraarticular arthrodesis, others have reported on methods of intraarticular arthrodesis, and still others have combined the two methods. In reviewing the literature it is apparent that internal fixation has been employed more frequently in recent years. Historically most authors have recommended external immobilization, although recently reports of glenohumeral arthrodesis without external immobilization have appeared.[27,49] I will now discuss extraarticular arthrodesis, intraarticular arthrodesis, the use of internal fixation, and the use of external fixators.

Extraarticular Arthrodesis

Extraarticular arthrodesis is primarily a historical procedure used before the antibiotic era to treat tuberculous arthritis. This treatment method was used to avoid entering the tuberculous joint and to obliterate motion at the joint without activating and spreading the infection. Watson-Jones[60] described a technique utilizing a Cubbin's approach[12] to the shoulder, decorticating the superior and inferior surfaces of the acromion. A bone flap was then cut into the greater tuberosity and both the clavicle and the acromion were osteotomized. The arm was abducted and the acromion positioned to lie between the two edges of the bone flap in the proximal humerus. A spica cast was applied for 4 months.

Putti[42] described a technique whereby the spine of the scapula and the acromion were exposed subperiosteally. The spine of the scapula was detached, the acromion split, and the medial and lateral portions and the upper end of the humerus exposed. The lateral surface of the humerus was split similar to the method described by Watson-Jones and the spine of the scapula driven down into the humerus with the arm abducted. Spica cast immobilization was necessary following this procedure. Neither Watson-Jones' nor Putti's technique was truly extraarticular because the shoulder joint was usually entered when creating the split in the proximal humerus.

Brittain[6] described a true extraarticular arthrodesis. This arthrodesis used a large tibial graft that was placed between the medial humerus and the axillary border of the scapula. The graft was maintained in position by its "arrow" shape (the pointed end was inserted into the humerus and the opposite, notched, end into the axillary border of the scapula). The graft was stabilized by its shape and adduction of the arm, which produced a compressive force along the long axis of the graft. DePalma[14] reported that the failure rate of the arthrodesis was high owing to fracture of the long tibial graft.

Intraarticular Arthrodesis

Gill[19] combined intraarticular and extraarticular arthrodesis. Gill used a U-shaped incision centered 2 cm below the acromion combined with a downward limb to the incision. Gill denuded the superior and inferior surface of the acromion and excised the rotator cuff. The glenoid fossa was decorticated as was the cartilaginous surface of the humeral head. An osseous flap was elevated from the anterolateral surface of the humerus, and a wedge-shaped slice of bone, with its base superiorly, was removed from the humerus. The arm was then abducted and impacted onto the acromion. The position was maintained by suture of the capsule and rotator cuff to the periosteum on the superior surface of the acromion. This technique is predicated on the assumption that it is desirable to fuse the glenohumeral joint in a large amount of abduction. This can be desirable in children when internal fixation is not used because, with time, the amount of abduction decreases. The technique is undesirable in adults owing to the likelihood of excessive abduction being retained following arthrodesis.

Makin[32] has reported on a method of glenohumeral arthrodesis in children that preserves the growth potential of the proximal humeral epiphysis. Makin fused the shoulder in 80 to 90 degrees of abduction, fixing the humerus to the glenoid with Steinman pins inserted first into the humerus in a proximal–distal direction and then driven in the reverse direction into the glenoid. Makin followed his children to adult life and noted that there was only a small loss in humeral length and no change in position of the fused shoulder. He recommended this technique, stating that this amount of abduction was necessary to maintain the growth potential of the proximal humeral epiphysis. I have had no experience with this technique. If this amount of abduction was maintained, the shoulder would be dysfunctional in adulthood.

Moseley[36] reported division of the rotator cuff insertion and excision of the intraarticular portion of the biceps tendon. Moseley advocated suture of the biceps tendon into the bicipital groove after division of its origin. This is an important step to remember in those patients who have functioning biceps, to avoid the unsightly cosmetic deformity identical with that seen in rupture of the long head of the biceps tendon. The author performs a biceps tenodesis during glenohumeral arthrodesis in all patients who have a functional biceps. Moseley denuded the inferior surface of the acromion as well as the articular cartilage of the humeral head and glenoid fossa. This is an important step in performing glenohumeral arthrodesis because the humeral head presents such a small area to the glenoid across which fusion can occur.

Beltran et al.[4] performed glenohumeral arthrodesis through an anterior approach. They osteotomized the coracoid and created a tunnel that crossed the humerus and entered the glenoid cavity. They utilized a screw for internal fixation and in addition used a Cloward reamer to position a fibular graft from the proximal humerus into the infraglenoid area. Other techniques for glenohumeral arthrodesis have been described by May[33] and Davis and Cottrell.[13]

Internal Fixation

A variety of methods of internal fixation have been advocated for glenohumeral arthrodesis. It is generally agreed that internal fixation is desirable because it maintains the position of the arthrodesis and can decrease the length of time that plaster immobilization is necessary to obtain an arthrodesis. Makin advocated the use of Steinman pins in children who are undergoing glenohumeral arthrodesis at an early age. Carroll[6] reported on the use of a wire loop to maintain the position of glenohumeral arthrodesis. Carroll advocated the use of 22-gauge wire passed through the head of the humerus and the anterosuperior lip of the glenoid. He employed this method of arthrodesis in 15 patients, and all patients achieved solid bony union between the third and fourth month following surgery. Carroll noted that it was possible to manipulate the shoulder following surgery and change the position of the arthrodesis. As time has gone by, most authors have advocated more rigid forms of internal fixation. At present, few surgeons would use a wire loop as a method of internal fixation when performing glenohumeral arthrodesis.

Other authors have reported the use of screws to obtain fixation during glenohumeral arthrodesis. May[33] used a single stabilizing wood screw crossing the humerus and

entering the glenoid fossa. Davis and Cottrell[13] used a similar technique and added a muscle pedicle bone graft that was fixed in place with wood screws. Cofield and Briggs[11] and Leffert[29] also reported on the use of compression screw fixation without the use of a plate. Beltran[4] developed a special fixation device using a screw bolt and washer to obtain glenohumeral arthrodesis. In addition, Beltran used an acromiohumeral screw and a fibular graft as methods of internal fixation.

The Association for Osteosynthesis (AO) group first advocated the use of plate fixation in 1970. They described this method of arthrodesis as not requiring supplementary plaster immobilization. The AO group advocated the use of two plates for internal fixation.[37] The first plate was applied along the spine of the scapula and then bent down over the humerus, maintaining a position of 70 degrees of abduction between the vertebral border of the scapula and the humerus. The object of this position was to obtain a clinical position of 50 degrees of abduction, 40 degrees of internal rotation, and 25 degrees of flexion. They anchored this plate to the scapula with a long screw placed down through the plate and the acromion and into the neck of the glenoid (Fig. 21-5). They also noted that fixation could be improved by the insertion of two long screws inserted through the plate and the humeral head and into the glenoid. "If necessary," a second plate applied posteriorly was advocated to improve the internal fixation. I have rarely found it necessary to use two plates when performing glenohumeral arthrodesis.

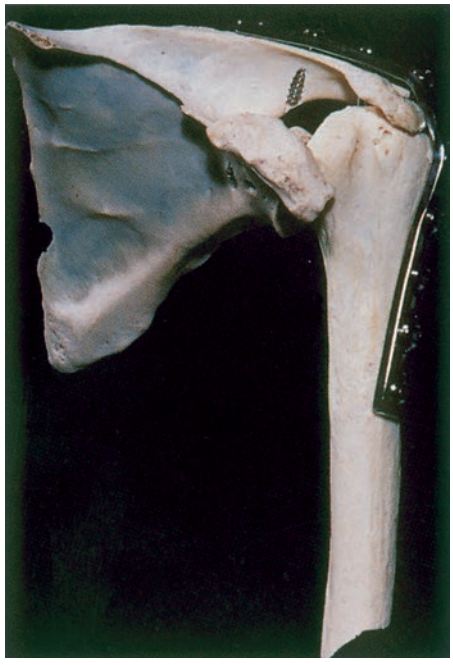

Figure 21-5 Appearance of the humerus and scapula after glenohumeral arthrodesis with a single plate. Screws have been passed from the spine of the scapula through the plate into the area of the coracoid base. Excellent fixation in the scapula can be obtained with this technique.

Kostuik and Schatzker[27] have reported on the use of the ASIF technique. They did not use external immobilization postoperatively and reported good results in their patients. Riggins[49] reported on shoulder fusion without external immobilization in 1976. Both the AO group and Riggins supplemented their arthrodeses with bone grafts. Riggins treated four patients with the use of a plate for internal fixation. Two of the patients had above-elbow amputations. The arthrodesis was successful in each case. Miller et al. analyzed five fixation techniques for shoulder arthrodesis biomechanically and found double plate fixation to provide the highest bending and torsional stiffness in comparison to single plate fixation, external fixation with screws, external fixation alone, and screws alone (techniques listed in decreasing level of stiffness). Ruhmann et al.[55] found the strength of three humeroglenoid screws and three acromiohumeral screws to be similar to plate arthrodesis.

My colleagues and I have reported on the results of a modified method of glenohumeral arthrodesis using internal fixation in 14 adult patients with brachial plexus palsy.[44] We first used a single 4.5 AO/ASIF dynamic compression (DC) plate applied over the spine of the scapula onto the shaft of the humerus. We advocate placement of two cancellous compression screws passing through the plate and the proximal humerus into the glenoid first to achieve compression at the glenohumeral arthrodesis site. The plate is anchored to the scapula with a long screw passing through the spine of the scapula into the area of the coracoid base. Anchorage of the plate by this method, as opposed to the AO method, which inserts the screw into the glenoid neck, provides good fixation, yet leaves room for the large compression screws in the glenoid, which are felt to be more important in obtaining arthrodesis. Initially, we advocated the use of a postoperative spica cast because adult patients with brachial plexus injuries generally have significant osteoporosis, poor muscular control, and decreased proprioception, resulting from their neurologic injury. Bone grafts were not used in this series, and no nonunions occurred. More recently, we have begun to use thermoplastic thoracobrachial orthoses when performing glenohumeral arthrodesis and no longer use a spica cast (see following discussion).

We reported a modification of the technique described in 1985.[45] The current technique uses a malleable plate for internal fixation. In the modified procedure a single 10-hole 4.5 AO pelvic reconstruction plate is used for internal fixation. This plate, although weaker than the 4.5 DC plate, is much easier to contour in the operating room and is much less prominent as it passes over the acromion onto the shaft of the humerus. None of the 11 patients whose shoulders were fused by this method complained of plate prominence. Fusion was obtained in each instance without failure of the internal fixation device. External cast immobilization was used for 6 weeks postoperatively. Plate

prominence can also be decreased postoperatively by notching the acromion laterally where the plate crosses this structure.

External Fixation

Charnley and Houston[8] reported a method of compression arthrodesis of the shoulder utilizing two Steinman pins. The first Steinman pin was inserted posterosuperiorly into the base of the acromion and then into the main mass of the scapula just proximal to the glenoid. The second pin was inserted posterolaterally in relation to the shaft of the humerus and perpendicular in relation to the axis of the humerus to transfix the region of the surgical neck. A compression apparatus was then applied to the two pins. After application of the compression apparatus, a plaster spica cast was applied and worn for an average of 4.8 weeks. After removal of the pins and compression clamps, a second plaster cast was applied for an average of 5.3 weeks. Other methods of external fixation have been reported.[23,26,38,57]

Currently, the indications for glenohumeral arthrodesis using external fixation are limited. I have used this method occasionally in patients with active septic arthritis of the shoulder and in patients with massive trauma resulting in bone and soft tissue loss. I have utilized pins placed through the clavicle and acromion and a second set of pins inserted in a separate plane into the spine of the scapula and neck of the glenoid. Two half-frames are then constructed to stabilize the shoulder. The two half-frames can be cross-connected for increased stability. This technique is desirable if there is an open, infected wound draining from the shoulder joint. This method allows dressing changes and care of the soft tissues without the increased dissection and soft tissue disruption necessary to place an internal fixation device. If the soft tissue envelope improves, internal fixation can be performed later.

AUTHOR'S PREFERRED TREATMENT

Position of Arthrodesis

A wide variety of different positions for glenohumeral arthrodesis have been advocated in the literature. Perusal of the literature reveals that no two authors agree on exactly the same optimum position for glenohumeral arthrodesis. There was sufficient controversy in the literature that the American Orthopaedic Association established a committee to determine, among other things, the optimum position for glenohumeral arthrodesis. This committee reported in 1942[1] and concluded that the optimum position for glenohumeral arthrodesis was 45 to 50 degrees of abduction (measured from the vertebral border of the scapula), forward flexion from the plane of the scapula 15 to 25 degrees, and 25 to 30 degrees of internal rotation. This report caused a great deal of controversy in the literature following its publication. Part of the controversy revolved around the method of measurement of abduction. Some authors recommended using the angle formed by the vertebral border of the scapula and the axis of the humerus to determine abduction, whereas others argued that the angle between the arm and the side of the body was more appropriately measured (clinical abduction).

Rowe noted in 1974 that the amount of abduction that had been recommended was excessive for adults.[52] This position had been recommended primarily for patients who were having their shoulders fused as children in whom internal fixation was not used. In this situation, the amount of abduction present at the time of surgery was commonly lost during the period required for arthrodesis to become secure, as well as during continued growth. If the same position were used in adults, excessive scapular winging would occur and the scapula would not comfortably rest at the side. Furthermore, Rowe noted that the measurement of clinical abduction was more practical and recommended this method, rather than measuring abduction from the vertebral border of the scapula. Rowe recommended that the arm be placed nearer the center of gravity of the body, with enough abduction to clear the axilla and sufficient flexion and internal rotation to bring the hand to the midline of the body.

Other authors have recommended a variety of positions for glenohumeral arthrodesis. All authors agree that abduction and forward flexion are desirable. Most have recommended internal rotation. In my opinion, the optimum position for glenohumeral arthrodesis is one that brings the hand to the midline anteriorly so that with elbow flexion the mouth can be reached. The amount of abduction should not be excessive so that the arm can rest comfortably at the side. I recommend a position of 30-degrees abduction (measured clinically), 30-degrees forward flexion, and 30-degrees internal rotation. The so-called 30-30-30 position is easily obtained in the operating room and usually provides patients with the ability to reach their mouth, their front pocket, and their back pocket. It must be recognized that the position cannot be measured exactly at the time of surgery. I have found in my series of shoulder arthrodeses that it is usually possible to arthrodese the shoulder within 10 degrees of the desired position.

Technique

The patient is placed in the semisitting position.[3] The arm is free draped. An incision extends from the spine of the scapula to the anterior acromion and down the anterior aspect of the shaft of the humerus. Uematsu has recommended a posterior approach when performing glenohumeral arthrodesis.[59] Other authors have described arthroscopically assisted glenohumeral arthrodesis.[35] The deltoid muscle is detached from the anterior acromion and

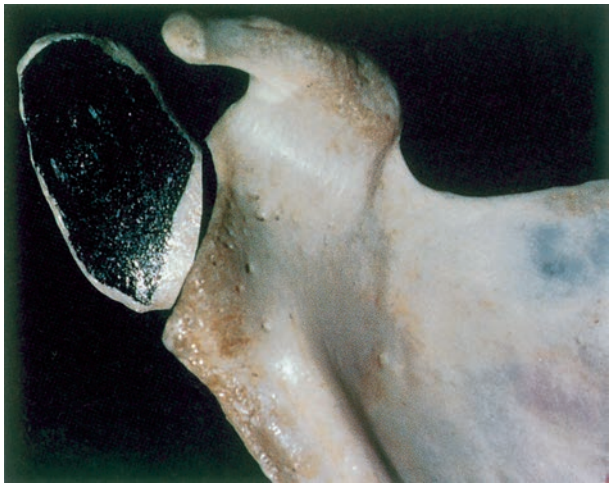

Figure 21-6 The undersurface of the acromion is decorticated (*shaded area*). Decortication of the acromion provides a much larger area across which fusion can occur. When the arm is placed in the "30-30-30" position, the superior aspect of the humerus, when it is brought proximally, abuts against the undersurface of the acromion.

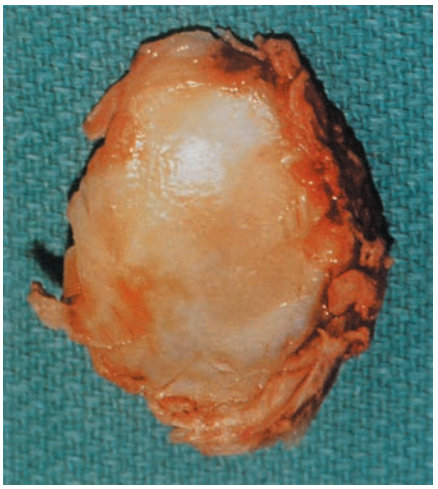

Figure 21-7 The glenoid articular surface, the glenoid labrum, and the underlying subchondral bone are resected. This is most easily accomplished with a curved osteotome.

its fibers are split distally. Because deltoid function is not present in patients with brachial plexus palsy undergoing glenohumeral arthrodesis, denervation of the muscle is not usually a concern. If deltoid function is present, the incision should be curved over to the deltopectoral interval and the shoulder approached in this fashion. By using this approach, deltoid bulk will be maintained and the patient will experience a more satisfactory cosmetic result. Sparing the deltoid may prevent the development of pain from neuromas arising from the axillary nerve postoperatively. The rotator cuff is resected. The undersurface of the acromion and the humeral head are decorticated (Fig. 21-6). An attempt is made to obtain arthrodesis of both the glenohumeral and acromiohumeral articulations because the glenoid fossa offers such a small area for fusion with the humeral head. Decortication of the undersurface of the acromion increases the potential fusion area. The articular surface of the glenoid, the glenoid labrum, and the subchondral bone are resected with a curved osteotome (Fig. 21-7). A 10-hole 4.5-mm pelvic reconstruction plate is used for internal fixation during the procedure (Fig. 21-8).

After resection of the rotator cuff and decortication of the joint surfaces, the shoulder is supported in 30-degrees flexion, 30-degrees abduction, and 30-degrees internal rotation. Abduction is measured from the side of the body. This method of measurement does not accommodate for individual variations in muscle mass or body fat. However, clinical experience has shown it to be accurate within 10 degrees in any plane. The humeral head is brought proximally to appose the decorticated undersurface of the acromion. When the humerus is abducted and flexed 30 degrees, the humeral head apposes both the undersurface of the acromion and the glenoid fossa (Fig. 21-9). The position is maintained by supporting the arm with sterile folded sheets. An assistant is assigned to maintain the position while the plate is contoured. Thirty degrees of internal rotation brings the hand to the midline. I have not found it necessary to measure abduction radiographically.

Handheld bending irons are used to contour the plate along the spine of the scapula, over the acromion, and

Figure 21-8 Specially modified 4.5 pelvic reconstruction plate used for shoulder arthrodesis. The plate is bent as it crosses the acromion and twisted just past the bend. The plate can be easily bent in all planes with the handheld bending irons.

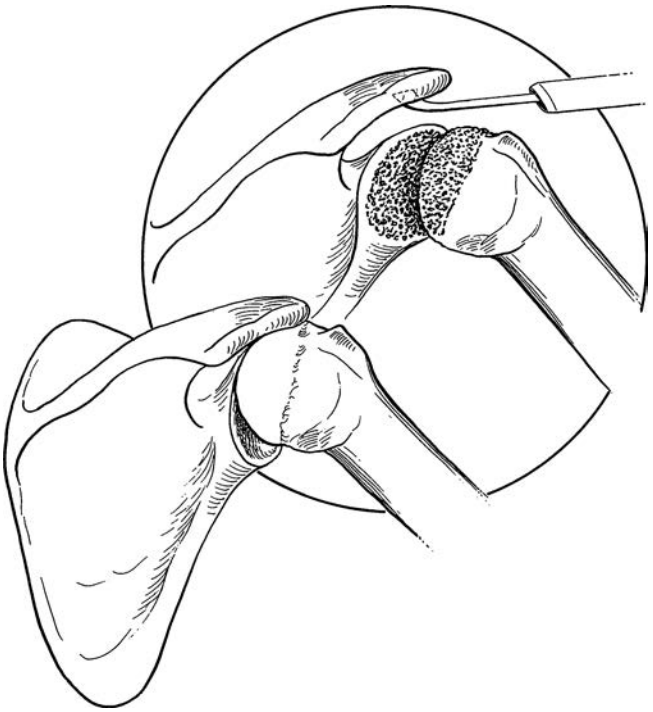

Figure 21-9 The glenoid, humeral head, and undersurface of the acromion are decorticated and shaped so that the humeral head has the greatest surface area contact. This technique increases the likelihood of a solid fusion.

down onto the shaft of the humerus (Figs. 21-10 and 21-11). The malleable nature of the plate allows precise intraoperative contouring of the implant to the specific local anatomy in any given patient. The plate must be bent 60 degrees over the acromion and twisted 20 to 25 degrees just distal to the bend to appose the shaft of the humerus. The reconstruction plate has holes that allow angulation of the screws as they are passed through the plate.

The screws passing through the plate and the humeral head into the glenoid fossa are inserted first (Fig. 21-12).

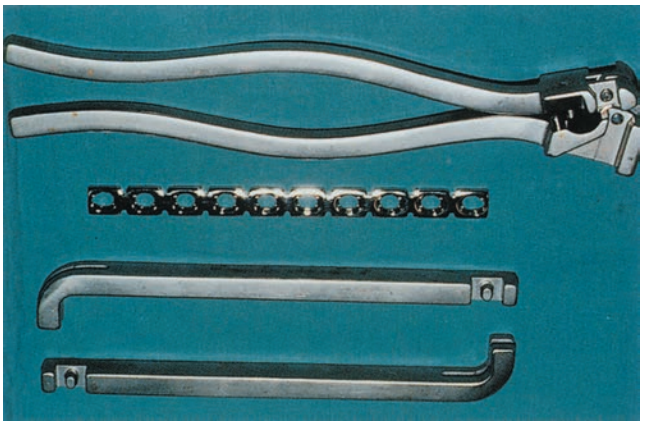

Figure 21-10 Handheld plate press and bending irons used to contour the pelvic reconstruction plate intraoperatively.

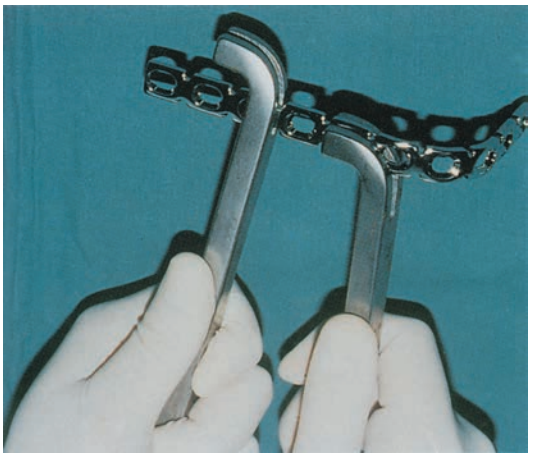

Figure 21-11 Plate must usually be contoured in the sagittal plane to appose the surface of the underlying humerus.

Two (usually) or three (sometimes) 6.5-mm compression screws can be inserted in this fashion. If the glenoid is dysplastic, only one screw can be used. These screws compress the arthrodesis site. A screw should be directed next from the spine of the scapula into the base of the coracoid process. Because of the cortical bone in this region, care must be taken not to break the drill bit when drilling into the scapula. Another cancellous screw is placed across the acromiohumeral fusion site, and the remaining holes of the plate are secured with cortical screws (Figs. 21-13 and 21-14). The acromion is not osteotomized, for it is used to augment fixation of the scapula to the humerus. Creating a notch in the lateral acromion may reduce plate prominence in this area (Fig. 21-15). Autogenous bone graft is not used routinely. If there is deficiency of the glenoid or humerus, a bone graft should be used. This is often the situation when the procedure is performed for failed total shoulder arthroplasty (see later discussion).

The arm of the patient is supported postoperatively with a pillow and swathe. A thermoplastic thoracobrachial

Figure 21-12 Transluminated scapula: There is little bone available for fixation in the scapula except in the area of the glenoid and coracoid base. Apart form the borders of the scapula, these are the only areas that do not transluminate.

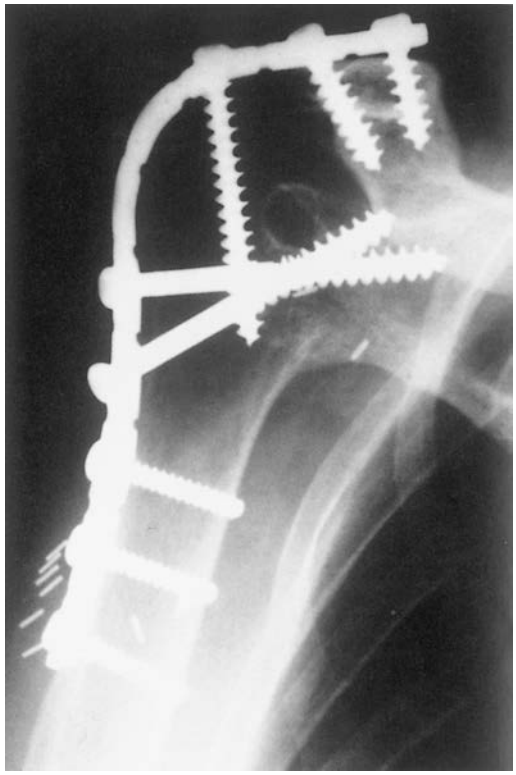

Figure 21-13 Anteroposterior radiograph following glenohumeral arthrodesis with a single plate. Two screws have been inserted into the glenoid to provide fixation. These screws are inserted first to compress the arthrodesis site and augment the fixation by interdigitation of the cancellous bone surfaces.

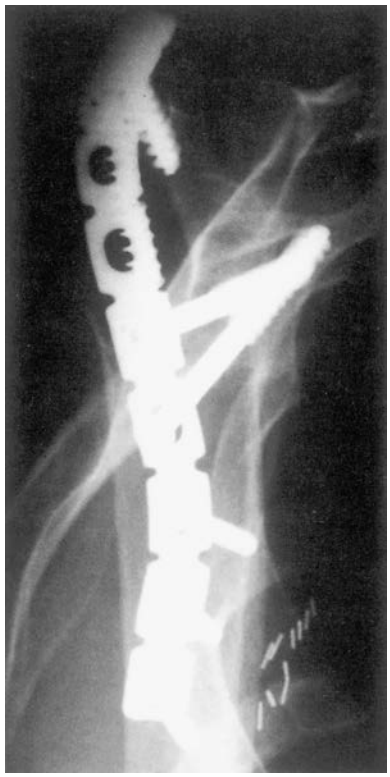

Figure 21-14 Lateral radiograph following glenohumeral arthrodesis with a single plate. The plate has been contoured in the sagittal plane to appose the surface of the humeral shaft.

orthosis is applied 24 hours postoperatively (Fig. 21-16). If possible, the orthosis can be fabricated preoperatively and then adjusted in the postoperative period as necessary. The orthosis is worn for 6 weeks postoperatively. During this time it can be removed for short periods while the patient showers, providing the arm is supported. If quality of the patient's bone is good, the fixation obtained intraoperatively solid, and the patient reliable, sling immobilization alone, without the use of an orthosis, can be considered. Six weeks postoperatively the patient is examined radiographically, and the stability of the fusion is tested manually in a gentle fashion.

If there is no radiographic sign of loosening of the internal fixation after 6 weeks, the patient's arm is placed in a sling (Fig. 21-17). Gentle range-of-motion exercises are allowed until radiographic union is achieved. It is difficult to be certain when fusion occurs radiographically because the fixation is sufficiently rigid that very little callus forms around the arthrodesis site. Muscle strengthening is encouraged after removal of the thermoplastic thoracobrachial orthosis, but return to strenuous activity is delayed for at least 16 weeks postoperatively. Thoracoscapular strengthening and mobilization exercises can usually be started 3 months following surgery.

The plate and screws are not routinely removed. If the internal fixation device is prominent and the overlying soft tissues tender and/or subject to irritation from clothing or prosthetic harnesses, the fixation should be removed. If the internal fixation is removed, the patient should be informed that there is an initial risk of humeral fracture due to the stress-raising effect of the screw holes until the local bone has a chance to respond to the change in stress distribution created by the loss of the support provided by the fixation device.

Special Problems

Elbow Flexorplasty

Some forms of elbow arthroplasty can be performed at the time of glenohumeral arthrodesis. If arthrodesis of the shoulder and elbow flexorplasty can be accomplished under one anesthetic, morbidity and the time required for rehabilitation can be decreased. Pectoralis major tendon transfer is an example of a form of elbow flexorplasty that can be performed at the time of glenohumeral arthrodesis (Figs. 21-18 and 21-19). When performing this type of flexorplasty at the same time as glenohumeral arthrodesis, it is important to not set the tension on the transfer until the position of the shoulder has been secured with a plate.

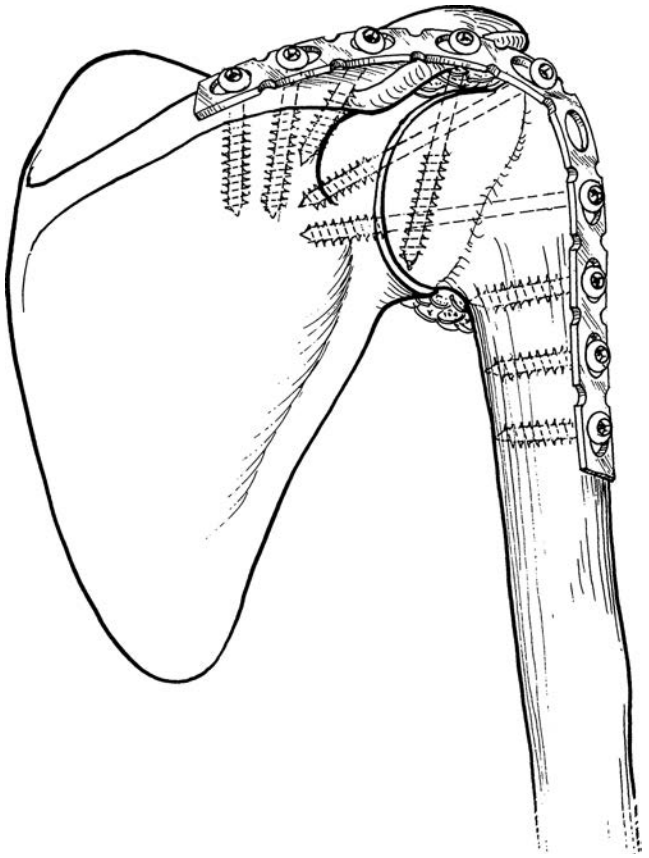

Figure 21-15 Drawing of a 10-hole Association for Osteosynthesis (AO) reconstruction plate applied to stabilize the arthrodesis. The humeral head contacts the acromion and the glenoid, and there is cancellous bone graft packed into the interval between the inferior humeral neck and the glenoid.

Figure 21-16 Patient wearing a thermoplastic thoracobrachial orthosis a few days following glenohumeral arthrodesis. The orthosis can be constructed preoperatively and adjusted in the immediate postoperative period. The orthosis is worn full time for 6 weeks and part time (when out of the house) for another 6 weeks postoperatively.

This type of reconstruction can accomplish a great deal for the patient with a flail elbow and shoulder who has a favorable type of brachial plexus injury (Figs. 21-20 and 21-21). Doi et al.[16] have reported on the use of free tissue transfer in conjunction with shoulder arthrodesis and other procedures for upper extremity reconstruction following complete avulsion of the brachial plexus.

Bone Loss

Bone loss can be a significant problem when performing glenohumeral arthrodesis. The usual etiologic factors are trauma, previous glenohumeral joint arthroplasty, and tumor resection. The extent of bone loss should always be assessed preoperatively. It is usually possible and practicable to assess the extent of humeral bone loss on conventional radiographs. It is harder to assess the extent of glenoid bone loss on plain films; therefore, a computed tomography (CT) scan may be required. Certainly, if the patient has had a previous glenoid replacement or has a scapular tumor, additional imaging is required. If bone loss is present, bone grafting must be performed in conjunction with glenohumeral arthrodesis. The various types of bone grafting are discussed in the following section, and the method selected will depend on the extent of bone loss determined preoperatively.

Bone Grafting

Routine bone grafting is advocated by some authors during glenohumeral arthrodesis. The AO/ASIF group recommends prophylactic bone grafting at the termination of a plate if a plate is used for internal fixation. Of Cofield and Briggs' 71 patients, 11 had autogenous bone grafts at the time of their initial procedure.[11] Richards et al. obtained successful arthrodesis without primary bone grafts in patients with significant osteoporosis.[45] I do not routinely use bone grafts in adults when performing glenohumeral arthrodesis. Bone grafting is indicated to fill large defects in patients who are undergoing glenohumeral arthrodesis for complex and revision problems. Bone grafting is also indicated following tumor resection. If large defects have been created following such resection, free vascularized bone grafts may be indicated. Nonunion of shoulder arthrodeses should be treated by revision of the arthrodesis combined with bone grafting. An attempt must be made to obtain rigid internal fixation during the arthrodesis. This method of treatment has, in my own experience, been successful when used.

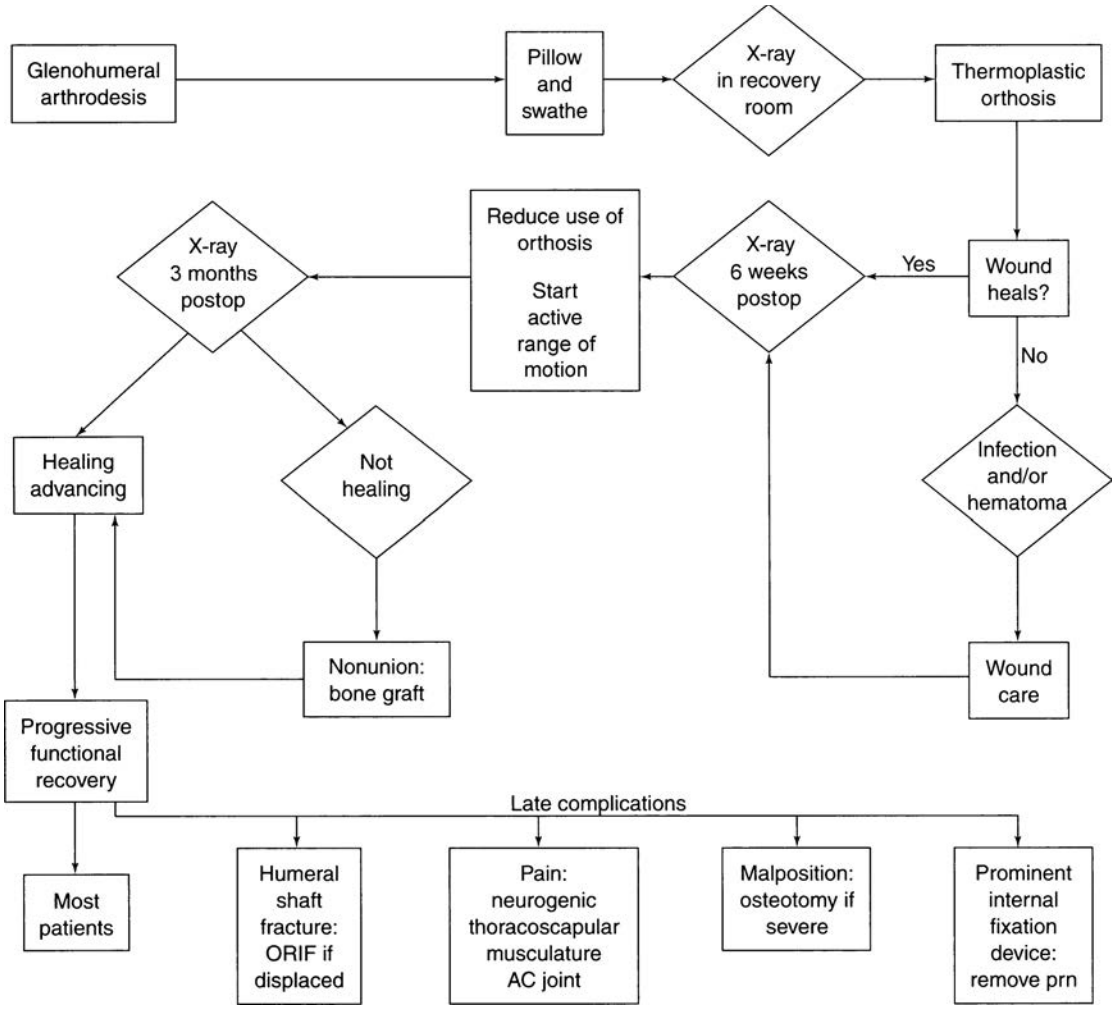

Figure 21-17 Algorithm for management following glenohumeral arthrodesis. AC, acromioclavicular; ORIF, open reduction, internal fixation.

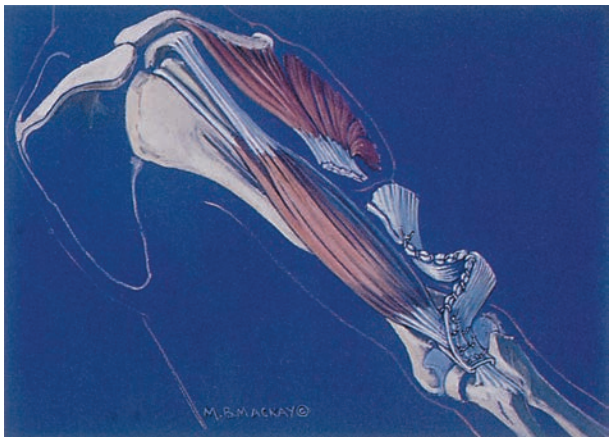

Figure 21-18 Simultaneous glenohumeral arthrodesis and elbow flexorplasty. A tube of fascia lata harvested from the opposite thigh is sewn to the biceps tendon distally. The fascia lata is passed subcutaneously proximally to the area of the pectoralis major insertion.

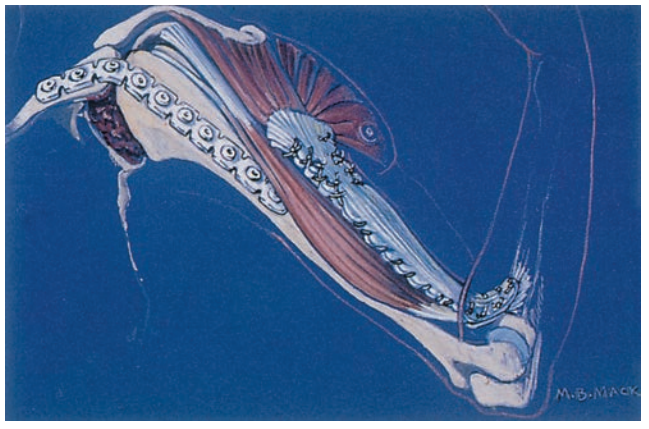

Figure 21-19 After stabilization of the glenohumeral arthrodesis with a plate, the fascia lata is sewn at the appropriate tension to the pectoralis major tendon. Patients are treated in a thermoplastic orthosis as described in Figure 21–16.

Figure 21-20 Male patient with an irreparable lesion of the brachial plexus a few months following simultaneous glenohumeral arthrodesis and elbow flexorplasty using fascia lata to the pectoralis major tendon.

Figure 21-21 The patient can actively flex his elbow through a functional range. The pectoralis major tendon transfer is seen as a web in the anterior axillary fold.

Nonstructural Autogenous Bone Grafting

Autogenous bone grafting can be used to provide osteoinductive and osteoconductive material at or in the arthrodesis site to increase the likelihood of successful fusion. Some authors routinely use cancellous bone harvested from the humeral head when performing glenohumeral arthrodesis. In complex and revision cases the humeral head usually is not present, and it is necessary to look elsewhere for autogenous bone. My preference is to harvest bone from the inner aspect of the iliac crest when a nonstructural autogenous graft is required. This is conveniently performed with the patient in the semisitting position unless the patient is obese, in which case the bone graft must be harvested with the patient supine and the position changed after closure of the iliac crest wound. I resect the superior and inner cortex of the iliac crest with a sagittal saw. A Capener gauge is then used to excavate strips of cancellous bone. If large amounts of bone are required, it may be necessary to use both iliac crests. If the patient has had previous surgery in these areas, a bone graft can be harvested from the posterior aspect of the iliac crest, although the position of the patient must be changed intraoperatively. Alternatively, nonstructural autogenous bone can be obtained from the greater trochanter or provided by morselized rib grafts.

Structural Autogenous Bone Grafting

Structural autogenous bone grafting uses an anatomically intact bone graft, such as a full-thickness iliac crest or fibular strut graft. Following unsuccessful glenohumeral joint arthroplasty, there is a significant defect relating to the often extensive resection of the humeral head and excavation of the proximal humerus. A full-thickness (tricortical) iliac crest graft can be harvested and incorporated into the fusion mass (Fig. 21-22). I have placed such grafts underneath the plate with screws passing through the plate and the tricortical iliac crest bone graft, and into the glenoid. Such a graft will maintain the contour of the shoulder and provide autogenous osteoconductive tissue across which fusion can occur. The disadvantage of an anatomically intact graft is prolonged time required for incorporation. Accordingly, when such a graft is used, it should be supplemented with small chips of cancellous bone. It is usually necessary to harvest the cancellous bone from the other iliac crest. I have not used fibular grafts when performing glenohumeral arthrodesis except in conjunction with a vascularized fibular graft. Nonvascularized fibular grafts consist of dense cortical bone, have a limited biologic (but initially a good structural) potential, and probably should not be used in isolation.

Vascularized Autogenous Bone Grafting

Vascularized autogenous bone grafting has the advantage of providing tissue with great biologic potential. When the surgeon is faced with an intercalary defect that is larger

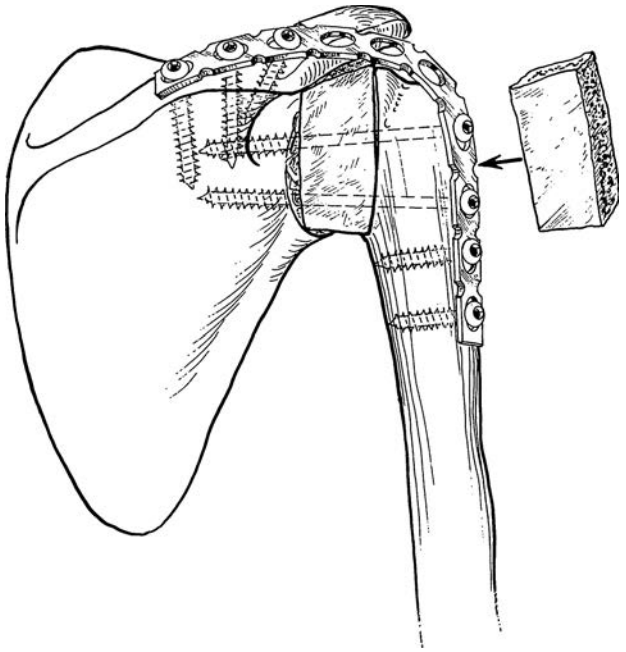

Figure 21-22 A corticocancellous block of autogenous iliac crest graft can be used to promote a fusion in the setting of proximal humeral deficiency, as may occur after a failed arthroplasty procedure.

than 6 cm, a vascularized bone graft should be considered. Following tumor resection, intercalary defects of this magnitude are common, and I have used vascularized fibular bone grafts in this situation. The bone grafting is performed in conjunction with plate fixation. My preference is to use a very long heavy plate spanning the entire intercalary defect (Fig. 21-23). The vascularized fibular bone graft should be fixed in position with a minimum amount of internal fixation at each bony juncture. Vascularized fibular bone grafts of up to 25 cm can be used to span very large defects. The vascular anastomosis is performed between the peroneal artery and its vena comitantes and a branch of either the axillary or the brachial artery. Vascularized fibular bone grafts should be supplemented with chips of autogenous bone at each juncture to maximize the likelihood of fusion occurring.

Vascularized full-thickness iliac crest bone grafts can be used to provide both bone and soft tissue. The use of such a bone graft creates a significant donor deficit, but can be considered if a bone and soft tissue defect coexist. The shape of the iliac crest limits its usefulness somewhat for long intercalary defects, although the graft can be osteotomized to straighten it. Alternatively, vascularized iliac crest bone grafts can be used in conjunction with a nonvascularized fibular graft or an allograft.

Allograft Reconstruction

Allograft reconstruction can be considered in conjunction with bone grafting when performing glenohumeral

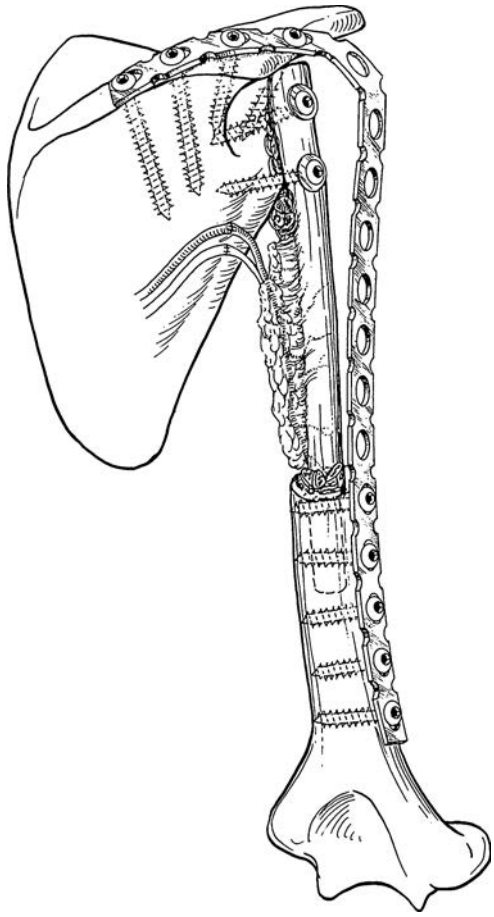

Figure 21-23 Drawing of the technique of vascularized fibular graft used to span a deficiency of the proximal humerus. Cancellous bone graft has been placed at both ends of the fibular graft.

arthrodesis for complex and revision cases.[9] It is my preference, wherever possible, not to use allograft bone if autogenous bone is available. For instance, if bone grafting is required to supplement a vascularized fibular graft, I would consider using the contralateral fibula or iliac crest graft to supplement the vascularized fibular graft in preference to an allograft. At times, allograft must be used owing to the nonavailability of autogenous bone. Both structural and nonstructural allograft bone can be used. More commonly, allograft is used in conjunction with a large implant to perform an arthroplastic reconstruction following tumor resection. This is discussed elsewhere in Chapter 37.

Soft Tissue Loss

Soft tissue loss can be caused by trauma or the necessity to resect soft tissue to obtain clear margins following tumor resection. The most common soft tissue deficiency about the shoulder relates to the rotator cuff. Isolated rotator cuff deficiency does not require special consideration and, in fact, our conventional method of glenohumeral arthrodesis involves resection of the rotator cuff in its entirety.

Local Flap Techniques

If deltoid resection is required for tumor ablation or if the deltoid has been lost as a result of trauma, consideration should be given to augmentation of the soft tissue envelope. In particular, it is important to have well-vascularized muscle surrounding the arthrodesis site. The presence of good muscle coverage is associated with enhanced bone formation and enhanced revascularization of autogenous dysvascular or avascular bone as seen following bone grafting.[48] Fortunately, local, pedicled, and free flap techniques are available to provide a stable soft tissue envelope about the shoulder. The latissimus muscle provides a local source of autogenous tissue that can be rotated to cover the shoulder. The entire latissimus muscle can be elevated on its vascular pedicle and rotated easily to cover the shoulder. The latissimus has an excellent blood supply, and most surgeons are familiar with its dissection. The flap can be rotated as either a pure muscle flap or as a myocutaneous flap, depending on the patient's requirements. If the nerve supply to the latissimus is kept intact, it can also be used as a flexorplasty, as described by Zancolli and Mitre.[62] The requirement to use a latissimus flap in this manner might be seen following trauma to the shoulder and proximal humerus resulting in the loss of biceps function. The latissimus flap should be considered to be the mainstay of treatment when soft tissue is required locally. Hasan et al.[22] have described the use of a pedicled forearm musculofasciocutaneous flap elevated just before above-elbow amputation and subsequently transposed to the shoulder to provide coverage of hardware used to perform shoulder arthrodesis in three patients.

Free Tissue Transfer

If a latissimus flap cannot be used, consideration can be given to a free tissue transfer. Such a transfer is required relatively infrequently about the shoulder. The trapezius should not be used to cover a soft tissue defect following glenohumeral arthrodesis. Good function of the trapezius is required to motor the shoulder girdle following arthrodesis, and its anatomy should not be disturbed.

"Hybrid" Methods of Reconstruction

Complex and revision problems about the shoulder require special techniques when performing glenohumeral arthrodesis. In some cases a combination of techniques is required and such hybrid methods of treatment are in the patient's best interest when faced with complex bone and soft tissue deficits. For instance, following tumor resection, it may be necessary to perform a vascularized fibular bone graft, a pedicled latissimus muscle flap, an autogenous iliac crest bone graft, and a pectoralis major to scapular transfer (owing to long thoracic nerve palsy) to stabilize the shoulder girdle and provide enough tissue so that arthrodesis can occur. The timing and staging of such complex procedures must be assessed on an individual basis.

PERSONAL SERIES

I have performed 91 glenohumeral arthrodeses. The ages have ranged from 19 to 64. Seventy-two patients were operated on for irreparable lesions of the brachial plexus, six patients for recalcitrant shoulder instability, five patients for osteoarthritis, four patients for failed total shoulder arthroplasties, and two patients for sepsis. The technique used was that of combined glenohumeral and acromiohumeral arthrodesis, as described earlier. In my home community tumor surgery is almost exclusively performed by other colleagues. Accordingly, I have had less experience with glenohumeral arthrodesis for this indication.

Results

Solid arthrodesis has usually been obtained with the primary procedure. Two patients developed only an acromiohumeral fusion and one of these patients developed a broken screw. This patient had a secondary bone-grafting procedure. Infection occurred in only one case. Clinical examination showed all shoulders to be fused within 10 degrees of the desired position. We have not used special techniques to assess the position of the arthrodesis.[25] Eighteen patients have required surgery for plate removal, and two patients sustained fractures of the humerus distal to the plate.

Activities of Daily Living

In a review of 33 patients we assessed functional outcome in detail.[47] The patients' ability to perform specific activities of daily living (ADLs) following their glenohumeral arthrodesis were ranked as seen in Table 21-1.

TABLE 21-1
ACTIVITIES OF DAILY LIVING*

Working at waist level	29
Getting dressed	29
Sleeping	27
Lifting bags	25
Using knife	25
Washing face	25
Reaching front pocket	23
Working at shoulder level	21
Doing up buttons	21
Reaching opposite axilla	20
Eating	17
Managing toileting	13
Reaching back pocket	11
Bathing	7
Washing back	6
Doing hair	5

*Number of patients/Total patients (33)

The ability to perform ADLs was heavily dependant on the adequacy of hand function in patients with brachial plexus injuries. Regression analysis revealed the underlying indication (preoperative diagnosis) for glenohumeral arthrodesis to be the single best predictor of the ability of patients to perform their ADLs following glenohumeral arthrodesis. The patient's compensation board status was also a predictor of outcome. Patient satisfaction was highest in those patients undergoing the procedure for a brachial plexus injury, osteoarthritis, and failed total shoulder arthroplasty.

Five patients who had neurogenic pain preoperatively continued to complain of significant neurogenic pain postoperatively. Several other patients who had compensable injuries continued to complain of postoperative pain in spite of solid arthrodesis. Most patients can reach both their front and back pockets following surgery, and many have returned to fairly heavy occupations. These occupations have included tool and dye manufacture, gardening, heavy equipment operation, and brick laying. Patients who had glenohumeral arthrodesis for compensable injuries did not return to their previous occupations. The author has not experienced acromioclavicular joint problems following glenohumeral arthrodesis. Some authors recommend excision arthroplasty of the acromioclavicular joint following arthrodesis in an effort to maximize shoulder motion.[41] I have not found it necessary to perform this adjunctive procedure in my series of patients. In the following paragraphs I will discuss fusion, function, and pain following glenohumeral arthrodesis in more detail.

Fusion

Fusion rates following glenohumeral arthrodesis are high. Most older series do not report any nonunions. Recent series that have used internal fixation devices report high union rates. It is often difficult to judge radiographic union in patients who have had internal fixation. The internal fixation device commonly obscures the arthrodesis site, and rigid internal fixation prevents the formation of periarticular new bone. Because I routinely use rigid internal rotation, I tend to arbitrarily discontinue or decrease external immobilization by 6 or 8 weeks. The shoulder is examined clinically and, assuming that the patient is comfortable, the patient is placed in a sling at this time. Any attempt to actively mobilize the thoracoscapular musculature is avoided until at least 3 months after arthrodesis. By using this method of postoperative rehabilitation, I have found delayed union or nonunion to be distinctly uncommon.

Function

The amount of improvement in shoulder function following arthrodesis is dependent on the function that was present preoperatively. Patients who have flail shoulders experience a significant improvement in glenohumeral function following arthrodesis because they can actively position their extremities in space (Fig. 21-24). Shoulder subluxation is relieved, and function is significantly improved. Most, but not all, patients can use their extremity to lift, dress, tend to personal hygiene, eat, and comb their hair (Figs. 21-25 and 21-26). In Cofield and Briggs' series, hair combing was the most difficult task to perform following arthrodesis of the glenohumeral joint.[11]

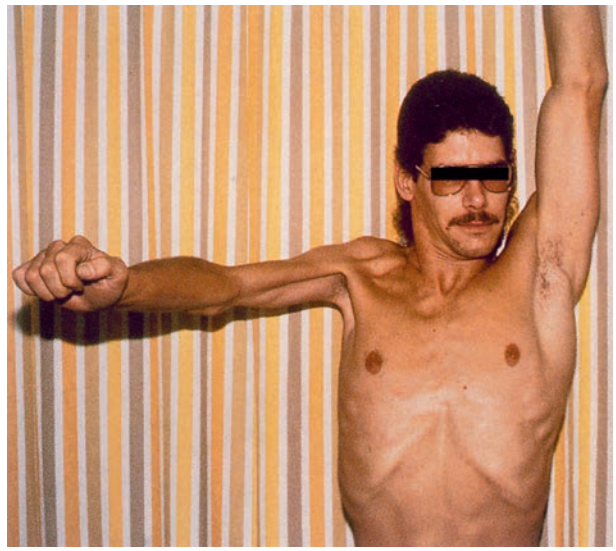

Figure 21-24 Range of shoulder elevation following glenohumeral arthrodesis. Most patients recover approximately two-thirds the normal range of motion following glenohumeral arthrodesis. The procedure effectively prevents glenohumeral rotation.

Chammas et al.[7] report that glenohumeral arthrodesis improved function in patients who had recovered active elbow flexion after brachial plexus even when the hand remained paralyzed.

Pain

Pain relief is not universal following glenohumeral arthrodesis. In fact, some pain is commonly present in patients whose shoulders have been successfully arthrodesed. The presence of moderate to severe pain following successful glenohumeral arthrodesis is difficult to explain, but occurs not infrequently. It is thought that such pain is related to the contiguous soft tissues. Glenohumeral arthrodesis places a substantial amount of strain on the periscapular musculature and requires the patient to make a profound functional adjustment. Pain surrounding successful fusions has also been reported following hip and knee arthrodesis. Most patients experience less pain with longer follow-up periods, and this suggests that with time the periscapular musculature adjusts to the changes imposed on it by glenohumeral arthrodesis. Clare et al.[10] have reported that excessive abduction or flexion of the arthrodesis is associated with chronic postoperative pain.

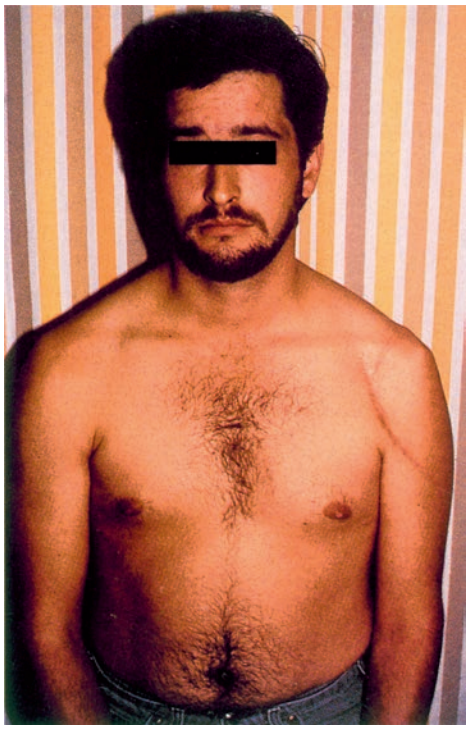

Figure 21-25 An anteroposterior clinical photograph of a patient following successful glenohumeral arthrodesis in the functional position. The arm can drop comfortably to the side even through thoracoscapular movement even though the glenohumeral joint has been arthrodesed in the 30-30-30 position.

Patients with neurogenic pain caused by brachial plexus injury will not experience pain relief following glenohumeral arthrodesis. Shoulder function is improved in these patients, but they should understand fully that the procedure is being performed to improve function and not to relieve pain. It has been our impression that if function is improved, such neurogenic pain is generally better tolerated by the patient. Therefore, we concentrate on the restoration of limb function, rather than on relief of pain by neurosurgical means.

Figure 21-26 Most patients can reach their mouth, their ear, their front pocket, and the side of their hip following glenohumeral arthrodesis.

Acromioclavicular Joint Pain

Acromioclavicular joint pain has been reported following glenohumeral arthrodesis.[41] Some methods of glenohumeral arthrodesis require osteotomy of the acromion. Such osteotomies disturb the normal acromioclavicular relation. If pain arises from the acromioclavicular joint postoperatively, excision arthroplasty is indicated if local injections confirm that the pain is arising from the acromioclavicular joint. Some authors have reported anecdotally that excision arthroplasty of the acromioclavicular joint improves motion following glenohumeral arthrodesis.[41] In our experience, acromioclavicular joint pain is rare following glenohumeral arthrodesis when the acromion is left in the anatomic position. Therefore, I do not recommend acromial osteotomy to approximate the acromion to the superior surface of the humeral head. I do advocate moving the humerus proximally to appose both the acromiohumeral and glenohumeral arthrodesis sites. The glenoid presents a small surface to the humeral head across which arthrodesis can occur. This is why decortication of the undersurface of the acromion as well as the glenoid and humeral head is advocated. The presence of a broader surface across which arthrodesis can occur is desirable, and it is believed that this increases the arthrodesis rate.

COMPLICATIONS

Nonunion

Nonunion following glenohumeral arthrodesis is surprisingly infrequent in view of the magnitude of the procedure, the high stresses across the arthrodesis site, and the difficulty of postoperative immobilization. In Cofield and Briggs' series of 71 shoulder arthrodeses, only three patients went on to nonunion.[11] All three patients' shoulders were successfully fused following a second operative procedure. If nonunion occurs following glenohumeral arthrodesis, a repeat operation with revision of the internal fixation device is indicated. Bone grafting should be used to augment the arthrodesis site if a nonunion has occurred. I have seen a few patients who have obtained an acromiohumeral fusion, but have not fused solidly at the glenohumeral joint. In one such patient the internal fixation device failed and bone grafting was necessary.

Infection

Infection is relatively uncommon following glenohumeral arthrodesis owing to the excellent vascularity of the periarticular tissues. Infection following glenohumeral arthrodesis

should be treated with surgical drainage and the appropriate parenteral antibiotics. The internal fixation should not be removed if it is providing stability at the arthrodesis site. An attempt should be made to obtain solid arthrodesis before removal of any fixation device.

Malposition

Excessive abduction of the extremity during glenohumeral arthrodesis can place a substantial strain on the thoracoscapular musculature. Adult patients have great difficulty adapting to positions of greater than 45 degrees of abduction. Hyperabduction at the arthrodesis site causes significant winging of the scapula for the arm to drop to the patient's side. Indeed, in some patients the arm will not approximate the trunk if the shoulder has been arthrodesed in too much abduction. Women in particular are unhappy with the cosmetic appearance of a shoulder that has been fused in too much abduction owing to the significant prominence of the scapula that is created when the arm is adducted.

Fusion of the shoulder in too much internal rotation can occur. If this occurs, the patient cannot easily bring his or her hand to the mouth and cannot reach either his or her front or back pocket. Rotational osteotomy of the humerus may be necessary for patients whose extremities have been positioned in too much internal rotation.[54] I have performed three such osteotomies for this indication. Suprascapular nerve entrapment has been reported following glenohumeral arthrodesis.[58]

Prominence of the Internal Fixation Device

Many patients who have had internal fixation devices applied over the spine of the scapula have significant skin tenderness in the area of the appliance. This can be particularly troublesome when the patient must wear a prosthetic harness, and skin irritation and ulceration has been reported.[44] Cofield and Briggs[11] report a significant incidence of tenderness over the internal fixation device, which required its removal in 17 of their patients. My experience has been similar, although use of a malleable reconstruction plate has decreased skin tenderness and made the need for removal of internal fixation less frequent.[45] The development of appliances specifically designed for glenohumeral arthrodesis is likely to decrease the incidence of tenderness of the skin overlying internal fixation devices even further.

Fracture of the Humerus

Fracture of the humerus at the distal end of the internal fixation device is sufficiently common that the AO group has recommended prophylactic bone grafting of this area.[37] Cofield and Briggs[11] report a fracture in the fused extremity occurring in 10 of their 71 patients. This complication has occurred in our series as well in association with significant trauma to the arthrodesed shoulder. If an unstable fracture does occur at the end of the internal fixation device, I advocate removal of the device together with internal fixation of the fracture if the arthrodesis has solidly healed. If the fracture is relatively undisplaced, it can be treated by closed means, but in our experience this is uncommon (Fig. 21-27).

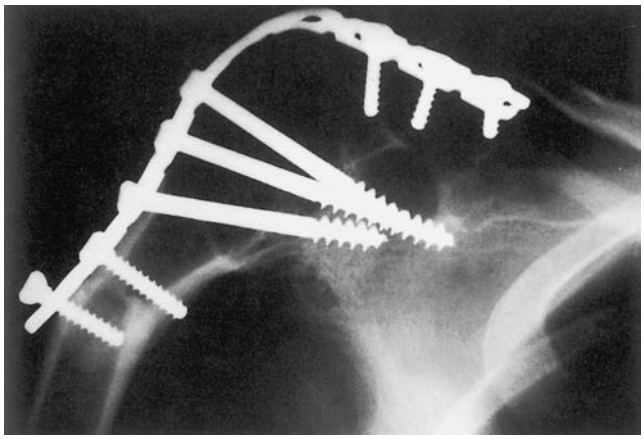

Figure 21-27 Fracture of the humerus at the end of the plate. This particular patient had a solid glenohumeral arthrodesis when he fell onto his arthrodesed arm. The fracture is minimally displaced in this case and can be treated nonoperatively, unless further displacement were to occur.

SUMMARY

Glenohumeral arthrodesis remains an important procedure that should be part of every shoulder surgeon's armamentarium. The procedure is indicated for patients with paralytic disorders, those who require en bloc resection of their glenohumeral joint together with the rotator cuff or deltoid for tumor, and those whose shoulder joints have been destroyed by septic arthritis. The procedure has been useful in a few patients with failed total shoulder arthroplasties and shoulder instability. It is rarely indicated for patients with rotator cuff tears, periarticular malunions, osteoarthritis, or rheumatoid arthritis.

Although a variety of different positions for glenohumeral arthrodesis has been advocated in the literature, my recommendation is a position of 30-degrees abduction (measured clinically), 30-degrees flexion, and 30-degrees internal rotation. This position brings the hand to the midline anteriorly so that, with elbow flexion, the patient can reach his or her mouth. With the shoulder fused in this position the arm drops comfortably to the side. I recommend fusion of both the acromiohumeral and glenohumeral articulations and the use of internal fixation when performing glenohumeral arthrodesis. Complications following glenohumeral arthrodesis are relatively infrequent and most can be dealt with successfully if they do occur.

The improvement in shoulder function following glenohumeral arthrodesis is dependant on the patient's preoperative shoulder function. Patients with flail shoulders experience a significant improvement in function. Patients with significant mechanical pain in the glenohumeral joint experience relief following the procedure. Glenohumeral arthrodesis does not relieve neurogenic pain and requires a significant functional adjustment on the part of patients who have had the procedure. Glenohumeral arthrodesis is primarily indicated to restore function. Bone grafting is routinely used for complex and revision procedures. The procedure is contraindicated in any patient with a progressive neurologic disorder affecting the periscapular musculature.

Acknowledgments

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22 Unconstrained Prosthetic Arthroplasty for Glenohumeral Arthritis with an Intact or Repairable Rotator Cuff: Indications, Techniques, and Results

Gerald R. Williams, Jr. *Joseph P. Iannotti*

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INTRODUCTION

History

The first replacement arthroplasty of the glenohumeral joint dates to 1893 when a French Surgeon, Pean, used a platinum and rubber implant to replace the proximal one-half of the humerus in a patient suffering from tuberculous arthritis of the shoulder.[110] The implant required removal approximately 2 years later for persistent, uncontrollable infection. Additional materials used in replacement of the proximal humerus predating the modern era of shoulder arthroplasty included fibular autografts, resurfacing grafts of fascia lata, and ivory and acrylic prostheses.[1,83,94,142]

The modern era of unconstrained shoulder arthroplasty was founded in the early 1950s, when prosthesis design

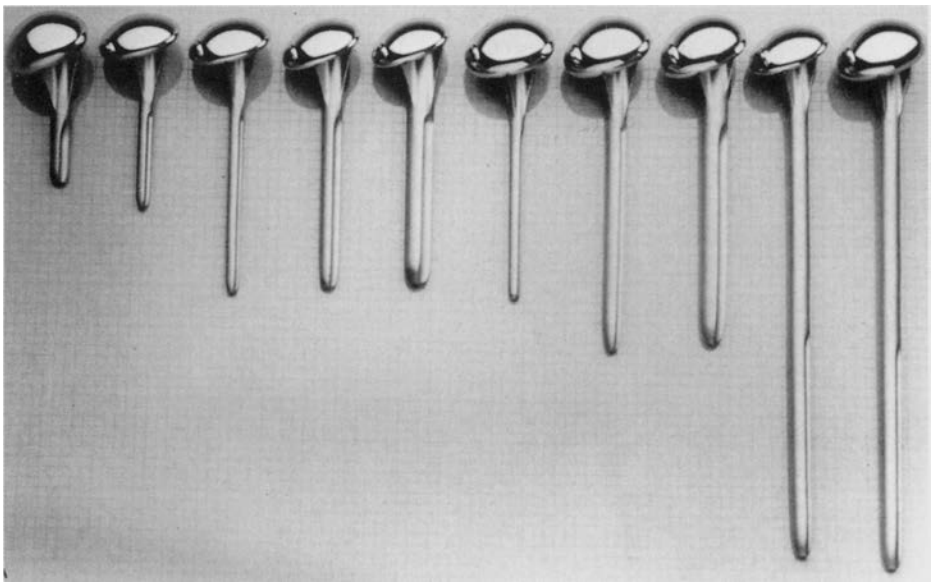


Figure 22-1 The Neer II system nonconstrained prosthetic arthroplasty system. Humeral components with two stem diameters, two neck lengths, and three stem lengths; standard polyethylene component; standard metal-backed component; revision 300% glenoid component. (From Neer CS II. Glenohumeral arthroplasty. In: Neer CS II, ed. *Shoulder reconstruction.* Philadelphia: W.B. Saunders Company, 1990:143, with permission)

sought to replicate the anatomic shape of the humeral head. The specific geometries of the earliest nonconstrained components were derived from measurements taken from cadaveric humeri.[97,128] In 1951 Krueger reported the implantation of the first anatomically designed humeral head prosthesis for a patient with aseptic necrosis.[97] The short-term results of this single surgery demonstrated promise, with relief of pain and favorable function being achieved. At the same time, Neer, being prompted by his observation of the unsatisfactory results of conventional surgical treatments for proximal humeral fracture–dislocations, was designing his first anatomic prosthesis, which was introduced in a report in 1953.[128] In 1955 Neer described the use of his second prosthesis, a redesigned vitallium humeral head replacement (Neer I prosthesis) in 12 cases, including patients with acute four-part proximal humeral fractures or proximal humeral fracture–dislocations and patients with avascular necrosis from prior fractures.[123] The short-term results in which 11 of 12 patients were free from pain provided momentum, furthering the application of humeral head replacement surgery. In 1964 Neer published a follow-up report in which the indications for the procedure expanded to include patients with degenerative arthritis of the glenohumeral joint.[129] With a follow-up ranging from 2 to 11 years, results remained favorable and supported the use of a humeral head replacement for acute trauma, posttraumatic arthritis, and both inflammatory and noninflammatory degenerative arthritis.

In the early 1970s, several authors reported on the implantation of a polyethylene glenoid component for the humeral head prosthesis to articulate with and, as such, the age of total shoulder arthroplasty arrived.[52,91,124,176] Neer designed a new humeral head prosthesis (Neer II prosthesis) for implantation with any of his glenoid component designs or as a humeral head replacement independent of a glenoid (Fig. 22-1). The Neer II system was the most widely used and reported nonconstrained shoulder replacement throughout the 70s and 80s.

Poor functional results with unconstrained prosthetic implants in certain patients with rotator cuff deficiency resulted in the development of constrained and semiconstrained devices. Constrained total shoulder arthroplasty conceptionally seemed desirable because of the inherent stability achieved at the articulating surfaces and the belief that, with the maintenance of a stable fulcrum of rotation, the deltoid would be able to assume the role of the rotator cuff in a cuff-deficient shoulder. A variety of designs were introduced, most of which used a fixed fulcrum ball-and-socket type construction (Fig. 22-2). Neer and Averill designed three constrained prosthetic systems, each with a modification to improve on the previous design.[130] In 1974 Neer abandoned the use of any constrained prosthesis because of dissatisfaction with mechanical failure.[125] Although pain relief was satisfactory in many cases of constrained total shoulder arthroplasty, these designs lost popularity owing to relatively high rates of component mechanical failure, disassembly, and loosening at the bone–implant fixation sites.[42,104,139,140]

Grammont and Baulot reintroduced the concept of constrained arthroplasty in cases of irreparable rotator cuff deficiency in the early 1990s.[71] Their design is a reverse ball-and-socket design with a relatively large glenoid sphere with no neck (Fig. 22-3). This places the center of rotation within the glenoid vault, thereby reducing the lever arm on the glenoid anchoring point.[19,71] This medialization of the center of rotation improves the deltoid moment arm and allows for improved function. Subsequent to Grammont and Baulot's prosthesis, Frankle et al. introduced a reverse ball-and-socket design with a slightly more lateralized center.[61] Early and midterm results demonstrate promising results and better survivorship than earlier constrained

Figure 22-2 A constrained total shoulder design with a reversed ball-in-socket articulation.

Figure 22-3 The Delta (Depuy, Warsaw, IN) prosthesis, introduced by Paul Grammont, is characterized as a reverse ball in socket with a relatively large metal sphere with no neck.

arthroplasties.[12,13,21,61,71,82,101,144,148,165,173] It is likely that constrained reverse prostheses will continue to have a role in the management of glenohumeral arthritis with irreparable cuff deficiency. The biomechanics, indications, and results of these prostheses are discussed in more detail elsewhere in this book.

Semiconstrained glenoid components or hooded glenoid components were designed to articulate with anatomic humeral head components for patients with rotator cuff–deficient shoulders. These glenoid components were modified with a roof-like extension or hood at the superior rim[127] (Fig. 22-4). The purpose of the hooded modification was to block superior translation of the humeral head from superiorly directed displacement forces inherent to rotator cuff deficiency. In theory, the semiconstrained design should allow the deltoid to independently achieve greater degrees of arm elevation. In the few articles that report on the use of semiconstrained prostheses, improvements in active arm elevation, if they occurred at all, were not dramatic, although pain relief was satisfactory.[29,50] The need for revision surgery and the number of complications were higher than in nonconstrained designs.[29,50,54]

Nonconstrained prostheses are the devices of choice for glenohumeral replacement with an intact and functional rotator cuff. Most arthroplasty systems utilize a stemmed humeral component in conjunction with a polyethylene glenoid component. However, humeral head resurfacing without an intramedullary stem has proponents, particularly when the glenoid is not being resurfaced.[2,57,106,108] Controversy exists with regard to the indications for glenoid resurfacing. This controversy arises predominantly as a result of the lack of well-controlled comparison data between total shoulder arthroplasty and hemiarthroplasty. Moreover, there may be a difference between hemiarthroplasties performed with stemmed implants and resurfacing arthroplasties.[161]

The topic of this chapter is replacement arthroplasty for conditions characterized by an intact or reparable rotator cuff. The two most representative conditions are osteoarthritis and avascular necrosis. Therefore, these two conditions will be considered most prominently. However, some cases of inflammatory (i.e., rheumatoid arthritis) and posttraumatic (i.e., arthritis of instability) arthritis fall under this category. The surgical technique is similar in these conditions and, therefore, is discussed all inclusively. Technical points germane to each individual diagnosis are inserted where appropriate.

Indications

In general, the indications for prosthetic replacement in osteoarthritis and avascular necrosis are similar to those

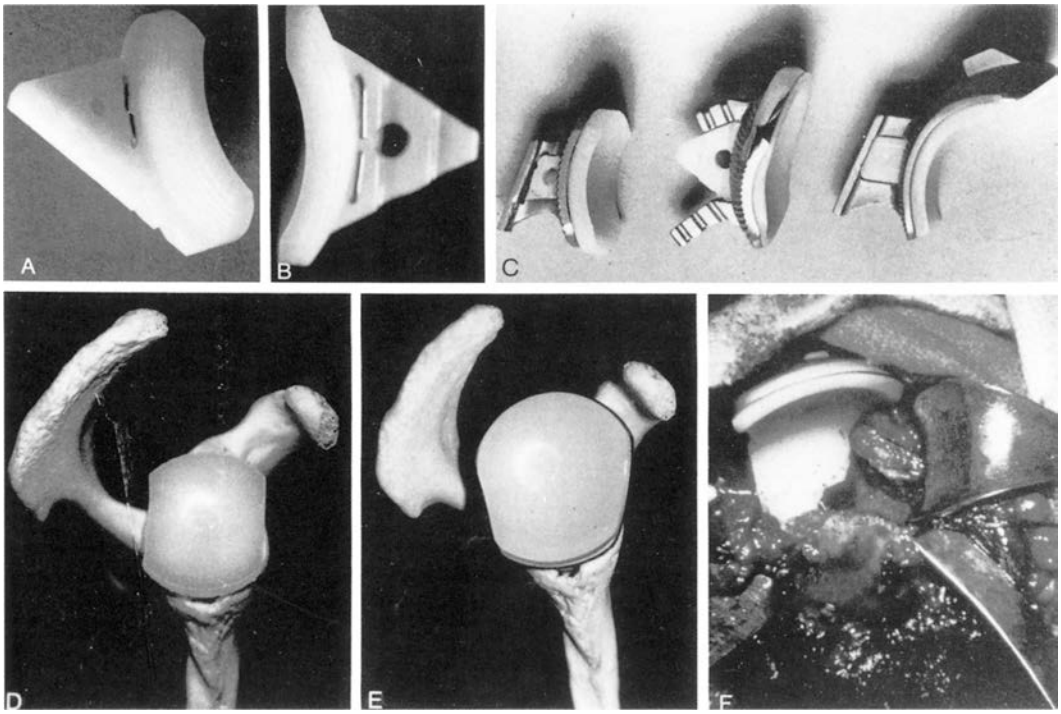


Figure 22-4 **(A)** The initial Neer glenoid component; **(B)** the modified all-polyethylene component; **(C)** the Neer initial design of the metal-backed glenoid components showing **(D)** the standard surface area glenoid; **(E)** the 200% surface area glenoid; and **(F)** the 600% surface area glenoid. (From Neer CS II. Glenohumeral arthroplasty. In: Neer CS II, ed. *Shoulder reconstruction.* Philadelphia: W.B. Saunders Company, 1990:143, with permission).

stated for the other pathologic processes discussed in this text. Severe pain that has failed to respond to conservative measures and a degree of dysfunction that is unacceptable to the patient are the primary reasons to consider arthroplasty. The average age of patients undergoing shoulder replacement is younger than those of other major joint replacements (i.e., hip and knee). Fortunately, less weight is borne by the shoulder than hips or knees. However, 15-year survival rates of 73% and 84% for hemiarthroplasty and total shoulder arthroplasty, respectively, have been reported for patients 50 years and younger.[155] Therefore, total shoulder arthroplasty is generally reserved for patients over the age of 50, while hemiarthroplasty is considered for patients younger than 50 and older than 40. These ages are only general guidelines and should be considered in addition to other variables such as activity level, degree of glenoid wear, degree of humeral head collapse, and general medical condition.

Patients 40 and younger, or older patients who are extremely physically active, with symptomatic glenohumeral arthritis with an intact rotator cuff requiring surgical intervention present a significant treatment dilemma. Prosthetic replacement puts them at high risk for multiple revisions. Other surgical options such as débridement, interposition arthroplasty, and resurfacing arthroplasty should be considered as alternatives to traditional hemiarthroplasty or total shoulder arthroplasty in these patients. These options are best presented as temporizing measures, used to provide some symptom relief and functional improvement without compromising bone stock required for future arthroplasty or other surgical options such as arthrodesis.

Little information is available regarding the longevity of resurfacing and interpositional arthroplasty. Levy and Copeland's review of their series of resurfacing arthroplasties revealed that those patients with primary osteoarthritis had the best outcome, and that only 8% required revision during the 5- to 10-year follow-up.[107] Long-term studies are needed to provide more information about the indications and life span of these prostheses. A simple treatment algorithm for osteoarthritis is shown in Fig. 22-5.

Surgical indications for patients with avascular necrosis are very similar to those for osteoarthritis. Osteonecrosis patients are generally much younger than their counterparts with osteoarthritis. However, with associated diseases such as sickle cell anemia or renal failure, their life span may be shorter. Therefore, the indications for hemiarthroplasty and total shoulder arthroplasty are based more upon the stage of avascular necrosis present, the projected life span of the patients, and the activity level of the patients, rather than their age. Stage I disease is silent and, therefore, generally does not require treatment. There is debate about whether core decompression of the humerus changes the incidence of late collapse. Obviously, if it does, stage I disease may be an

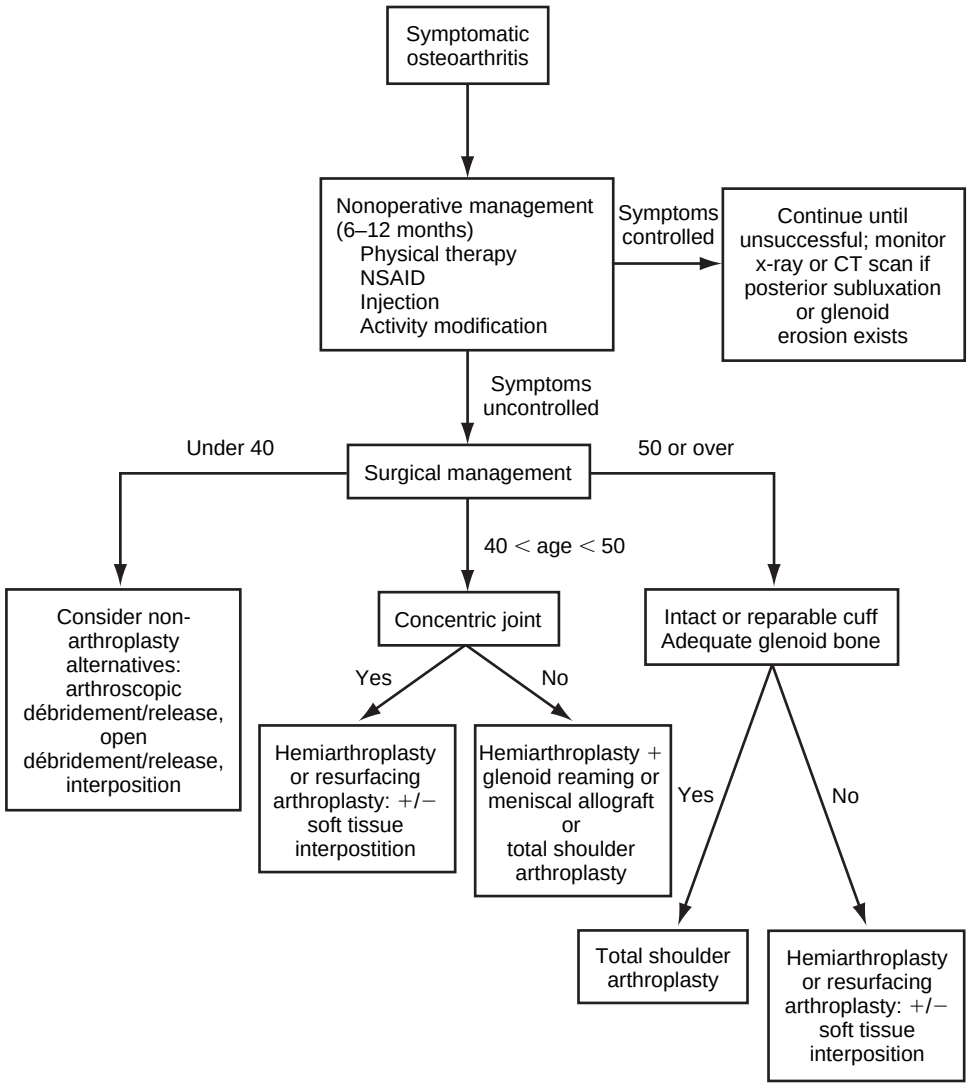


Figure 22-5 Treatment algorithm for osteoarthritis of the shoulder. CT = computed tomography; NSAID = nonsteroidal antiinflammatory drug.

indication for humeral head decompression. Until these data are available, most patients with stage I avascular necrosis are not surgical candidates. In stage II disease that does not respond to standard nonoperative treatment, core decompression with or without bone graft is indicated. Stage III (subchondral fracture) and IV (collapse) avascular necrosis may be indications for hemiarthroplasty, depending on the degree of collapse and patient activity level. Stage V osteonecrosis (glenoid involvement) is an indication for total shoulder arthroplasty so long as the cuff is intact or reparable and the activity level and life expectancy are appropriate. In active patients with a normal or near-normal life expectancy, an alternative to total shoulder arthroplasty is hemiarthroplasty with soft tissue interposition. A simple treatment algorithm for avascular necrosis is shown in Fig. 22-6.

The only absolute contraindication to prosthetic arthroplasty is active infection. Relative contraindications include concomitant rotator cuff *and* deltoid dysfunction, Charcot arthropathy, and severe brachial plexopathy.[75] Arthroplasty in patients with a remote history of infection should be approached cautiously. Although it is impossible to predict for certain the likelihood of recurrent infection, the risk is lower in patients with greater time intervals from the original infection, normal erythrocyte sedimentation rate, normal C-reactive protein, and negative cultures.

Risks of Nonoperative Treatment

The concept of risk associated with nonoperative management may not initially seem valid. However, if one assumes that a given patient with osteonecrosis or osteoarthritis will eventually require prosthetic replacement, the development or worsening of concomitant conditions known to have a negative effect on prognosis following arthroplasty is a relative risk of nonoperative treatment. Two such conditions in patients with osteoarthritis are stiffness (external rotation less than 10 degrees) and posterior subluxation.[81] Therefore, patients with osteoarthritis and mild stiffness or posterior

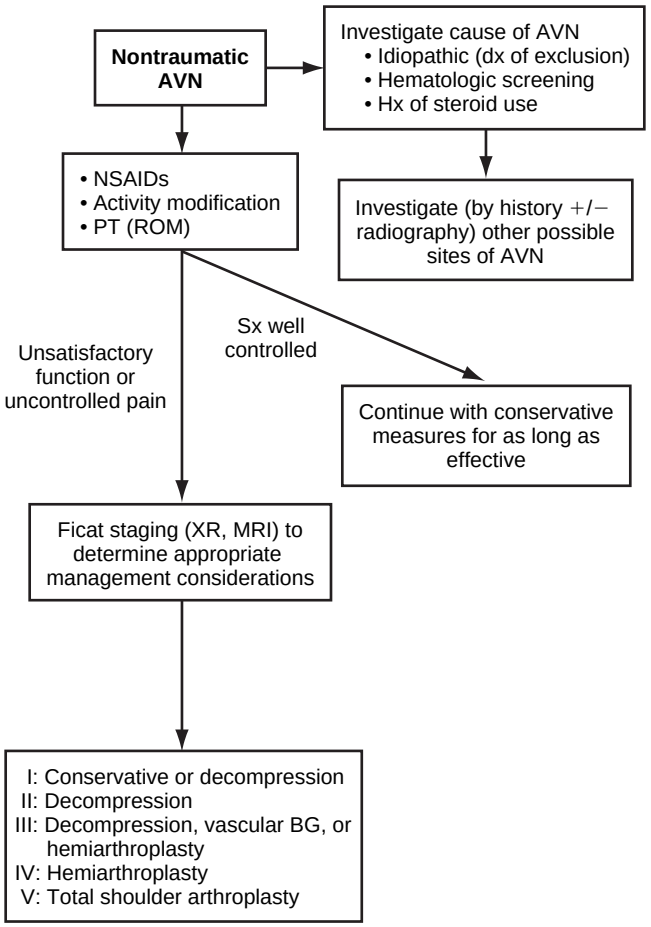


Figure 22-6 Treatment algorithm for osteonecrosis of the shoulder. AVN = avascular necrosis; MRI = magnetic resonance imaging; NSAIDs = nonsteroidal antiinflammatory drugs; PT = physical therapy; ROM = range of motion; XR = x-ray.

erosion should be counseled about the effect on prognosis of stiffness, pronounced posterior glenoid erosion, and posterior subluxation should they develop with further nonoperative management. Young patients (less than 50) with avascular necrosis should likewise be told that nonoperative management prolonged enough to develop significant glenoid involvement may subject them to the premature need for a glenoid component. Moreover, rheumatoid patients with an intact and functional cuff and good bone stock should be cautioned about the development of irreparable cuff deficiency and nonreconstructible bone loss with further nonoperative management. These treatment decisions are difficult and should be individualized; ultimately, the patient must decide between the relative risks of prosthetic replacement and nonoperative treatment.

Results

Results specific to individual diagnosis are discussed at the conclusion of this chapter. However, several comments regarding the results of shoulder arthroplasty in general deserve mention here. First, it is difficult to compare the results of shoulder arthroplasty from multiple publications and often difficult to assess the results within individual publications for several reasons. Data for pain, motion, function, patient satisfaction, and overall results have not been reported between publications with uniform methods. Factors such as method of component fixation and the status of the rotator cuff vary from report to report as well. Patients within a publication often represent a mixed group from whom the results of several diagnostic categories are tallied together and reported as a single data point. At times, results of patients receiving a humeral head replacement are not separated from patients receiving a nonconstrained total shoulder arthroplasty.

Neer et al. noted that the main factors influencing the overall results—the condition of the rotator cuff and deltoid musculature and the required details of surgery—varied depending on the diagnostic category being addressed.[127] As such, Neer et al. emphasized the importance of grouping patients to diagnostic categories when assessing the results of total shoulder arthroplasty.

Neer et al. reported their results using an overall results grading system that subdivided patients into two categories.[127] Depending on the status of the rotator cuff and deltoid musculature and the stability of the implant at the completion of surgery, patients were assigned to either a full rehabilitation category or a limited-goals rehabilitation category. A patient was assigned to a full rehabilitation category if the muscles were intact or reparable and component stability was verified intraoperatively.[127] Clinical results for full rehabilitation patients were graded as either excellent, satisfactory, or unsatisfactory, depending on the degree of patient satisfaction, pain relief, strength, and motion. An excellent result required that the patient had no pain or slight pain; had active abduction within 35 degrees of normal and 90% of normal external rotation; and was satisfied with the result. A satisfactory result was achieved if the patient had no pain, slight pain, or moderate pain only with vigorous activities; had active abduction of more than 90 degrees and 50% of normal external rotation; and was satisfied with the procedure. An unsatisfactory rating required failure to achieve any of these criteria.

A limited-goals rehabilitation category was assigned if the muscles were detached and not capable of recovering function after repair because of fixed contracture or denervation, or if the stability of the implant was judged to be problematic.[127] Clinical results for the limited-goals category were graded as either satisfactory or unsatisfactory, depending on patient satisfaction, pain relief, and restoration of limited, but useful, shoulder function. A satisfactory result required no pain, slight pain, or moderate pain only with vigorous activity, active abduction of more than 70 degrees, and external rotation of more than 20 degrees.

Pain relief is the most predictable benefit of total shoulder arthroplasty and appears to vary little between diagnostic categories. Approximately 90% of patients can be expected to attain good or excellent pain relief. When inadequate pain relief occurs, the source is often explained by an identified intraoperative or postoperative complication. More common reasons for inadequate pain relief include glenoid component loosening, malpositioning of the components, improper soft tissue balancing, rotator cuff tears, inadequate postoperative rehabilitation, and infection. In our experience it is uncommon for inadequate postoperative pain relief to lack explanation.

Range of motion has been a difficult area of assessment because only recently has a standardized system for measuring motion been recognized.[143] Assuming the arthroplasty was technically well performed, postoperative rehabilitation has been adequate, and the patient is motivated, active range of motion most directly correlates with the status of the rotator cuff and deltoid musculature. Osteoarthritis and osteonecrosis rarely have rotator cuff tears and most consistently obtain the greatest motion. Full or close to full active range of motion is not unusual postoperatively.[10,35,77,127] Gains in motion in rheumatoid arthritis patients are more variable, and several factors are likely responsible. The severity of disease varies and often affects other joints that interfere with the ability to rehabilitate the shoulder. The quality of the rotator cuff tissue is often compromised, being either atrophic or possessing tears of variable size and thickness.[35,90,119] In general, the longer the disease process persists, the more atrophic the deltoid musculature becomes and the greater the rotator cuff involvement is. Therefore, it should be anticipated that rehabilitation in rheumatoid patients will require more time.[127,131]

The functional results of nonconstrained total shoulder arthroplasty have been obtained by measuring the patient's ability to perform specific tasks of daily living. Most functional grading systems include activities that require varying degrees of forward elevation of the arm, with many being performed at waist level and fewer being performed at shoulder level or higher. Functional evaluation systems vary from report to report, making comparison between studies difficult. In the publications in which patients are separated into groups, based on diagnosis, functional scores are consistently higher in osteoarthritis than in rheumatoid arthritis. Functional scores tend to be lower for arthroplasty performed for old trauma.[9,10,127] The functional results of shoulder arthroplasty are dependent on both pain relief and gains in active range of motion. A patient who does not obtain gains in motion, but has less pain, is more inclined to use the extremity in functional activities within the range of motion available to him or her. This is especially so in patients who require only low-demand use of the extremity within limited arcs of motion. However, if significant gains in active range of motion are also achieved, the patient can participate in a broader spectrum of functional activities.

Our review of the literature yielded over 50 publications reporting the results of nonconstrained shoulder arthroplasty for glenohumeral arthritis and an intact or reparable cuff.[4–8,10,11,14,20,22,26,30,31,33–36,38,40,41,43,44,47,49,51,54,55,60,62,66,68–70,72,73,77,86,89,90,93,107–109,111,112,113,114,116–118] Reports of patients with rheumatoid or inflammatory arthritis were included, despite the fact that some of the included patients had dysfunctional or irreparable rotator cuffs. Only 10 of these reports had a minimum 5-year follow-up and, therefore, were considered at least midterm follow-up.[40,44,72,109,112,150,154,155,162,164] Only two studies had a minimum 10-year follow-up[44,156] and only five provided predicted survivorship.[26,44,112,154,155] While the data from these reports are not completely comparable, some conclusions can be made. First, shoulder arthroplasty results in predictable and durable pain relief. Second, functional results vary with rotator cuff integrity but are also predictable and durable in patients with an intact or reparable cuff. Third, rotator cuff disease continues after arthroplasty in many patients with inflammatory arthritis as evidenced by progressive proximal humeral migration.[150] Finally, survivorship varies with age and activity level but, in general, is 95% to 98% at 5 years, 93% to 97% at 10 years, 84% to 88% at 15 years, and 80% to 85% at 20 years.[44,112,155]

Plain film evaluation has shown the prevalence of glenoid radiolucent lines to range between 30% and 80% at various periods of follow-up.[10,24,35,127] However, the high prevalence of radiolucent lines has not correlated strongly with the presence of symptoms. As such, the significance of these lines remains a controversial subject. Of 46 reoperations performed at the Mayo Clinic between 1976 and 1988 addressing complications of nonconstrained total shoulder arthroplasty, 13 (28%) were for glenoid loosening.[39] As in the Mayo Clinic article, most articles report that the most common prosthetic-related complication of total shoulder arthroplasty is glenoid component loosening. The need for reoperation for this problem was summarized from 19 reports (1,413 procedures) by Brems.[23] At an average of 5 years of follow-up, radiolucent lines were present in 546 (38.6%) procedures, and in 42 (2.9%) reoperation was required for glenoid component loosening. The rate of radiolucent lines requiring revision glenoid surgery was 7.7% (42 of 546). Therefore, relatively few of these radiolucencies became symptomatic at midterm follow-up. The incidence of radiographic evidence of glenoid component loosening appears to increase with longer periods of follow-up.[27,163]

PREOPERATIVE EVALUATION

History

Patients with primary osteoarthritis typically present with an insidious onset of pain, which has been slowly progressive over past years. Progressive stiffness is often associated with the discomfort. As is the case in other arthritic joints, the

pain is often activity-related. Stiffness is most notable after a period of immobilization, and improves with gentle motion.

Patients' complaints will often relate to functional limitations such as difficulty internally rotating to reach their back pocket or fastening a brassiere. Questioning patients as to how their symptoms have interfered with their daily routines will provide insight into the degree of pain and disability. Understanding the patients' occupation, hobbies, and activity levels also helps gauge the impact of their disease. Documentation of treatments that have been or are being used also gives information about disease course and severity.

Awareness of the patients' profile of comorbidities is important not only for presurgical screening, but also as a means of evaluating what other conditions might limit activity and rehabilitation.

Patients suspected of having avascular necrosis should also be questioned regarding the onset, course, severity, and impact of their symptoms as this is valuable information in management decision making. Typically, these patients are younger than those presenting with osteoarthritis. Identification of risk factors can aid in determining the cause of osteonecrosis. Exposure to steroids, alcohol use/abuse, liver pathology, and personal or family history of coagulation disorders should be addressed. Careful questioning about pain in the contralateral shoulder and other joints at risk (hips, knees, ankles, etc.) is also important. Although only 3% of patients with osteonecrosis have multifocal involvement,[99,100] 80% of patients with multifocal osteonecrosis will have humeral head involvement.[99]

Physical Examination

A complete shoulder examination should be performed with particular attention to range of motion. Typically, both osteoarthritis and avascular necrosis cause progressive global loss of motion, with particular loss of external rotation. Postcapsulorrhaphy often has the most severe loss of external rotation. Any internal rotation contracture must be noted and documented as it dictates how the subscapularis release is managed at the time of surgery. Active and passive motion should be compared and the rotator cuff strength noted. This can sometimes be difficult to determine on physical examination alone, as there is often pain-related weakness. Painful crepitus is common. Tenderness to palpation over the acromioclavicular joint can indicate arthritis, which potentially can contribute to the symptom complex. This is an important finding to identify as it can also be addressed at the time of surgery if needed.

Imaging Studies

Plain radiographs are the single most important investigation required in the diagnosis of osteoarthritis and arthroplasty planning. A standard x-ray series (anteroposterior [AP], transscapular lateral, and axillary lateral) is typically performed, and each provides different information required for preoperative preparation. The AP x-ray, which often is done in internal and external rotation, allows assessment of bone quality, identification of inferior osteophytes, and diameter of the humeral canal. Preoperative templating is performed using the AP and axillary views. Also, although not universally reliable because of slight variations in angle-of-beam projection, evaluation of the acromiohumeral distance can suggest the presence of significant rotator cuff deficiency if the distance measures less than 7 mm. The axillary radiograph is useful in identifying glenoid version as well as the posterior glenoid wear and resultant posterior subluxation that is often associated with osteoarthritis (Fig. 22-7).

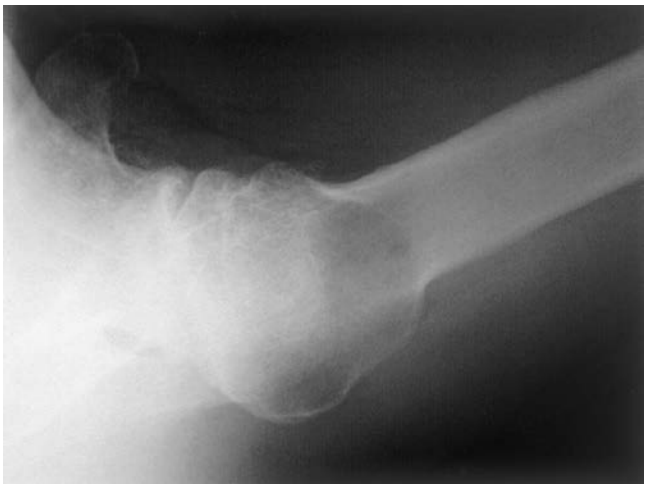


Figure 22-7 Axillary radiograph revealing posterior glenoid erosion associated with posterior subluxation that is commonly seen in osteoarthritis.

A computed tomography (CT) scan provides a more definitive assessment of glenoid bone stock and version. It also allows accurate determination of whether glenoid replacement is feasible and if bone grafting may be necessary (Fig. 22-8). One radiographic study of a series of patients with primary glenohumeral osteoarthritis who were awaiting shoulder arthroplasty found that 45% have posterior subluxation.[167] Average glenoid retroversion in this population was found to be 15.4 degrees (normal being 1 to 2 degrees of retroversion).[63] Humeral version can also be accurately determined from CT pictures of the humeral head and its relation to the transcondylar axis.[78] Since posterior glenoid erosion and posterior subluxation are common with severe internal rotation contracture, CT scanning is ordered in all patients with external rotation of 30 degrees or less.

Magnetic resonance imaging (MRI) may be useful in cases of suspected rotator cuff tear. In general, full-thickness rotator cuff tears are exceedingly uncommon in patients with osteoarthritis or osteonecrosis (5%). However, in patients who have had prior rotator cuff surgery or demonstrate a decreased acromiohumeral distance on plain radiography,

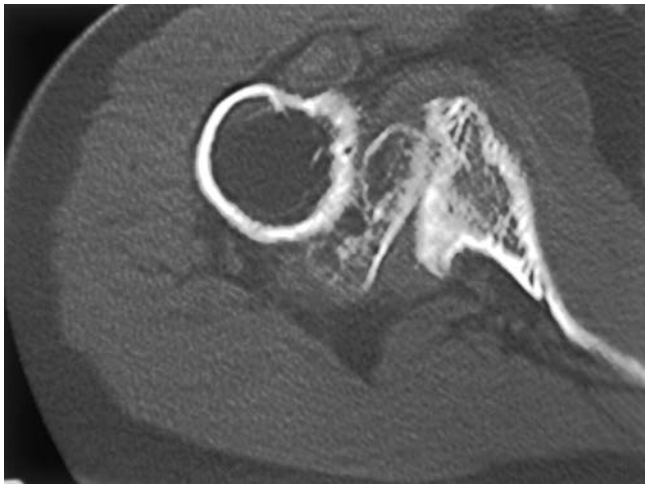


Figure 22-8 Computed tomographic scanning can be used to quantitate the amount of posterior glenoid deficiency and posterior subluxation.

rotator cuff tears may be more common. Under these circumstances, MRI scanning can reveal glenoid erosion, abnormal glenoid version, and full-thickness rotator cuff tears.

MRI is also useful in staging avascular necrosis. Moreover, avascular necrosis in certain disease states, such as renal failure, may be associated with rotator cuff deficiency. Therefore, MRI scanning is performed frequently in patients with early avascular necrosis, particularly when it is associated with renal failure.

Implant Choices

When considering the humeral side, the choice of implants generally is between traditional stemmed humeral replacement and humeral head resurfacing. The advantages of traditional stemmed implants include easier glenoid exposure, greater familiarity, and a larger and longer experience. However, replacement of the humeral head with a stemmed implant requires greater bone removal, potentially more extensive exposure, and potentially a greater number of implant choices to recreate normal humeral anatomy. Humeral head resurfacing without a stemmed implant is a very reasonable option in patients with adequate bone stock, a concentric or minimally diseased glenoid, and a need for bone preservation (i.e., a young patient with high likelihood for revision). Although glenoid resurfacing is possible without removing the head, it is more difficult than with the head removed. The results of humeral head resurfacing are promising but sparse. Levy and Copeland's review of their series of resurfacing arthroplasties revealed that those patients with primary osteoarthritis had the best outcome, and that only 8% required revision during the 5- to 10-year follow-up.[107]

Stemmed humeral head replacements are the most popular humeral implants for shoulder replacement, particularly when the glenoid is also being resurfaced. There are many stemmed humeral implants available and none has been shown to be superior to others with regard to clinical outcome. Principles that have gained in popularity and have some basis in scientific evidence include humeral head modularity, humeral head offset, and anatomic reconstruction.[18,56,80,136]

There is a large variety of glenoid replacement prostheses also available for shoulder arthroplasty. These include all-polyethylene designs, metal-backed designs, and hybrid designs with metal peg sleeves but no metal backing. There is also variability with regard to articular conformity and constraint. There is some evidence to show that arthroplasties characterized by nonconforming radii of curvature yield more physiologic translations and exhibit lower loosening scores than conforming designs.[88,168] However, more data are necessary to confirm this. At the very least, the surgeon should be familiar with the conformity characteristics of the glenoid being used so that excessive contact stresses are not created.

SURGICAL APPROACHES

This section will discuss the preferred surgical approach, anesthesia, patient positioning, surgical technique, and implant considerations for shoulder arthroplasty, with particular emphasis on considerations specific to osteoarthritis, avascular necrosis, and other conditions characterized by an intact or reparable rotator cuff.

Preferred Approach

Shoulder arthroplasty is a demanding surgical procedure, the outcome of which is at least partially the result of balancing the concepts of surgical exposure and preservation of soft tissues. Several surgical exposures have been described for shoulder replacement, including superior acromial splitting, superior deltoid reflecting, anterior deltoid reflecting (with and without clavicular osteotomy), posterolateral posterior cuff reflecting, superolateral deltoid reflecting, and anterior deltopectoral deltoid-sparing approaches.[46,65,87,95,98,124,127,141] The deltoid-reflecting approaches offer superior exposure at the expense of potential deltoid morbidity or osteotomy nonunion. Fortunately, in cases of primary osteoarthritis and nontraumatic avascular necrosis, virtually every reconstructive situation encountered can be adequately performed through the deltopectoral approach described and popularized by Neer, without detaching the deltoid origin or insertion. This is even true in cases of osteoarthritis requiring posterior glenoid bone grafting. Therefore, our preferred approach for primary osteoarthritis, nontraumatic avascular necrosis, and other conditions with an intact or reparable cuff is an extended deltopectoral approach.[124,127]

Disease-Specific Considerations

Preoperative Planning

Prior to surgery, several important planning steps should be undertaken to ensure smooth execution of the surgical plan. Any medical comorbidities should be optimally treated, and medical clearance, if indicated, should be obtained. In addition, medical consultation to assist in perioperative medical management of conditions such as sickle cell anemia, renal failure, and diabetes mellitus should be sought. Orders should be written and arrangements made for perioperative antibiotic prophylaxis. A first-generation cephalosporin is generally the agent of choice as it covers the usual skin contaminants.[45,53,74,96,137,146,157] Vancomycin or clindamycin is commonly used in the face of a penicillin allergy.

Controversy exists with regard to the usefulness of preoperative templating of radiographs. With the popularity of modular systems and offset humeral heads, templating affords the surgeon a reasonably accurate assessment of the size of the implants needed at surgery and whether or not an offset or centered humeral head will be necessary. In addition, having a good idea of the size of the humeral canal prior to surgery may prevent inadvertent perforation because of a misaligned reamer. For instance, if the preoperative templating revealed a 12-mm humeral canal but the surgeon is having difficulty getting an 8-mm reamer to pass into the canal, it is likely that the entry point is inaccurate and the reamer is in valgus, or more likely, varus.

Anesthesia and Patient Positioning

Anesthetic options for shoulder arthroplasty include general anesthesia, regional anesthesia (i.e., interscalene block), or a combination of general anesthesia and an interscalene block. Each of these choices has advantages and disadvantages; the choice of anesthetic method is always left to the patient after a thorough discussion with the anesthesiologist, who usually obtains a separate informed consent for the anesthetic portion of the procedure. Interscalene block, in the hands of an experienced anesthesiologist, is safe and provides muscle relaxation as well as intra- and postoperative pain relief to facilitate early postoperative motion.[28,158] The block also reduces the depth of general anesthetic required (when used in combination with general anesthesia), thus diminishing anesthesia-related morbidity.[28,158] However, the use of an interscalene block may result in added preoperative induction time and increased risk of neurologic, pulmonary, and vascular complications.[16,138,170,171] Using a combination of general anesthesia and an interscalene block combines the advantages of both methods, namely, excluding interactions with the patient during surgery and prolonged postoperative pain control. For these reasons, a combination of anesthetic methods is often used.

Following induction of anesthesia, the patient is placed in the beach-chair position, with the torso at approximately 45 degrees to the horizontal. The head and body should be stabilized and all pressure points padded appropriately. The affected arm should be free enough to be placed into a fully extended and adducted position (to allow dislocation of the humeral head) without restriction by the bed or positioning devices (Fig. 22-9). Once the patient has been positioned as desired, a final review of passive range of motion should be noted (especially external rotation) with the patient under anesthesia. The entire shoulder and upper extremity is sterilely prepped, and an impermeable stocking is placed over the hand. Draping should allow access to the medial clavicle proximally and to the wrist distally.

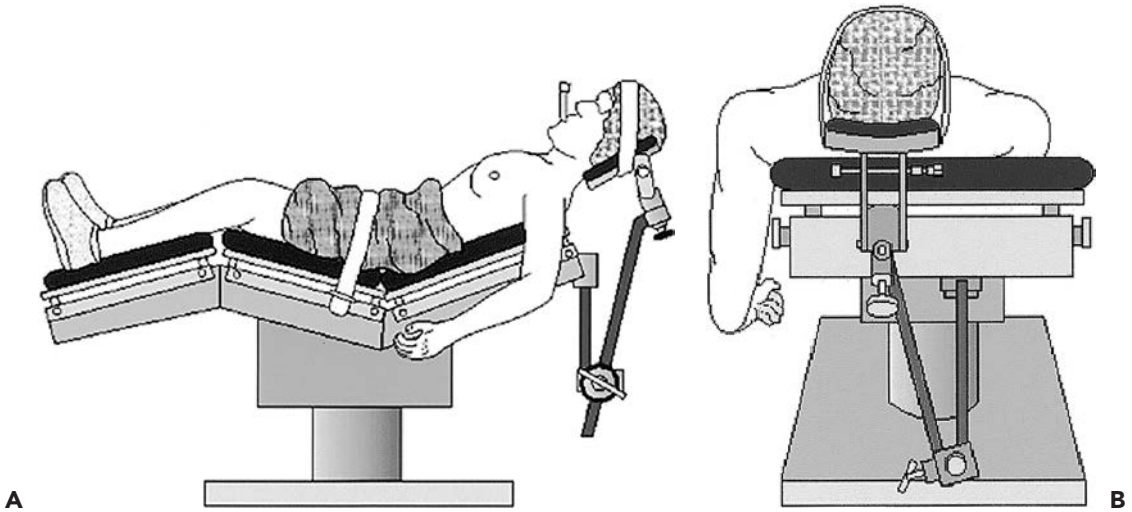


Figure 22-9 Total shoulder arthroplasty requires that the patient be placed in a beach-chair position **(A)** with the operative shoulder completely off the edge of the table to allow adduction and extension **(B).**

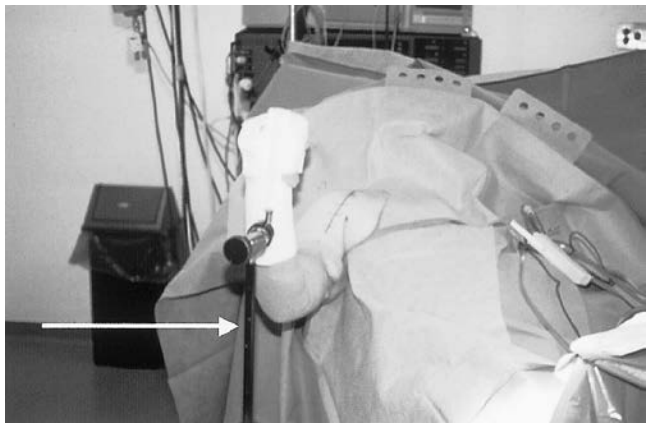


Figure 22-10 A sterile, mechanical arm-holding device makes arm positioning very convenient. (From McConnell Arm Holder, Ft. Worth, TX.)

Several portions of a shoulder arthroplasty procedure require the arm to be held or supported. This can be done using an assistant, a padded Mayo stand, or a mechanical arm-holding device. The latter can be very useful, as it consists of an articulated extension that is sterile and can attach to the arm via a sterile, disposable arm sleeve. This construct attaches to a universal ball joint that is suspended from the operating table. A foot peddle releases the ball joint and allows optimal arm positioning; when this position has been reached, the peddle is released and the arm stays in the previously selected position (Fig. 22-10). Even with this articulated arm holder, shoulder arthroplasty is easiest when performed with a knowledgeable assistant and scrub technician.

Incision and Superficial Exposure

The deltopectoral approach begins with a skin incision approximately 10 to 15 cm long, beginning at the clavicle just superior and medial to the tip of the coracoid and extending distally along the estimated location of the deltopectoral groove. When the arm is placed in approximately 30 to 40 degrees of abduction, this should be a straight incision (Fig. 22-11).

Subcutaneous fat is split in line with the incision and the cephalic vein is identified, serving as a landmark for the deltopectoral groove. The cephalic vein is a large draining tributary for the upper extremity. Therefore, its preservation seems intuitive and is supported by most shoulder surgeons. This is particularly true in patients with potential venous outflow difficulties such as those with a history of ipsilateral radical mastectomy or irradiation.

There is less consensus, however, with regard to which direction the vein should be retracted to ensure its preservation. Two general schools of thought exist. The first school bases its logic on the fact that most of the contributing branches to the cephalic vein in the brachium enter from the deltoid side. Therefore, the vein should be retracted laterally, with the deltoid. The second school correctly observes that the vein crosses the surgical field from lateral to medial in the superior portion of the wound to

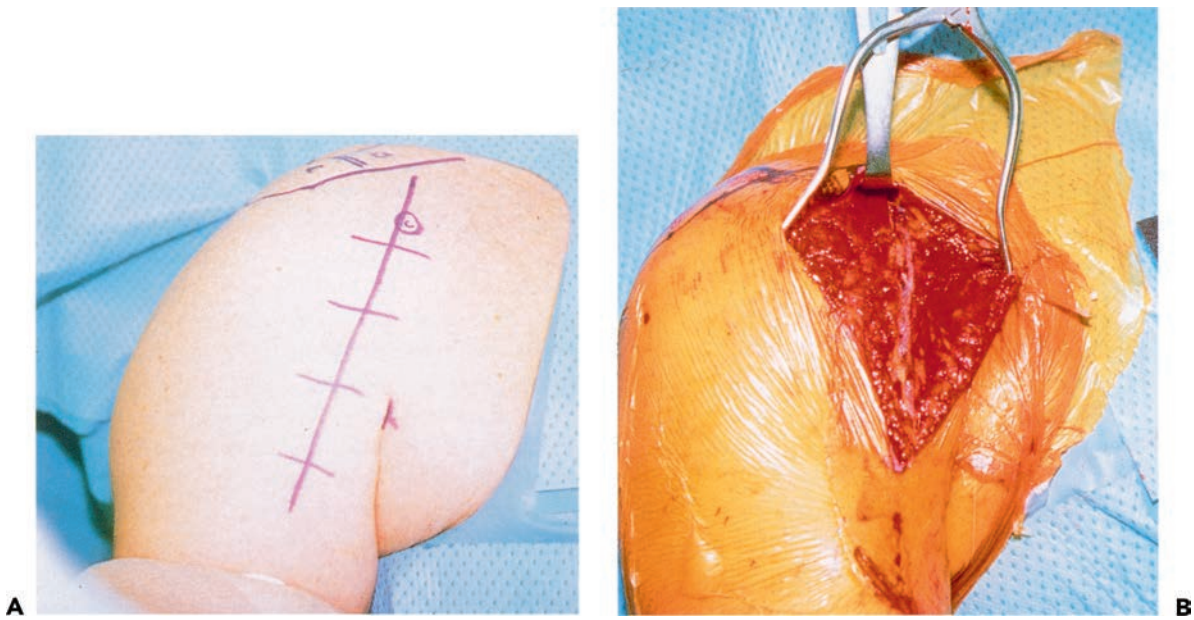


Figure 22-11 **(A)** The skin incision for a routine total shoulder arthroplasty is centered over the deltopectoral interval and the tip of the coracoid. The incision begins near the lateral third of the clavicle and extends near the deltoid tuberosity. **(B)** The cephalic vein is exposed and may be dissected medially with the pectoralis major or laterally with the deltoid. The latter technique is generally preferred.

enter the clavipectoral fascia. This course puts the vein at risk when it is retracted laterally. Consequently, disciples of the second school advocate taking the vein medially with the pectoralis major to prevent excessive traction and subsequent rupture of the vein. There is a certain amount of truth in both schools of logic. However, if one exercises atraumatic soft tissue handling and protects the vein during periods of vigorous retraction with a moist sponge, it is usually possible to retract the vein laterally and preserve it throughout the procedure (see Fig. 22-11).

The first step in gaining adequate exposure is freeing the deltoid from the pectoralis major, starting at the clavicle and extending to the deltoid insertion on the humeral shaft. The deltoid is then retracted laterally and the interval between the surgical neck of the humerus and the overlying deltoid is bluntly dissected using a Cobb elevator. Care is taken to keep the tip of the elevator on bone because the axillary nerve traverses the deep surface of the deltoid in this region. A sponge can then be used to sweep away any deltoid adhesions from the underlying humerus all the way to the deltoid tuberosity. This sponge can be left in place during the procedure, between the humerus and distal deltoid insertion, to tamponade any small bleeding vessels.

The tip of the coracoid process is next identified and the overlying pectoralis major muscle belly is separated from the conjoined tendon of the short head of the biceps and the coracobrachialis. The pectoralis major muscle belly is retracted anteriorly, away from the underlying conjoined tendon, and the tendinous insertion on the distal portion of the lateral lip of the bicipital groove is identified. The upper 1 cm of the pectoralis insertion may then be released from the humerus, if necessary, to improve exposure of the inferior portion of the humeral head and to aid in correction of any internal rotation contracture. A self-retaining retractor, such as the Koebel retractor, is placed with one limb under the deltoid laterally and one limb under the pectoralis major medially. With the deltoid and pectoralis major retracted, the deep exposure can be carried out.

Deep Exposure

The lateral edge of the conjoined tendon is identified. The associated muscle bellies of the short head of the biceps and coracobrachialis usually extend more lateral than the conjoined tendon itself. The clavipectoral fascia is incised at the most lateral extent of the conjoined tendon and associated muscles. This incision is carried distally to the level of the inferiormost extent of the subscapularis and proximally to the coracoacromial ligament. Digital palpation is used to identify the axillary nerve as it courses along the superficial surface of the subscapularis to reach the quadrilateral space at the inferior aspect of the glenohumeral joint. The musculocutaneous nerve enters the deep aspect of the conjoined tendon a variable distance from the tip of the coracoid.[58] Therefore, it cannot always be palpated within the surgical field. One should attempt to palpate it, however, because it can enter the conjoined tendon within 1.5 to 2.0 cm from the tip of the coracoid. In this position, the nerve could be injured while retracting the conjoined tendon.

The incision in the clavipectoral fascia stops superiorly at the anterior border of the coracoacromial ligament. This ligament is an important restraint to anterosuperior subluxation, especially in patients with large or massive rotator cuff tears.[59,102,151,174] Rotator cuff tears in osteoarthritis of the glenohumeral joint or in avascular necrosis are uncommon, usually small, and reparable.[48,81,124,127,132] Therefore, in theory, the coracoacromial ligament may be incised, excised, or partially excised in these cases to improve visualization of the superior glenoid. However, exposure is almost always adequate without coracoacromial ligament sacrifice. Moreover, rotator cuff tears can develop after shoulder arthroplasty. Therefore, the coracoacromial ligament can be completely preserved during shoulder arthroplasty for osteoarthritis and avascular necrosis, even if the rotator cuff is intact. In patients with inflammatory arthritis, even if the cuff is intact, the coracoacromial ligament is preserved.

Adequate visualization of the humeral head during arthroplasty requires that the humerus be completely delivered into the wound. Several important steps are necessary for this to occur. One of the earliest of these steps is clearance of any and all adhesions between the acromion and underlying rotator cuff. This is done by passing a Cobb elevator between the leading edge of the coracoacromial ligament and the supraspinatus tendon. Sharp dissection of this interval may be required in patients who have a history of prior surgery. The acromiohumeral interface can be completely freed of adhesions by sweeping the elevator over the top of the humerus. At this point, a reverse retractor, such as a modified Taylor or Brown deltoid retractor, can be placed, superior to the lateral limb of the Koebel retractor. The medial limb of the Koebel retractor is moved from the superficial side of the conjoined tendon to the deep surface. If the musculocutaneous nerve has been found to have a high penetration into the conjoined tendon, excessive pressure on the conjoined tendon should be avoided. With the conjoined tendon retracted medially and the deltoid retracted laterally, the humerus is externally rotated approximately 30 degrees and the anterior humeral circumflex vessels are clamped, cut, and tied off or coagulated.

Subscapularis and Capsular Incision

Subscapularis management is predicated on the amount of internal rotation contracture present. The goal is to restore optimal subscapularis length with the assumption that optimal function will follow. In almost all cases of primary osteoarthritis, significant loss of external rotation exists. Avascular necrosis is also characterized by joint stiffness with preferential loss of external rotation, but to a lesser degree. Postcapsulorrhaphy arthropathy often exhibits the

greatest degree of internal rotation contracture, especially if the prior surgical procedure involved subscapularis shortening (i.e., Magnuson-Stack or Putti-Platt).

When the internal rotation contracture is mild (passive external rotation is greater than 30 degrees with the arm at the side, under anesthesia), the subscapularis is incised intratendinously and repaired anatomically. Alternatively, the subscapularis can be reflected with the lesser tuberosity with subsequent bone-to-bone repair.[67] With moderate degrees of internal rotation contracture (passive external rotation of 30 degrees but not less than −30 degrees), the subscapularis is removed from the lesser tuberosity with maximum length and is advanced medially and repaired to the cut surface of the humeral osteotomy. In the most severe cases of internal rotation contracture (passive external rotation of less than −30 degrees), a coronal z-plasty of the subscapularis and capsule is performed (Fig. 22-12).

When intratendinous incision of the subscapularis is indicated, the incision is made approximately 2 cm medial to the lesser tuberosity insertion site. It starts at the superior border of the subscapularis tendon, is taken full thickness through both the tendon and the underlying capsule, and stops at the fleshy inferior one-third of the subscapularis.[92] At the superior border of the subscapularis tendon incision, the incision is taken superomedially, across the rotator interval, to the superior aspect of the base of the coracoid process. At the inferior extent of the subscapularis and capsular incision, a blunt elevator is used to develop the interval between the intact, fleshy portion of the subscapularis and the underlying anteroinferior capsule. The subscapularis is retracted distally, along with the more superficial axillary nerve. The remainder of the subscapularis and anterior capsule is released from the humerus.

An alternative approach involves osteotomy of the lesser tuberosity and reflection of the entire subscapularis.[67] Tenodesis or tenotomy of the biceps is routinely performed with lesser tuberosity osteotomy. As with the transtendinous approach, the interval between the inferior one-third of the

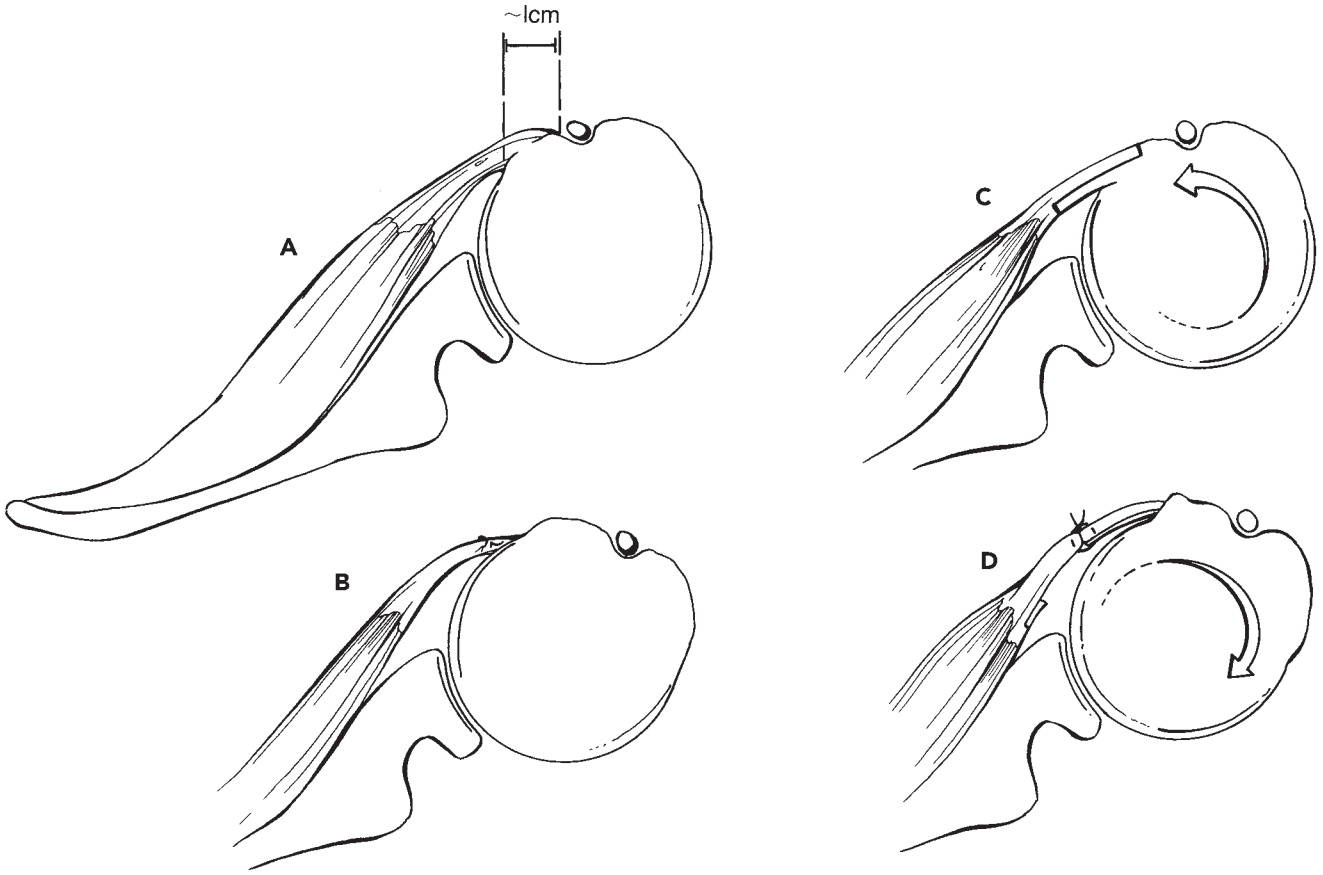


Figure 22-12 In cases with −30 degrees < external rotation <30 degrees, the subscapularis is released from the lesser tuberosity **(A)** and advanced to the osteotomy site at closure **(B),** thereby gaining approximately 1 cm of length and 30 degrees of external rotation. When preoperative external rotation <−30 degrees, a z-lengthening is performed by releasing the subscapularis from the lesser tuberosity and the capsule from the glenoid **(C),** and sliding the flaps on one another to gain appropriate length at closure **(D).** (From Iannotti JP, Schenk T. Prosthetic arthroplasty for glenohumeral arthritis and an intact or reparable rotator cuff: indications, techniques, and results. In: Iannotti JP, Williams GR, eds. *Disorders of the shoulder: Diagnosis and management*. Philadelphia: Lippincott Williams & Wilkins, 1999.)

subscapularis and the capsule can be identified, allowing complete release of the inferior capsule from the humeral neck. The potential advantages of this approach are preservation of the entire subscapularis attachment to bone, bone-to-bone healing, the ability to identify postoperative subscapularis detachment with a radiograph, and better postoperative subscapularis function.[67] The potential disadvantages include the possibility of intraoperative damage to the anterior humeral metaphysis during posterior humeral retraction and lesser tuberosity nonunion. It is tempting to consider a lesser tuberosity osteotomy in all cases, even those with substantial internal rotation contracture. However, the contribution of the subscapularis to the internal rotation contracture and the principle of subscapularis lengthening may limit the usefulness of this technique in some cases.

In cases of moderate internal rotation contracture, subscapularis advancement is indicated. The subscapularis is incised at its most lateral extent and is elevated along with the humeral capsule. At the superior subscapularis border, the rotator interval capsule is incised superomedially to the superior border of the base of the coracoid process. Inferiorly, the subscapularis and capsule are released in a single layer until the inferior one-third of the subscapularis is encountered, which is also released from the humeral insertion. This portion of the subscapularis is muscular, even at its humeral attachment site, and the interval between it and the inferior capsule can be easily identified. A blunt elevator is used to develop this interval. An electrocautery or knife is used to release the remaining subscapularis and anterior capsule.

With any of the above-mentioned methods of subscapularis and capsular release, the inferior capsule must be released to or past the 6 o'clock position. At the anteroinferior aspect of the humeral head, electrocautery is used to raise a periosteal/capsular flap that includes the upper 1 cm of the latissimus dorsi, the inferior periosteum, and the anteroinferior capsule to at least the 6 o'clock position. This can be done safely by progressively externally rotating the humerus and using a blunt Homans retractor between the inferiormost portion of the humeral head and the overlying capsule. As the incision gets close to the 6 o'clock position, the electrocautery is switched to a surgical knife to avoid inadvertent electrical injury to the axillary nerve by conduction through the metallic retractor. This inferior soft tissue release is a critical maneuver to allow not only adequate delivery of the humerus into the wound, but also adequate glenoid exposure.

Rarely is subscapularis z-lengthening required. In all instances but the most severe cases of internal rotation contracture, subscapularis advancement to the cut surface of the osteotomy is adequate. However, when z-lengthening of the subscapularis is required, the subscapularis must be elevated separately from the anterior capsule. The subscapularis is released from its attachment as far laterally as possible. As the tendon is being reflected medially, the interval between the capsule and subscapularis can be determined distally, where the inferior third of the subscapularis attachment is muscular rather than tendinous. This interval is bluntly dissected to provide the appropriate interval for dissection. The subscapularis tendon is then completely detached and reflected medially. A small portion of the tendon and muscle can be left behind on the anterior capsule for reinforcement.

With the subscapularis released and retracted medially, the capsule is released from its glenoid attachment, starting superiorly and extending to the 6 o'clock position on the glenoid. With the axillary nerve retracted, the capsule is then incised in a medial–lateral direction from the inferior glenoid margin to the humerus. This then creates a laterally based capsular flap that can be used to lengthen the subscapularis during closure. The remainder of the inferior capsule is released from the inferior humerus along with a small portion of latissimus dorsi and inferior humeral periosteum as described above.

The humerus is delivered into the wound using simultaneous adduction, external rotation, and extension. All humeral osteophytes are removed using a combination of an osteotome and rongeur. This allows the surgeon to estimate the location of the anatomic neck. Details about the humeral osteotomy and preparation are included later in this chapter.

Glenoid Exposure

Obtaining adequate glenoid exposure may be the most difficult part of total shoulder arthroplasty. Even if the glenoid is not going to be resurfaced, virtually every soft tissue release required for glenoid exposure should still be carried out to maximize postoperative range of motion following hemiarthroplasty. Therefore, the integral maneuvers for total shoulder arthroplasty[32] and hemiarthroplasty are the same.

There are five basic requirements for adequate glenoid exposure, all of which are within the control of the surgeon or surgical team: adequate muscular paralysis, adequate humeral bone resection, proper arm positioning, appropriate soft tissue contracture releases, and proper glenoid retractors. Each of these requirements is important. However, their importance is even more magnified in certain patients. Glenoid exposure is easiest in thin, small patients in whom there is little tissue between the skin and the glenoid. In more massive patients, particularly muscular males, glenoid exposure can be extremely challenging and all of the above-mentioned "requirements" take on added importance.

The easiest factor to control is muscular paralysis. If an interscalene block has been used, this alone can produce excellent muscular relaxation. However, in cases where a general anesthetic alone has been used or in cases where muscular relaxation from an interscalene block is not sufficient, muscular paralysis can be obtained by the anesthesiologist using various paralyzing agents. The degree of paralysis can be followed using a twitch monitor.

Failure to resect enough humeral bone may also lead to difficulties in glenoid exposure. In cases where a traditional stemmed humeral implant has been chosen, the line of humeral head resection should approximate the original anatomic neck. The rotator cuff attachment marks the most lateral or distal extent of potential bone resection. It is important to note that the cuff attachment is closer to the anatomic neck in the region of the supraspinatus insertion than it is in the infraspinatus and teres minor regions. Therefore, even if the most superior extent of the osteotomy is flush with the supraspinatus insertion, there will still be a small amount of bone remaining between the osteotomy and the infraspinatus and teres minor insertion sites. There is usually very little bone (2 to 3 mm at most) between the normal anatomic neck and the anteriormost fibers of the supraspinatus. The osteotomy should be made so that it exits at or very close to (within 5 mm) the anterior supraspinatus insertion site. Even 5 mm of extra bone can make a difference in glenoid exposure.

The persistence of humeral osteophytes is another source of inadequate bone resection in cases of osteoarthritis. The inferior humeral osteophyte is the largest and most obvious of the humeral osteophytes. It should be removed entirely before attempted glenoid exposure and, preferably, before humeral resection. However, osteophyte formation on the humeral head occurs circumferentially. Any bony structure that makes the humerus wider than normal will make posterior displacement of enough humerus to adequately expose the glenoid difficult. Therefore, humeral osteophytes should be excised circumferentially before attempted glenoid exposure. Care should be taken not to remove normal anterior cortex along with the anterior osteophytes, because this may lead to tuberosity fracture from the humeral head retractor.

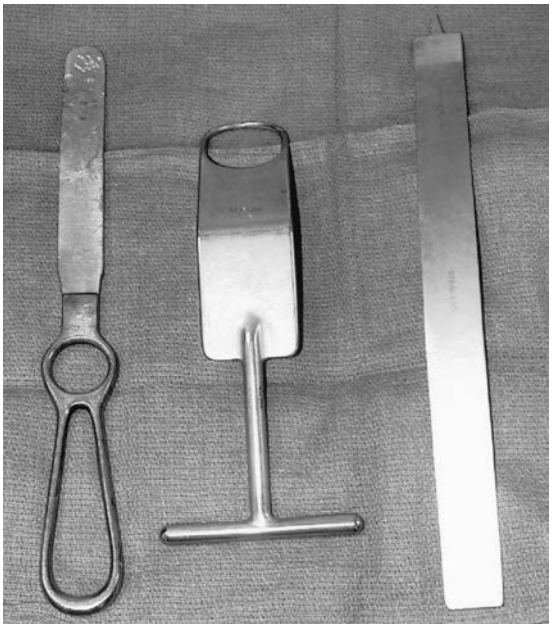
During glenoid exposure, the arm should be positioned such that the intact soft tissues will allow maximum posterior humeral displacement. Assuming that the anterior and inferior capsule has been adequately released or excised and that there are no other remaining soft tissue tethers, only the posterior and superior capsule remains. These two capsular regions are made slack by a combination of abduction, extension, and external rotation. Therefore, this is the preferred position for the arm during glenoid exposure. The arm can be held in this position using an assistant, a padded Mayo stand, or a mechanical arm-holding device.

Soft tissue releases are the most important steps in glenoid exposure. The anterior and inferior capsular regions have been released from the humeral side as far posteriorly as the 6 o'clock position during humeral exposure and preparation. The humerus is retracted posteriorly with a humeral head retractor. A small, sharp rake is used to pull the lateral aspect of the subscapularis and capsule anteriorly. A capsular incision is then made, starting at the superior glenoid near the base of the coracoid and extending to the 5 o'clock position (right shoulder). The incision is made at the capsular–labral junction and parallels the anterior glenoid rim. A periosteal elevator is passed through the capsulotomy, between the anterior scapular neck and the subscapularis muscle belly. A blunt elevator is then passed between the anteroinferior capsule at the 5:30 position (right shoulder) and the subscapularis muscle. The elevator is then replaced with a blunt Homans retractor, which is used to retract the subscapularis and protect the axillary nerve. The anteroinferior capsule is excised (if it appears thickened and pathologic) or released past the 6 o'clock position.

The biceps and superior capsule may also prevent posterior humeral displacement and should be palpated during attempted humeral retraction. If glenoid exposure is inadequate and the long head of the biceps becomes taught with attempted posterior humeral retraction, it is likely to be scarred or tenodesed within the bicipital groove. This is not uncommon in patients who have had prior rotator cuff surgery. Under these circumstances, the biceps should be released from the supraglenoid tubercle and the intraarticular portion should be excised. The remaining biceps can be tagged with a suture and incorporated into the rotator interval repair at the completion of the procedure. If after biceps release the superior capsule still appears to be taught, it can be released from the glenoid margin.

The last step in soft tissue releasing is labral excision. The labrum is excised circumferentially, except superiorly in the region of the biceps insertion if the biceps has not required release and subsequent tenodesis. Posterior labral excision accomplishes at least a partial release of the posterior capsule, which may aid in maximizing the amount of posterior humeral displacement. In cases of severe posterior glenoid bone loss and posterior subluxation, the posterior capsule can be spared until it becomes obvious that excessive posterior translation of the trial prosthesis does not occur. Excessive posterior translation of the prosthesis because of posterior capsular insufficiency, however, is uncommon. When there is preoperative humeral head subluxation of more than 25% of the humeral head diameter, a posterior capsulorrhaphy may be required to balance the soft tissues and maintain stability of the humeral head.

The choice of retractors and their placement are key elements in attaining good glenoid exposure. The basic retractors include a humeral head retractor (i.e., a Fukuda ring), a large Darrach retractor, and a single pronged Bankart retractor or reverse Homans retractor. The Fukuda ring retractor is placed between the humerus and glenoid, with the ring cupping the posterior glenoid rim and the T-handle of the instrument retracting the humerus posteriorly. The large Darrach retractor is placed through the anterior capsulotomy along the anterior glenoid neck, deep to the subscapularis. It is used to retract the anterior soft tissues anteriorly. When the Fukuda is being levered posteriorly and the Darrach is being levered anteriorly, there is potential for axillary nerve traction. Therefore, when using either one of



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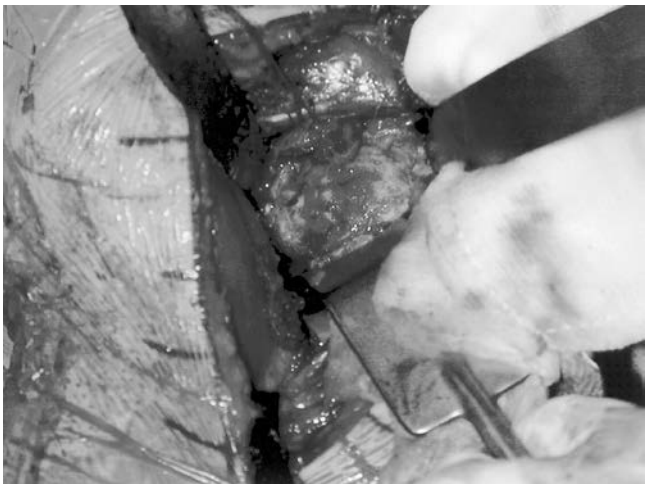


B

Figure 22-13 Retractors for glenoid exposure may include a Fukuda ring, large Darrach, and a single-pronged Bankart retractor **(A).** When the Fukuda ring is placed into the joint to retract the humeral posteriorly, when the large Darrach is placed on the anterior scapular neck to retract the anterior soft tissues, and when the single-pronged Bankart retractor is placed on the posterosuperior glenoid rim to retract the deltoid and posterosuperior cuff, excellent glenoid exposure is usually obtained **(B).**

these retractors vigorously, the other should be reciprocally relaxed. The final basic retractor is the single pronged Bankart retractor, which is placed posterosuperiorly, under the long head of the biceps (or in its place if it was released). A moist, folded sponge is placed between the deltoid and the Fukuda ring and Bankart retractor to protect the cephalic vein and the deltoid muscle fibers. These three retractors (the Darrach anteriorly, the Fukuda ring posteriorly, and the single pronged Bankart posterosuperiorly) are usually adequate for excellent glenoid exposure (Fig. 22-13). Occasionally, it is useful to place a fourth retractor (i.e., a reverse Homans) inferiorly. However, this is not routinely necessary. Having a variety of humeral head retractors, of different types and sizes, may also be helpful.

The two most challenging steps in glenoid preparation from the standpoint of exposure are concentric reaming of the glenoid surface and placement of drill holes for pegged glenoid designs, particularly if one of the drill holes is posterior and inferior on the glenoid surface. Concentric glenoid reamers for many implant systems are circular or make up some portion of a sphere. Often, the humeral head retractor (i.e., the Fukuda ring) interferes with seating of the reamer on the surface of the glenoid. If difficulty arises from the fact that the ring is too small to accommodate the reamer, a larger ring can be used. If this still is not satisfactory, removing the humeral head retractor entirely and using the shaft of the reamer to displace the humerus posteriorly to expose the glenoid works well. This method is used in over half of the cases in our practice. This can be done whether the reamer shaft is straight or angled, as some systems have angled reamers available. We prefer the straight reamer shaft in all cases. In addition, the reamer can be undersized by one size if the radius of curvature of the reamers in the system are the same between the reamer sizes.

Gaining adequate glenoid exposure to place posteroinferior glenoid drill holes for glenoid component pegs can be difficult. Four steps can be taken to potentially improve the situation. First, the surgeon should confirm that the five steps mentioned above have been accomplished, namely adequate muscular relaxation, adequate humeral bone resection, optimal arm positioning, complete soft tissue release, and appropriate retractor placement. Second, the Fukuda ring can temporarily be rotated so that the inferior portion is rotated posteriorly and laterally, away from the posteroinferior glenoid. Third, a blunt Homans retractor can be placed between the humerus and glenoid and used as a humeral head retractor, either with or instead of the Fukuda. We prefer to place the blunt Homans through the ring of the Fukuda so that, when the tip of the blunt Homans passes posterior to the posterior glenoid rim, the transverse component of the Fukuda ring is between the levering tip of the blunt Homans and the posterior glenoid bone (Fig. 22-14). Fourth, the humeral broach, which should be left in the humeral canal to protect against humeral injury, can be seated more deeply into the humerus so that less of the collar protrudes. One must allow enough of the collar to sit

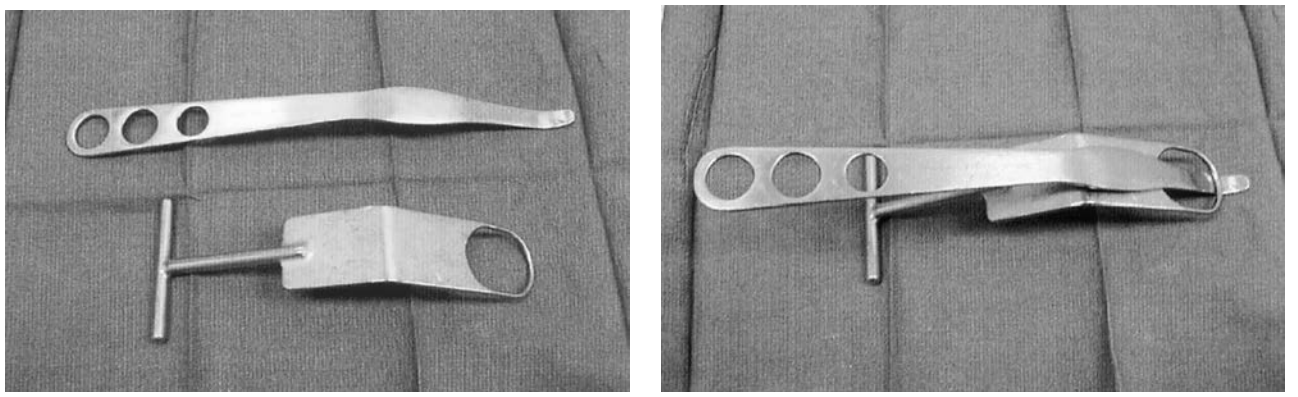


Figure 22-14 When added posterior glenoid exposure is needed, it may be accomplished by placing a blunt Homans retractor through the hole in the Fukuda ring **(A)** and onto the posterior glenoid **(B).** When the blunt Homans is levered posteriorly onto the surface of the Fukuda ring, posterior glenoid exposure is improved. Care must be taken to relax the anterior retractor simultaneous to this added posterior humeral translation to minimize axillary nerve traction.

above the humeral surface to accomplish complete seating and locking of the taper on the real implant, however. Using a combination of these four steps, one should be successful in implanting a glenoid component in all cases, assuming adequate bone exists to accept one.

Closure

The most important component of wound closure is a secure subscapularis repair. If the subscapularis was incised intratendinously, it is repaired anatomically, tendon to tendon. Even if the underlying anteroinferior capsule were pathologic and therefore excised, a portion is left attached to the deep surface of the retracted subscapularis tendon to provide a reinforcing layer to the subscapularis repair. A grasping-type suture, such as a modified Kessler, is placed in both the lateral tendon stump and the medial reflected tendon using heavy, nonabsorbable suture. Two of these sutures are placed (one superior and one inferior) within each side of the tenotomy. The sutures are then tied to one another so that the suture knot lies between the approximated tendon edges. In this way, tendon-grasping sutures are present on both sides of the repair site. Alternatively, if the subscapularis was reflected with a lesser tuberosity osteotomy, the osteotomy is approximated using two tension-band sutures that pass through the proximal humerus near and within the bicipital groove and then around the humeral stem. The two free ends of these sutures are passed through the subscapularis tendon at the tendon–bone interface.

The rotator interval is then closed laterally with an additional heavy, nonabsorbable suture, incorporating the biceps if it has been released.

If the subscapularis was released from its insertion at the lesser tuberosity because of the need for tendon lengthening, it is repaired to the anterior edge of the humeral osteotomy. This is done by placing three drill holes along the anterior humeral metaphysis, approximately 1 to 1.5 cm from the osteotomy edge. They are aligned from superior to inferior, starting at the superior margin of the lesser tuberosity, with at least 1 cm between them. Three heavy, nonabsorbable sutures are placed into the retracted edge of the subscapularis, which also contains the most lateral extent of the anterior capsule as reinforcement, using a modified Mason-Allen configuration. The most superior suture is placed approximately 1.5 to 2.0 cm inferior to the most superior edge of the tendon. This leaves adequate tendon tissue above the most superior reattaching suture to close the rotator interval laterally. The sutures are then passed through the anterior drill holes and tied over anterior bony bridges (Fig. 22-15). The rotator interval is then closed laterally using an additional

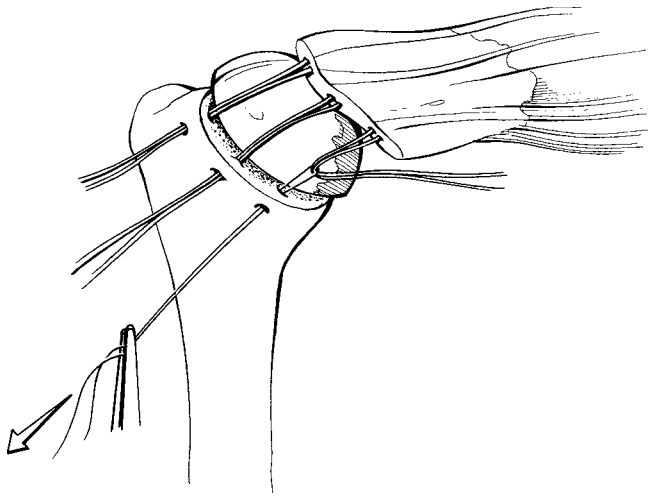


Figure 22-15 The technique for repair of the subscapularis will be dependent on the technique by which the tendon was excised. If the tendon was removed by subperiosteal dissection from the lesser tuberosity, the tendon is repaired to the osteotomy surface by transosseous sutures using 1-mm Dacron sutures. The sutures are passed through the tendon using a modified Mason-Allen or Kessler technique.

heavy, nonabsorbable suture, with incorporation of the biceps if it has been released.

The subscapularis repair is performed with a z-lengthening in cases of severe internal rotation contracture. There are two flaps of anterior soft tissue that are used to perform this repair: the medially based subscapularis tendon and the laterally based capsular flap that may contain some remaining subscapularis tendon and muscle fibers. The laterally based flap is brought deep to the medial flap and horizontally based mattress sutures are passed through the two superimposed flaps from deep to superficial. The sutures are passed so that external rotation of 45 degrees with the arm at the side is permitted without untoward tension on the sutures. The lateralmost extent of the medially based subscapularis tendon is then sutured to the underlying lateral capsular flap, more lateral than the previously placed sutures. The rotator interval is then closed laterally. All sutures are heavy, nonabsorbable sutures. The biceps is incorporated into the lateral rotator interval repair if it has been released.

The deltopectoral interval is sutured closed over a closed suction drainage system using absorbable suture. The subcutaneous tissue and skin are closed routinely with interrupted absorbable and running subcuticular monofilament suture.

Implant Considerations

The following sections will attempt to cover aspects of glenoid and humeral preparation particularly relevant to specific scenarios that might be encountered during shoulder arthroplasty for primary osteoarthritis, avascular necrosis, or other conditions with an intact or reparable cuff.

Humeral Preparation

Regardless of the type of humeral prosthesis to be implanted or the method of humeral head removal, the humerus must be completely delivered into the wound and cleared of all osteophytes. Removing the osteophytes before making any humeral cuts makes recognition of the previous anatomic neck and the rotator cuff attachment sites much easier. Therefore, the preferred first step, after dislocation of the humeral head, is removal of all humeral osteophytes.

Resurfacing Arthroplasty

In certain younger patients with a need to preserve bone for later revision or in patients in whom reaming of the intramedullary canal may not be prudent (i.e., previous osteomyelitis), humeral resurfacing, without humeral head excision, is indicated. In fact, if humeral bone stock is adequate, humeral resurfacing might be indicated in all patients undergoing hemiarthroplasty with a concentric glenoid. Although resurfacing arthroplasty has been described for both total shoulder arthroplasty and hemiarthroplasty in patients with osteoarthritis and avascular necrosis,[106] glenoid exposure is more difficult than it is when the humeral head has been resected. For this reason, we do not perform humeral resurfacing without humeral head resection during total shoulder arthroplasty. When humeral resurfacing is performed, even if the glenoid is not being resurfaced, the soft tissue releases described above for glenoid exposure may still need to be performed to maximize postoperative range of motion.

Humeral resurfacing is similar in concept to the cup arthroplasty performed for hip arthritis or avascular necrosis.[3] In humeral head resurfacing, the humeral head is retained and contoured to fit into a metallic shell using specialized contouring reamers.[106] The resurfacing arthroplasty used in our practice has five different head diameters (40 mm, 44 mm, 48 mm, 52 mm, and 56 mm) and two neck lengths with each head diameter (15 and 18 mm with the 40- and 44-mm heads and 18 and 21 mm with the 48-, 52-, and 56-mm heads). After delivering the humeral head into the wound and removing all osteophytes, the center of the head is estimated using a helmet guide and a guidewire is driven through the head at this center point and into the lateral cortex of the proximal humerus. The orientation of this guidewire is perpendicular to the plane of the anatomic neck. The appropriate head size will have been selected from preoperative templating. This size is verified intraoperatively and the appropriate reamer is chosen. The reamer is passed over the guidewire and is taken to the level of the cuff insertion. The humerus is trialed and a cruciform punch is used to cut the path for the stem. The selected head is then impacted onto the humerus in a press-fit manner. Bone graft can be placed between the humeral head and the native humerus. This implant can be used in patients with avascular necrosis so long as there is adequate live bone remaining (Fig. 22-16).

Replacement Arthroplasty

In all cases of total shoulder arthroplasty in our practice and in those cases of hemiarthroplasty in which resurfacing is not indicated, humeral preparation includes humeral head resection and reaming of the intramedullary canal. There are two basic methods of humeral head resection that are based on whether the implant used has a fixed neck–shaft angle or a variable neck–shaft angle. Most implants with a fixed neck–shaft angle employ either extramedullary or intramedullary cutting guides to assist the surgeon in making the humeral osteotomy at the appropriate angle for the prosthesis. Most variable neck–shaft angle implants encourage "free hand" resection of the humeral head, using the estimated anatomic neck or articular margin as a guide. Adaptability within the prosthesis itself will then allow the surgeon to place the humeral implant into a variety of positions.[166] In theory, this allows the surgeon to match the "normal" neck–shaft angle of the native humerus.

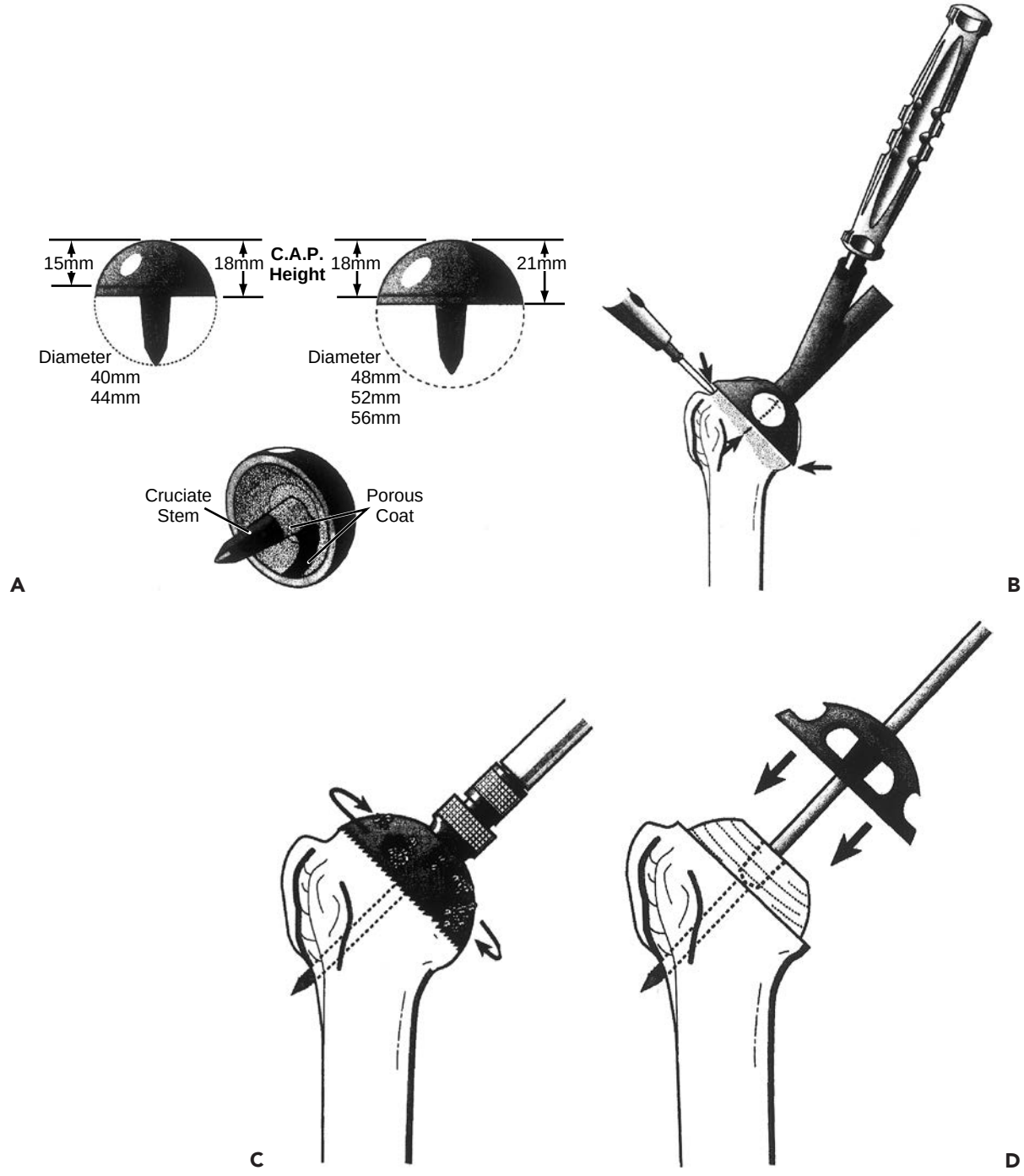
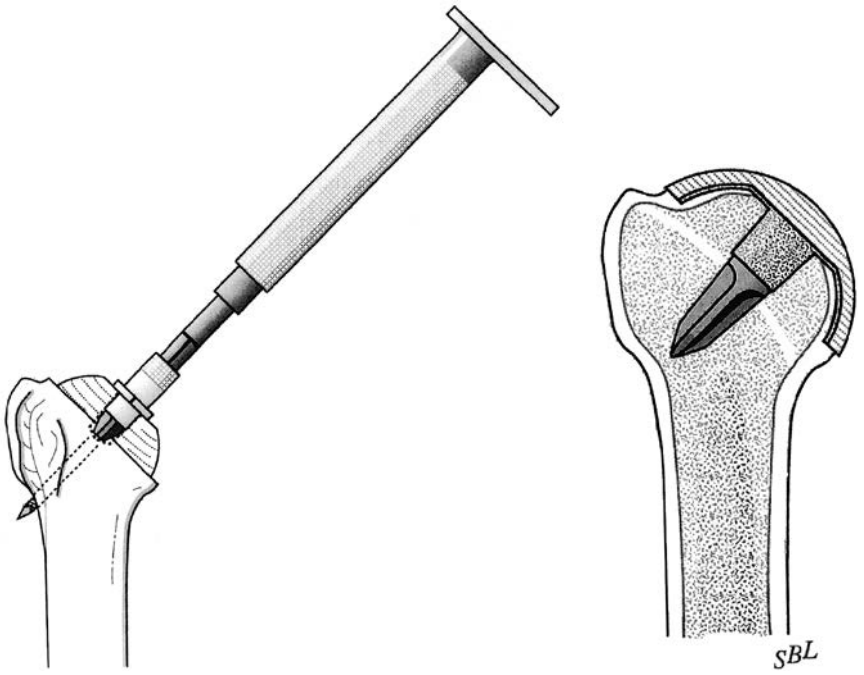


Figure 22-16 The Global CAP (Depuy, Warsaw, IN) has five head diameters with two neck lengths in each size **(A).** After osteophyte removal, humeral head size and center are selected with a helmet guide **(B)** and a guide pin is driven through the humeral head center into the lateral cortex. A reamer is then passed over the guidewire to shape the proximal humerus **(C).** The trial implant is placed and removed **(D);** the cruciform punch creates a path for the implant stem **(E)** and the guidewire is removed. The implant is then seated on the humerus **(F).**

The use of humeral prostheses with variable neck–shaft angles is based on the observation that the neck–shaft angle in normal humeri is variable and that anatomic reconstruction of the articular surface is desirable.[18,80,166] The disadvantage of this approach lies in the difficulty in identifying the normal neck–shaft angle in an arthritic humerus, even if the osteophytes have been removed. In the worst-case scenario, the humerus could be cut nonanatomically and reconstructed nonanatomically because of the ability of the prostheses to adapt to many



E F **Figure 22-16** (*continued*)

neck–shaft angles, even nonanatomic ones. Although it has been shown that anatomic reconstruction of normal cadaveric humeri is more likely to occur with prostheses with variable neck–shaft angles,[134,135] the same has not been demonstrated in humeri with primary osteoarthritis or avascular necrosis. However, it seems logical that using a prosthesis with an infinite number of neck–shaft angles within a preselected range, assuming that the native neck–shaft angle can be accurately identified, may encourage anatomic humeral reconstruction. More data are necessary to prove this, however.

In many cases, recreation of the normal humeral relationships is possible with a prosthesis with a fixed neck–shaft angle and the option of an offset humeral head. If one is going to use a fixed neck–shaft angle device, the humeral cut must be accurate for the prosthesis to sit flush against the cut surface of the humeral osteotomy. Therefore, a cutting guide is desirable. In many systems, the cutting guide is an extramedullary device that sits on the external surface of the humerus (Fig. 22-17). The guide is typically visually aligned with the shaft and held in place and the anterior humeral neck is marked. This defines the entry point for the humeral cut. The direction of the cut is determined by the amount of desired retroversion. In theory, the cut should approximate the retroversion of the native anatomic neck. In practice, the retroversion of the cut can be preselected at some average fixed number (i.e., 20 degrees) determined using the guide or the distal humeral epicondylar axis, or an attempt can be made to approximate the native retroversion using the posterior cuff attachment as a guide for the exit point of the cut. In either case, one should err on the side of removing too little posterior bone. Additional bone can be removed, but if too much bone is removed initially and the posterior rotator cuff attachment has been sacrificed, the situation cannot be salvaged.

Assuming that the humeral shaft is normal (i.e., straight), intramedullary cutting guides potentially offer

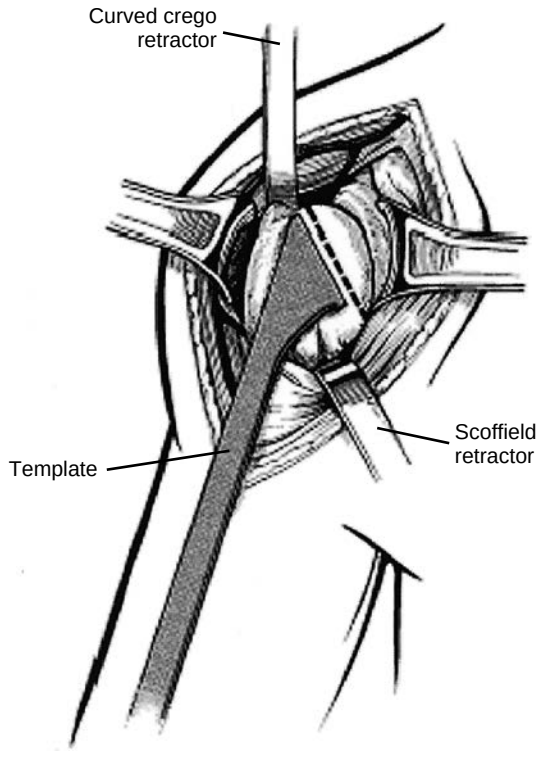


Figure 22-17 In many arthroplasty systems, an extramedullary guide is used to make the humeral osteotomy.

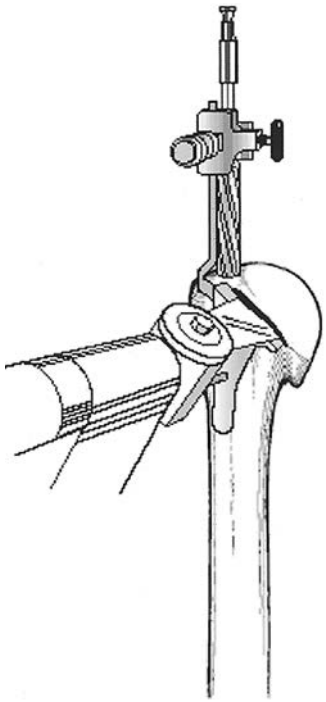


Figure 22-18 An intramedullary guide, based off the reamer, may also be used and may make a more accurate cut.

a more reproducible humeral cut. In this scenario, the humerus is reamed before the humeral cut has been performed (but after the osteophytes have been removed) and a cutting guide is attached to the reamer (Fig. 22-18). The entry point for the reamer on the superior surface of the humeral head should be the superior extension of the humeral intramedullary axis. In general, this point is approximately 1 to 1.5 cm posterior to the bicipital groove and approximately 0.5 to 1.0 cm onto the humeral articular surface. The humerus is reamed with sequentially larger reamers until the size that was estimated on the preoperative templating is reached. If there is too much resistance, even before the pretemplated size has been reached, the reamer is probably not coincident with the intramedullary axis. Under these circumstances, the surgeon removes the reamer and reams only the metaphysis with sequentially larger reamers until a reamer that is one or two sizes larger than the pretemplated reamer has been passed. Then he or she replaces the reamer that had been difficult to pass down the canal and redirect it. Overreaming the metaphysis will allow the reamer to be redirected down the shaft. If the pretemplated reamer still will not pass, the surgeon gets an intraoperative x-ray to evaluate reamer position and adjusts accordingly.

Once the final reamer has been passed, the surgeon leaves it in place. The cutting guide is then attached to the reamer. This guide will enforce the prosthetic neck–shaft angle. The height and retroversion of the guide should be such that the cut exits superiorly at the supraspinatus insertion site and posteriorly within 5 mm of the infraspinatus and teres minor attachment site. Again, one should err on removing too little bone, as more bone can always be removed. The remainder of the humeral preparation (i.e., broaching) is relatively straightforward and varies depending on the system used.

One caveat regarding humeral preparation in patients with avascular necrosis should be mentioned. The presence of hard, necrotic bone within the humeral metaphysis may make passage of the reamers and broaches difficult or even dangerous. If passage of the initial reamer is difficult, the surgeon uses a drill to make a hole in the metaphysis as large as the initial reamer, and then passes the reamers as usual. Once the humeral head has been resected, he or she is certain to remove enough of the hard, necrotic bone from the metaphyseal region to allow the footprint of the body of the broach to pass. The surgeon does not rely on the broach to compress the metaphyseal bone enough to allow complete seating. In the worst-case scenario, the increased hoop stresses resulting from the unyielding avascular bone may cause fracture of the proximal humeral metaphysis.

Glenoid Preparation

The prime decision to be made regarding glenoid preparation is whether or not the glenoid is to be resurfaced at all. In all other stages of avascular necrosis but stage V, the native glenoid surface is not diseased enough to require resurfacing. Patients with postcapsulorrhaphy are often young and quite active, making glenoid resurfacing potentially undesirable. In osteoarthritis, however, the native glenoid is abnormal in all but the earliest of stages of the disease process. The decision between hemiarthroplasty and total shoulder arthroplasty has been a controversial one, although the more recent data would support the use of a glenoid component in most cases of osteoarthritis.

Hemiarthroplasty Versus Total Shoulder Arthroplasty

Both hemiarthroplasty and total shoulder arthroplasty are considered acceptable treatment options in the face of the advanced degenerative change of osteoarthritis and osteonecrosis. Neer's initial description of prosthetic replacement for osteoarthritis included 47 hemiarthroplasties and one total shoulder arthroplasty.[124] Subsequent results of humeral head replacement at midterm follow-up have been favorable.[124,129] Some of these patients will, however, have persistent pain, and intuitively it seems that if glenoid wear is present that both sides of the joint should be addressed at the time of surgery. Recurrent or persistent pain after hemiarthroplasty is not uncommon, and it has been shown that later revision to a total shoulder arthroplasty results in symptomatic improvement and an increased range of motion.[155] In the interest of avoiding the need for a second surgery, many have tried to define what degree of glenoid involvement warrants resurfacing.

Greater technical difficulty and the increased incidence of glenoid-related complications have tempered enthusiasm for routine glenoid resurfacing in all patients.

Many reports describe the incidence of lucent lines under the glenoid, but few include exclusively osteoarthritic patients. Overall, lucent lines under the glenoid are seen radiographically in approximately 30% to 50% of patients immediately postoperatively.[68,103,145] The large majority of these lucencies, however, do not signify loosening and are not correlated with poor outcome.[35,145,172] However, there is some concern that longer follow-up will result in a correlation between lucent lines and loosening in some cases.[35,162] If "glenoid loosening" is defined as migration, dislocation, or a complete radiolucent line measuring greater than 2 mm, the overall reported rate is approximately 10% at 10 years.[10,22,79,127]

Boyd et al. compared patient satisfaction and functional improvement in patients who received either hemiarthroplasty or total shoulder arthroplasty. They found that only those patients with inflammatory arthropathy had significantly better pain relief, range of motion, and overall satisfaction with total shoulder replacement as opposed to hemiarthroplasty. They recommended hemiarthroplasty for patients with osteoarthritis and avascular necrosis, provided that they had a concentric glenoid and absent synovitis preoperatively.[22]

Iannotti and Norris reviewed[81] the results of total shoulder arthroplasty and hemiarthroplasty in 160 patients (176 shoulders) with primary osteoarthritis. The choice of hemiarthroplasty was left up to the surgeon, with no control for the degree of glenoid involvement. Patients with asymmetric glenoid wear fared better with total shoulder arthroplasty than hemiarthroplasty. In this series, patients with an intact cuff, with minimum glenoid wear, and without humeral head subluxation had statistically better results with total shoulder arthroplasty.

In Cofield et al.'s series,[40] patients with osteoarthritis who received a hemiarthroplasty were less satisfied than a group of patients with rheumatoid arthritis after the same procedure. In fact, in this series, 18% of the patients with osteoarthritis later underwent conversion to a total shoulder arthroplasty because of poor pain control. Moreover, conversion to total shoulder arthroplasty was successful in relieving pain in most patients.

In another series of patients who underwent hemiarthroplasty for osteoarthritis, Levine et al. found that satisfactory results were obtained in only 74%, and that poor outcome most significantly correlated with the degree of posterior glenoid wear.[105] These authors also concluded that hemiarthroplasty should be reserved for patients with a concentric glenoid.

More recently, Gartsman et al. randomized a group of 51 shoulders awaiting prosthetic replacement to receive either hemiarthroplasty or total shoulder arthroplasty. After an average follow-up of 35 months, no significant difference was found in shoulder scores, but they did identify that patients undergoing total shoulder arthroplasty experienced greater pain relief and had greater internal rotation. Total shoulder arthroplasty was associated with an estimated increased cost of approximately $1,177 per patient.[66] However, three of the patients who initially underwent hemiarthroplasty had inadequate pain relief requiring revision to total shoulder arthroplasty. The mean incremental cost per patient for these conversions was approximately $15,998.

Edwards and colleagues[49] compared 601 total shoulder arthroplasties with 89 hemiarthroplasties performed for primary osteoarthritis. Although improvement in pain, mobility, and activity was seen in both groups, total shoulder arthroplasty provided superior results.

Clearly, more data are needed to determine whether there is any difference in pain relief or outcome score between hemiarthroplasty and total shoulder arthroplasty in patients with osteoarthritis. However, it seems that the only reason for not replacing a diseased glenoid is the potential for greater glenoid complications. We prefer to pay extreme attention to those details that are known to affect glenoid longevity (cement technique, circumferential bony support of the glenoid component, component orientation, patient age and activity level) and resurface the glenoid in all patients of appropriate age or activity level (discussed under indications above) with adequate bone, an intact or reparable rotator cuff, and a diseased glenoid.

Technical Aspects of Glenoid Resurfacing

Many of the technical details regarding glenoid resurfacing are implant specific. However, there are two principles that are universal and will be emphasized here. These principles are normalization of glenoid version and concentric osseous support for the entire glenoid component.

Restoration of normal glenoid version, by definition, is only required when the orientation of the native glenoid is abnormal. In most cases of avascular necrosis, glenoid orientation or version is normal. However, primary osteoarthritis and postcapsulorrhaphy arthropathy are often associated with posterior glenoid deficiency, increased glenoid retroversion, and posterior humeral subluxation. When these deficiencies are not congenital in nature (i.e., Erb's palsy or congenital hypoplasia), an attempt should be made to correct them. The simplest way is to asymmetrically ream the high (anterior) side so that the remaining glenoid is concentric and oriented with proper retroversion. This can be done so long as the amount of anterior glenoid to be removed does not compromise the base of the coracoid process or the amount of bone left in the glenoid vault to accept a prosthesis. In most cases, 1 to 1.5 cm is the limit for asymmetrical reaming. Since the glenoid cavity is funnel-shaped, the more medial the surface taken is, the narrower it gets. Furthermore, as the anterior aspect of the glenoid is asymmetrically reamed, the center point of the glenoid surface will move

closer to the anterior edge. Therefore, we prefer to change glenoid version with a reamer that does not key off the center of the surface and to switch to a piloted reamer that sits in a central drill hole to fine tune the surface reaming.

In cases of posterior bone loss that is greater than 1 to 1.5 cm with respect to the anterior glenoid rim, posterior glenoid bone grafting may be necessary (Fig. 22-19). This can be estimated on preoperative CT scans or MRI scans. If the need for glenoid bone grafting is suspected on the basis of preoperative studies, asymmetrical anterior glenoid reaming should not be initially performed but may be performed after the graft is securely placed. The goal of anterior glenoid reaming is to achieve a concave surface that matches the contour of the posterior aspect of the glenoid component

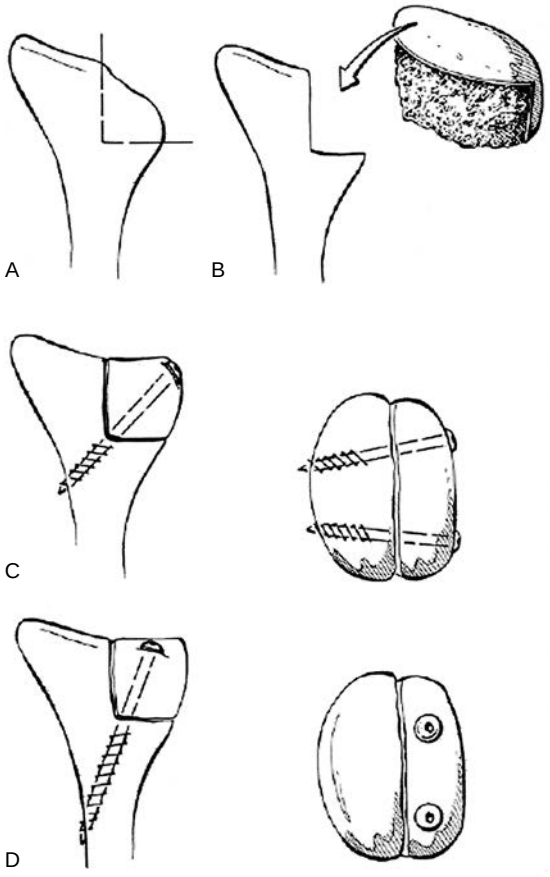


Figure 22-19 Glenoid deficiency (e.g., posterior) can be managed by creating a perpendicular surface on the native glenoid using a burr **(A)** and using a piece of the humeral head to recreate the glenoid surface in appropriate version **(B).** Screws used to hold the graft in place can be passed through the posterior edge of the graft from posterior to anterior—usually through a separate posterior stab wound **(C).** Alternatively, the screws can be passed in a more medial to lateral direction through the lateral surface of the graft **(D).** In the latter case, the screw heads should be recessed below the surface of the graft so as not to interfere with seating of the component. (From Iannotti JP, Schenk T. Prosthetic arthroplasty for glenohumeral arthritis and an intact or reparable rotator cuff: indications, techniques, and results. In: Iannotti JP, Williams GR, eds. *Disorders of the shoulder: diagnosis and management.* Philadelphia: Lippincott Williams & Wilkins, 1999.)

up to at least the midpoint of the glenoid surface. The surface of the glenoid will then be biconcave, with an anterior half in the appropriate version with a radius of curvature that matches the back of the glenoid component and a posterior half that slopes from the higher center edge to the lower posterior cortical margin. A burr is used to create a flat surface, perpendicular to the scapular axis, without removing any of the remaining posterior cortex. A graft is fashioned to fit this perpendicular surface and is provisionally held with superior and inferior peripheral K-wires. The graft is then secured with two screws countersunk so that their heads are 2 to 3 mm below the graft surface. The K-wires are then removed (Fig. 22-20).

The glenoid is then prepared to receive the glenoid component. If a keeled component is to be used, a slot is made for the glenoid keel. If a pegged design is chosen, it is important to place some, if not all, of the pegs within the bone of the native glenoid rather than the graft. A burr is used to contour the posterior bone graft to fit the back of the glenoid component. The component is then cemented into place. Other alternative methods of glenoid bone grafting have been described. The complication rate (early loosening, hardware loosening or migration, or hardware breakage) is higher with any grafting technique than with glenoid replacement without the need for bone grafting.[80,126,156]

Cemented Versus Uncemented Implants

The use of cement in the fixation of both humeral and glenoid components is also controversial. Many humeral components consist of a thin, distally tapering stem that is somewhat cylindrical, even in the proximal metaphyseal portion of the implant. Although these implants are often used successfully in a press-fit mode, reports of subsidence or loosening have surfaced.[37,155,162] It is possible that a surface coating encouraging biologic in-growth will improve the situation.[153] In addition, radiographic follow-up of press-fit prostheses that are enlarged proximally to fill the metaphysis have shown low loosening rates.[115] However, one must be careful in attempting to gain distal fixation with this implant in elderly patients with an enlarged canal because the larger the stem is, the larger the proximal body is. It is possible to encounter a situation in which the distal portion of the implant fits the shaft but the proximal portion is so large that it may fracture the metaphysis. We prefer this proximally filling implant without cement in almost all cases of primary osteoarthritis, avascular necrosis, and postcapsulorrhaphy and most cases of rheumatoid arthritis. Some rheumatoid patients have such poor bone quality that cement is necessary. The one combination that deserves caution because of difficulty in revision is the use of an implant coated with a surface to encourage biologic in-growth in the presence of cement.

The reported experience with uncemented glenoid components is much less extensive than the experience with

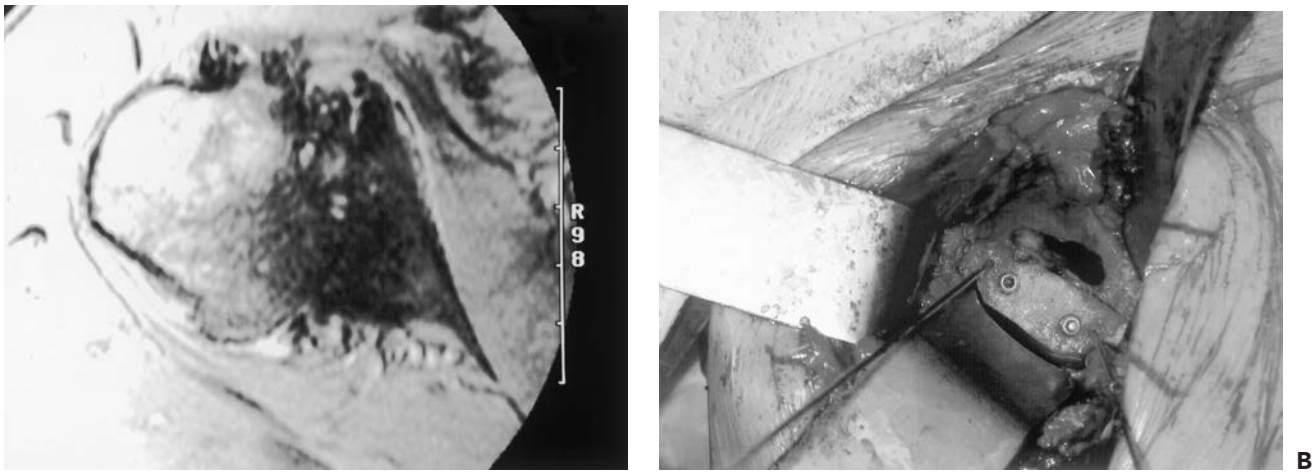


Figure 22-20 **(A)** Preoperative magnetic resonance image demonstrating greater than 1.5 cm of posterior glenoid bone loss and **(B)** intraoperative photograph with posterior bone graft in place. The K-wires will be removed and the screws countersunk before placement of the glenoid component.

uncemented humeral components.[17,34,37,38,120,153] In addition, the experience, until very recently, has not been outstanding. Cofield has the earliest and longest experience with uncemented glenoid components. His glenoid is a metal-backed component with a porous coating to encourage biologic in-growth. Early experience with this implant has been cautiously encouraging. However, concern for increased polyethylene wear and polyethylene dissociation has limited its use to patients with bone loss too severe to allow cementing of a keeled component and certain patients with intuitively good quality glenoid bone.[34,37,38,153]

A recent randomized controlled study directly compared the radiographic appearance and longevity of cemented polyethylene glenoid components and uncemented metal-backed components.[17] It was found that cemented polyethylene glenoid components have a much higher incidence of radiolucency on immediate postoperative x-rays, but that this line was rarely associated with progression and almost never a prognostic indicator. On the other hand, uncemented metal-backed components rarely have a lucency present on initial x-rays, but when they do, it usually is associated with progression and loosening. The overall incidence of loosening is higher in some metal-backed components, and thus metal-backed components are not used by most surgeons at this time.[17] However, continued research in this area is warranted because of the prospect of permanent biologic fixation.

Soft Tissue Balancing

Restoration of the proper tension in the deltoid and rotator cuff myofascial sleeves is the hallmark of shoulder arthroplasty. Only restoration of appropriate soft tissue tension and muscular length can restore optimal range of motion, strength, and function. Although restoration of normal soft tissue tension may not be possible in all cases, it should be the goal of all shoulder arthroplasties performed for avascular necrosis, primary osteoarthritis, and other conditions with an intact or reparable cuff.

The term "soft tissue balancing" is often used in shoulder arthroplasty. Tightening of tissues that are too loose is infrequently required. More often, "soft tissue balancing" requires releasing or lengthening of tissues that are too tight. Even in cases of posterior glenoid deficiency and posterior subluxation, the need for plicating loose posterior tissues is uncommon.

Assuming that the releases discussed above have been made during the surgical approach and glenoid exposure, the time to evaluate soft tissue balance is after the glenoid component has been finally fixed and before the humeral broach has been removed for the final humeral implant placement. A trial humeral head is selected based on preoperative templating, the size of the humeral head removed at surgery, and intraoperative humeral head trialing performed in the earlier portion of the procedure, prior to glenoid exposure. Once the trial head is placed on the broach, the size and humeral head offset are checked by verifying that the humeral head is centered on the humeral metaphysis, that the superior edge of the articular surface of the head meets the humerus at or slightly medial to the supraspinatus tendon–bone junction, and that the distance between the top of the humeral head and the top of the greater tuberosity is approximately 5 to 8 mm.

The humeral head center is, on average, offset posteriorly and medially with respect to the axis of the humeral shaft.[18] Since the stem of the implant or broach is centered within the shaft, a humeral head component with some offset is often required to place the head within the broach or stem and centered on the humeral metaphysis. However, there are occasions where the humeral head offset required

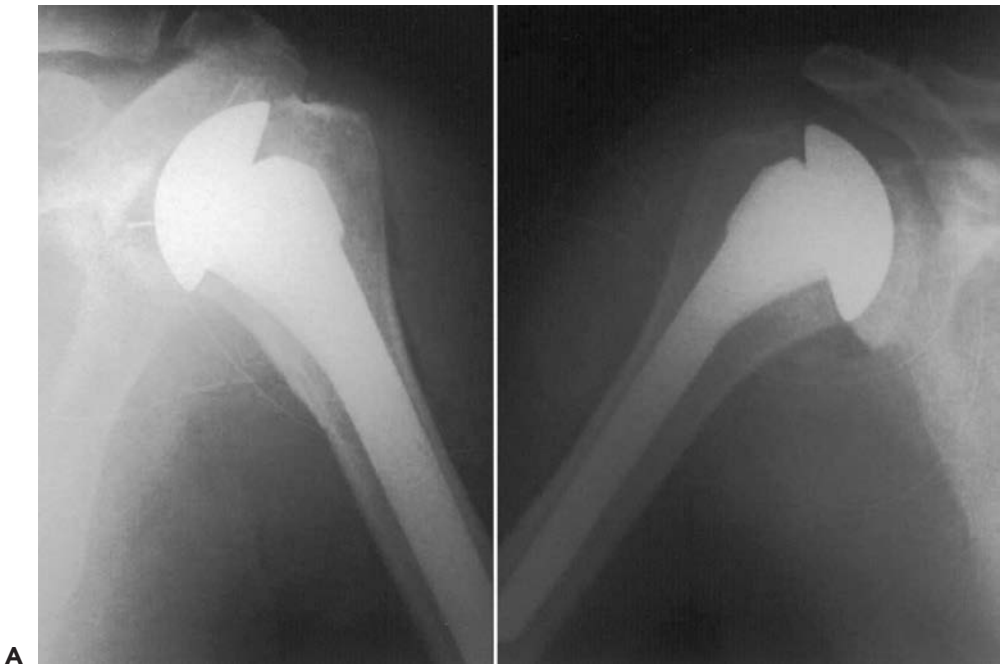


Figure 22-21 In some systems, it is possible to place an offset **(A)** or a centered **(B)** humeral head when needed.

is very little or none. Therefore, if one is using a system with no centered option for the humeral head, a decision must be made with regard to which direction the offset will be placed (i.e., which portion of the metaphysis will be overlapped by the humeral head component). Conversely, if the arthroplasty system has a centered option for the humeral head, it can be placed in this centered position without overlapping any portion of the metaphysis (Fig. 22-21). Alternatively, in either case, the stem can be downsized and cemented off-center within the humeral canal.

With the appropriate humeral head trial fixed to the humeral broach or trial stem in the appropriate position, the humerus is reduced into the glenoid to test for soft tissue tension. In general, the soft tissues are of adequate length if the joint can be put through a normal range of motion passively, except for external rotation, which will obviously be increased because the subscapularis is still detached. There are, however, more specific tests for soft tissue tension or length. If the soft tissues are the correct length, posterior translation of the humerus on the glenoid with the arm at the side and the humerus in neutral rotation should be approximately 50% of the width of the humeral head. Translation of the entire width of the head can also be accepted so long as the humerus returns to the glenoid spontaneously after the posteriorly directed force is removed, and the glenoid is not excessively retroverted. This should also be tested with muscular paralysis reversed, if there is any question. Adequate posterior soft tissue length can also be checked by ensuring that the hand can be brought to touch the opposite shoulder. In addition, proper soft tissue tension should allow inferior translation of one-fourth to one-half the width of the humeral head with the arm at the side in neutral rotation. The final soft tissue tension test is subscapularis length. The subscapularis should come to its projected reattachment site easily with the humerus in 45 degrees of external rotation with the arm at the side. If all of these soft tissue conditions are met, the current implants are selected and inserted.

If the soft tissues are too tight symmetrically, the joint is overstuffed relative to the soft tissue envelope, and there are three possible problems. First, the humeral cut may be wrong, with too much bone remaining. Second, the humeral head size may be too large. Third, the soft tissue releases may be inadequate. The humeral cut and soft tissue releases should be checked and corrected if needed. Inability to reseat the trial stem after the cut has been corrected could be indicative of stem malalignment, which can then be verified with intraoperative radiographs. If the proper humeral cut and adequate soft tissue releases have been verified or corrected and the joint is still too tight, the humeral head size should be decreased. Depending on the arthroplasty system used, the humeral head can be downsized by decreasing the radius of curvature, the humeral neck length, or both. Changing the neck length or head radius without correspondingly changing the other will have an effect on the surface area of the humeral head available for articulation with the glenoid. One should check to be sure that physiologic ranges of motion are possible without a nonarticular portion of the humerus hitting the glenoid surface.

If posterior and inferior translations are inadequate but subscapularis length is appropriate, an imbalance between the anterior and posterior–superior soft tissues exists, and there are two potential explanations: Either the subscapularis is too long or the posterior–superior soft tissues are too short. Again, the humeral bone cut and soft tissue releases should be checked and corrected if necessary. If the imbalance still exists, the humeral head should be downsized and the excessive subscapularis length corrected. This can be done by decreasing the amount of medialization of the attachment site to bone or by overlapping the repair. Alternatively, the excess length can be accepted with the likely resultant weakness, which is usually well tolerated. One other potential explanation of this imbalance is excessive posterior humeral head offset. Under these circumstances, the direction of the humeral head offset can be changed but may result in other unintended consequences, depending on the direction and amount of the change.

If the soft tissues are universally and symmetrically too long, the joint is too loose. Remember that, in general, it is better for a shoulder to be too loose than too tight. However, if the translation is truly excessive, it should be corrected. First, the rotator cuff attachment sites and tuberosities should be checked for inadvertent detachment or fracture. If this has not occurred, it is likely that the head size is too small. This can occur with excessive medialization of the joint line from glenoid reaming, for example. The excess laxity can be addressed by increasing the head size or lateralizing the joint line with a thicker polyethylene glenoid component, if that option is available. This is one argument for testing soft tissue tension before the glenoid has been permanently implanted. If the system used has this option and one is aware that glenoid reaming caused significant medialization of the joint line, a thicker glenoid trial can be used to test the soft tissue prior to permanent glenoid implantation.

When changing humeral head sizes to make up for either excessive joint laxity or tightness, it is important to know whether the system being used has any mismatch between the humeral head and glenoid radii of curvature. In some systems the radii are always equal; in others there is a fixed amount of offset between radii; and in still others the offset varies, depending on which combination of humeral head and glenoid is chosen. One should try to avoid having a humeral head with a larger radius of curvature than the glenoid component and having a radius of curvature of the glenoid that is larger than the humeral radius by more than 10 mm. Although some systems can allow this, a mismatch this large can cause stresses large enough to exceed the yield point of some polyethylenes.[64]

The final situation that can be encountered, although rarely, is excessive posterior translation with either appropriate subscapularis length or relative subscapularis shortening. Under either of these circumstances, the subscapularis should be checked to be sure that it is being released adequately. The two most common areas of continued soft tissue tenodesis are at the inferior border, where the inferior capsule may not have been adequately released from the glenoid, and at the superior border, at the base of the coracoid process. Both of these areas should be released completely, taking care to protect the axillary and musculocutaneous nerves. Release of adhesions on the superficial surface of the subscapularis muscle belly should not be taken farther than the lateral extent of the base of the coracoid to protect the superficially entering nerve supply.[175] In most cases, this will lengthen the subscapularis enough to allow increasing the size of the humeral head to address the posterior laxity.

If the posterior soft tissues are still too loose, there are three options. The first is to increase the humeral head size and accept less external rotation. One should not accept less than 30 degrees of external rotation. The second option is to change the offset of the humeral head posteriorly and accept the resultant changes in head position. In general, this will decrease the greater tuberosity–humeral head distance, which should not be less than 0.5 to 1.0 cm. The final option is to perform a posterior capsular plication through the joint, with the head removed.[122]

RESULTS

In general, results following shoulder arthroplasty are disease specific. The best results are in conditions with an intact or reparable cuff. Among those conditions, the order of decreasing results is nontraumatic avascular necrosis, osteoarthritis, arthritis of instability, and rheumatoid arthritis. In properly selected patients, arthroplasty for primary osteoarthritis or avascular necrosis provides dramatic relief of pain, improvement in function, and patient satisfaction. Approximately 90% of patients report complete or near-complete pain relief.[35,68,69,81,113,114,127,129,132,163] Applying literature estimation of outcome results to a population of exclusively osteoarthritic patients has been difficult in the past because most series have reported results from a mixed disease population. However, several recent case series of patients with osteoarthritis have documented significant functional improvement after arthroplasty for this indication.[55,68,70,132] The degree of postoperative functional improvement is inversely correlated to preoperative function. The degree of preoperative stiffness is correlated with the postoperative range of motion and function.[81] It has also been noted that outcome and patient satisfaction are not significantly different for those patients with repairable rotator cuff tears that received either hemiarthroplasty or total shoulder arthroplasty. The presence of a large tear or fatty infiltration of the rotator cuff is a negative prognosticator.[68,69] Overall, 95% of patients demonstrate a good to excellent outcome (pain relief, functional improvement, and patient satisfaction).[132]

The results of shoulder arthroplasty for osteonecrosis have been less frequently reported than those for osteoarthritis. The general belief is that the results are

among the best of any patients undergoing shoulder arthroplasty because of the integrity of the rotator cuff in most of these patients. This is true, with some provisos. First, assuming all other prognostic factors to be equal, arthroplasty for patients with steroid-induced osteonecrosis have better results than patients with other causes of osteonecrosis.[76] Second, glenoid changes tend to be underestimated at the time of surgery and can be associated with persistent pain and glenoid erosion in patients undergoing hemiarthroplasty.[133] Finally, patients undergoing shoulder arthroplasty may have difficulty in regaining their motion postoperatively and have lower functional scores because of this.[76] The results of arthroplasty may not be as good, relative to primary osteoarthritis, as previously thought; further work is necessary to further delineate the prognosis of patients undergoing shoulder arthroplasty for osteonecrosis.[133,147]

There are few reports specifically outlining the results of arthroplasty in patients with postcapsulorrhaphy arthritis or arthritis of instability.[15,25,72,116–118,152] When compared to other patients requiring shoulder arthroplasty, these patients are younger, predominantly male, and technically more demanding because of severe soft tissue contracture and frequent glenoid bone loss that requires substantial asymmetric reaming and bone grafting. As a result, the pain relief and return of function is not as reliable as in osteoarthritis and osteonecrosis. Moreover, there is a higher early revision rate, presumably because of glenoid-related pain in young patients undergoing hemiarthroplasty.

Inflammatory arthritis (i.e., rheumatoid arthritis) exhibits a spectrum of severity. Consequently, the functional results following arthroplasty are more variable than with osteoarthritis or avascular necrosis.[11,44,57,84,85,89,90,108,119,121,150,159,160,164,169] Patients with rheumatoid arthritis requiring shoulder arthroplasty are typically younger than patients with osteoarthritis and are also predominantly female. The choice between hemiarthroplasty and total shoulder arthroplasty is difficult because of the high incidence of rotator cuff tearing or dysfunction and glenoid erosion. Pain relief is predictable whether hemiarthroplasty, resurfacing arthroplasty, or total shoulder arthroplasty is performed. However, the functional results are less predictable because of rotator cuff deficiency. In addition, progressive proximal humeral migration following shoulder arthroplasty is common because of continued rotator cuff degeneration and superior–central glenoid bone loss.[149,150]

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23 Cuff Deficiency Arthropathy: Unconstrained and Constrained Shoulder Arthroplasty

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INTRODUCTION

The arthritic shoulder with a massive irreparable rotator cuff tear presents a unique set of surgical challenges distinct form either the massive tear or the arthritic shoulder when each present as separate pathology. Cuff-deficient shoulder arthropathy presents with severe articular and periarticular soft tissue damage. This clinical entity is an end-stage result of several diseases such as rheumatoid arthritis, rotator cuff tear arthropathy, Milwaukee shoulder syndrome, and psoriatic arthritis and posttraumatic problems associated with fracture of the tuberosities and failed prosthetic surgery. Large and massive defects in the rotator cuff tendons lead to a loss in the centering of the humeral head within the glenoid fossa and subsequently result in superior migration of the humeral head. Loss of a fixed center of rotation for the humeral head results in decreased

power of the deltoid. In addition, massive cuff deficiency leads to secondary severe damage to both the intraarticular and other extraarticular soft tissue and bony structures. The result is a painful, dysfunctional shoulder that necessitates, in many cases, a surgical procedure to decrease pain and provide better function. The lack of adequate stability provided by the rotator cuff and the defects in bone stock makes the task of replacing the damaged joint with a stable construct a difficult surgical goal. The aims of this chapter are to review the pathomechanics of this disorder, to evaluate the different treatment options, to discuss the indications for surgical treatment, and to elaborate on the different surgical options. We will establish a decision-making algorithm for the patient with a rotator cuff tear arthropathy based upon current treatment options.

PATHOMECHANICS

The glenohumeral joint lacks significant intrinsic bony stability, and thus relies largely on its soft tissue components for stability. The rotator cuff tendons provide a major contribution to the dynamic stabilization of the glenohumeral joint by creating a compressive force within the concavity of the glenoid fossa.[5,31,34,37,65] By its synchronous net inferior and compressive vector, the rotator cuff opposes the superior displacing effect of the strong deltoid muscle. The rotator cuff functions to keep the humeral head centered in the glenoid fossa throughout the movement of the shoulder.[57,70,71] The coupled compressive and inferiorly directed forces of the infraspinatus and subscapularis muscles has been shown to be a major factor in maintaining glenohumeral stability, while the contribution of supraspinatus is less significant.[35,36] A massive tear, consisting of the supraspinatus and at least one of the other rotator cuff tendons[11] (in most cases the infraspinatus), may render the rotator cuff's anterior and posterior force couple ineffective in both the vertical and the transverse planes. The result is a diminution of joint reaction force and a change in the overall direction of the joint force that leads to the superior destabilization of the glenohumeral joint.[47] In cases where the long head of biceps is still functional, it may oppose, to some extent, the superior migration of the humeral head.[35,36]

Once the proximal pull of the deltoid is left unopposed, the humeral head migrates superiorly toward the coracoacromial arch. The humeral head articulates with the coracoacromial arch superiorly and the superior glenoid rim inferiorly, leading to flattening of the superior part of the humeral head and tuberosities ("femoralization"), rounding and thinning of the coracoacromial arch ("acetabularization"), and destruction of the superior glenoid region (Fig. 23-1). This process was termed by Neer as rotator cuff tear arthropathy. The acromioclavicular joint is also frequently involved in the process, joining its intraarticular space with that of the now joined synovial intraarticular and subacromial bursae spaces. When the humeral head migrates superiorly, its fulcrum for rotation within the glenoid is lost and the deltoid has a smaller mechanical advantage, as a result of its shortened fiber length. The deltoid must therefore generate more force to perform its function.[62] The end result is an incongruous, unstable joint with a higher joint friction and superiorly malpositioned center of rotation.

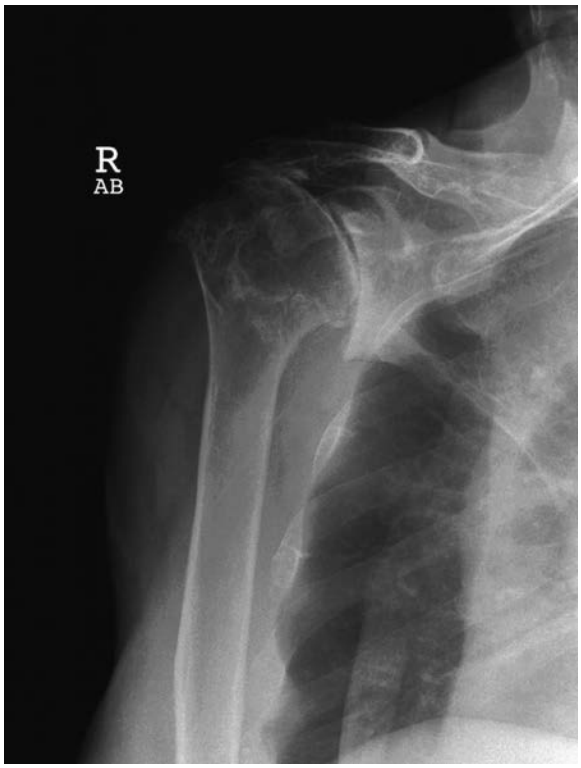


Figure 23-1 This x-ray image is typical and consists of a superiorly positioned humeral head, and in the end stages of the process an "acetabularized" socket consisting of a thinned sclerotic acromion and the eroded upper glenoid fossa.

CLASSIFICATION

A pathomechanistic and pathomorphologic classification of rotator cuff–deficient arthritis (RCDA) based on the position and stability of the humeral head is presented in Fig. 23-2.[56,63]

The classification is independent of the underlying pathologic conditions and is based on two critical issues for the function of the deltoid muscle: the glenohumeral center of rotation and the degree of anterior–superior instability. Four distinct groups have been formed on the basis of the biomechanics. The four types are distinguished by the degree of superior migration from the center of rotation and the amount of instability of the center of rotation.

Type Ia: Centered stable	Type I b: Centered – Medialized	Type II a: Uncentered – limited stability	Type II b: Uncentered unstable	Relationship of the lever-arms of deltoid and of the upper limb
No superior migration		Superior Translation	Anterior-superior Dislocation	
Acetabularisation of c.a. arch; Femoralisation of humeral head	Medial erosion of the glenoid	Minimum stability by c.a. arch	No stability by c.a. arch	

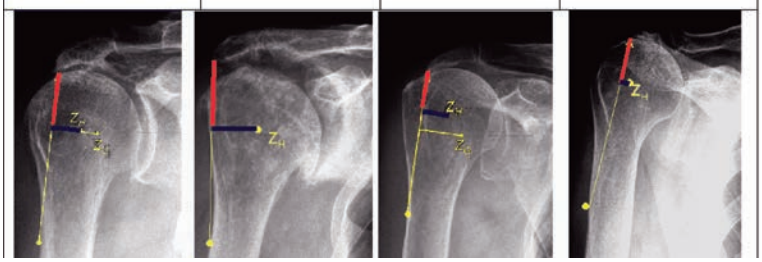


Figure 23-2 A pathomechanistic and pathomorphologic classification of rotator cuff–deficient arthritis, based on the position and stability of the humeral head, is presented.

In the type II cases the humeral head is not centered in the fossa or contained within the coracoacromial arch, and by this definition has an unstable fulcrum.

DIFFERENTIAL DIAGNOSIS

Although sharing a common end result, it is important to recognize the various diseases processes leading to glenohumeral RCDA.

Rheumatoid arthritis (RA) has been the most common cause of RCDA. The frequency of this cause of RCDA has decreased markedly with the use of disease-modifying drugs. Traditionally 48% to 65% of RA patients have significant glenohumeral joint involvement. About 24% of those having glenohumeral arthritis will have a simultaneous rotator cuff tear.[38,59] Superimposed on the aforementioned changes are severe osteopenia, erosions of the *entire* glenoid without osteophyte formation, and medialization of the glenohumeral joint.

Cuff tear arthropathy (CTA) is the extreme end result of a massive rotator cuff tear. The term, coined by Neer in 1983,[43] refers to a primary massive rotator cuff tear that by virtue of mechanical superior instability and nutritional effects leads to a secondary glenohumeral joint destruction.[22] It is believed that between 0% to 25% of massive rotator cuff tears will end up as CTA, but it is difficult, if not impossible, to predict which of the massive tears will result in CTA.

The Milwaukee shoulder syndrome was originally described by McCarty in 1981.[42] This is an uncommon entity affecting shoulders of elderly people, predominantly women. It consists of a massive rotator cuff tear, joint instability, bony destruction, and large blood-stained joint effusion containing *basic calcium phosphate crystals,* detectable protease activity, and minimal inflammatory elements. Its relation to rotator cuff arthropathy is not clear, and it might represent one spectrum of those mentioned above. The role of the basic calcium phosphate crystals in creating this syndrome is still controversial. Whether it is the cause of the articular damage through macrophage spillage of proteases or just the result of the osteoarthritic process is still unknown.[33]

Primary glenohumeral osteoarthritis is the most common reason for shoulder joint replacement; however, it is associated with rotator cuff tears in only 5% of patients, most of which are reparable. It is therefore uncommon for primary osteoarthritis to end up as RCDA.

Posttraumatic conditions can lead to RCDA when the tuberosities are fractures and ununited after traumatic rotator cuff tears or failed rotator cuff surgery.

CLINICAL PICTURE

Patients with arthritic shoulder and irreparable massive cuff deficiency secondary to classic cuff tear arthropathy are primarily elderly people with female gender predominance. When considering other causes of massive cuff deficiency with secondary arthritis, male patients predominate. The patient's main complaints are of severe shoulder pain, limited range of movement, and in some cases recurrent swelling of the shoulder. The pain is constant, aggravated by shoulder motion and felt at the periacromial region and the glenohumeral joint line. On physical examination the examiner can observe wasting of the infraspinatus and supraspinatus muscles, a decrease in active and passive glenohumeral motion, and a crepitus

while moving the patient's shoulder.[33] The x-ray image is typical and consists of a superiorly positioned humeral head and in the end stages of the process an "acetabularized" socket consisting of a thinned sclerotic acromion and the eroded upper glenoid fossa (see Fig. 23-1). Occasionally, the acromioclavicular joint and distal clavicle are also damaged and are thus included in the "socket." Cases of secondary stress fractures of the thinned acromion have also been published.[17]

The combination of the clinical and radiologic information is, in most cases, sufficient to make the proper diagnosis, although other modalities such as computed tomography (CT) may be needed for treatment planning.

TREATMENT

Rotator cuff tear arthropathy combines severe articular damage, bone destruction, osteoporosis, and loss of stabilizing rotator cuff tendons. In contrast to the more common primary degenerative shoulder arthrosis, the inherent instability of the rotator cuff–deficient shoulder necessitates specific consideration. Severe pain and shoulder dysfunction lead many of these patients to seek medical advice. There are several treatment options, and the ideal treatment for any one patient must be individualized to the patient's pathology, functional disability, and treatment goals. These options include nonsurgical treatment and surgical procedures such as humeral head replacement, total shoulder arthroplasty, and even arthrodesis and resection arthroplasty.

TREATMENT OPTIONS

Nonsurgical

Patients with cuff tear arthropathy may retain a surprising degree of function and motion if they have an intact deltoid and acceptable fixed fulcrum mechanics for rotation of the humeral head. Patients with mild symptoms and mild limitation in functional range of motion and activities of daily living should be treated nonsurgically. This includes the use of analgesics and physical therapy to maintain range of motion and to strengthen the deltoid muscle. It has been shown that by strengthening the middle third of the deltoid, some improvement with superior stability control can be gained.[27] The use of repeated steroid injections is discouraged, but an occasional injection may be helpful in managing the most acute symptoms.

In those patients with unremitting pain, significant motion-related pain, and limitation in range of motion and activities of daily living, a surgical intervention should be considered.

Glenohumeral Arthrodesis

The basic concept of fusion is to eradicate pain with elimination of motion. However, there are several drawbacks to its use in this condition:

1. Arthrodesis should ideally be done when there is good function in the opposite shoulder. In as many as 40% of patients with RCDA, the opposite shoulder is involved in a similar process.
2. The involved shoulder is in most cases severely osteopenic, and thus is more prone to failure of internal fixation and subsequent nonunion.
3. Increased scapulothoracic motion needed after glenohumeral arthrodesis exposes the already damaged acromioclavicular joint to excessive motion and therefore pain.
4. Most of the patients involved are elderly patients. Elderly patients have difficulties submitting to the demanding postoperative rehabilitation process necessary after this procedure.[43]

Cofield,[12] in 1979, reported on 12 patients who were an average age of 50 years with rotator cuff tears and who had their shoulder fused. Two of 12 patients developed nonunion and 6 of 12 necessitated a second operation for acromioclavicular pain, nonunion, or proximal migration.

It seems proper to apply Arntz et al.'s[2] and others'[11] recommendations and to consider using arthrodesis in irreparable rotator cuff tears, only in combination with irreparable deficiencies of the deltoid muscle, or in the younger patient with demands for substantial strength at low angles of flexion.

Resection arthroplasty generally results in poor function but is reliable for improvement of pain. Resection arthroplasty yields an unstable, nonfunctional shoulder. In the elderly patient with severe bone loss and massive cuff deficiency often seen with chronic dislocation, resection arthroplasty may be one of the few surgical options to offer the patient pain relief. It should not be entirely abandoned as an option in very selected cases.

Constrained Total Shoulder Arthroplasty

The lack of joint stability led researchers and surgeons in the early 1970s and early 1980s to use constrained designs of total shoulder arthroplasty in cases of rotator cuff tear arthropathy to create a fixed fulcrum for deltoid action (Fig. 23-3). Although initial reports showed good clinical results, longer-term follow-up showed a high percentage of glenoid component loosening and material failure of implants. Post[49] reported that glenoid component radiolucent lines appeared in 30% of primary constrained arthroplasties used in rotator cuff tear arthropathy. Lettin[39] and colleagues reported on 10 of 49 shoulders that developed relatively early glenoid component loosening. It appeared

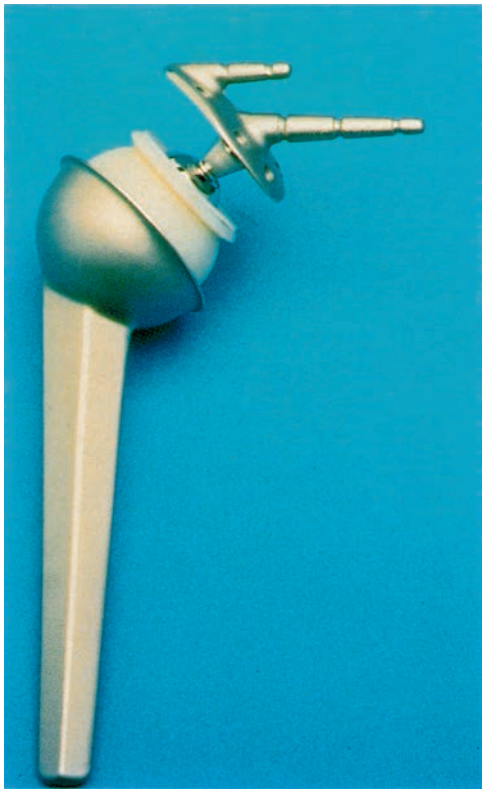


Figure 23-3 Constrained designs of a fixed fulcrum used in the 1970s and early 1980s.

that the inherent constraints in the prosthesis along with a lateral center of rotation transferred strong shear forces to the glenoid component–bone interface. The increased shear forces combined with the osteopenic nature of the bone and the small surface area of the interface led to the glenoid loosening. Given the unacceptable high failure rates, the use of these early types of fully constrained, fixed fulcrum, total shoulder construct for rotator cuff–deficient shoulders was abandoned. The failure of these early designs of constrained arthroplasty resulted from a center of rotation that was lateral to the glenoid component–bone interface.

Semiconstrained Shoulder Arthroplasty

Another prosthetic design used in the early years of shoulder arthroplasty was to enlarge the glenoid component by adding to it a superior hood, which was intended to resist superior migration of the humeral head. Amstutz[1] reported on a subset of 10 hooded glenoid Dana total shoulder arthroplasties performed on patients with massive rotator cuff tears. Pain decreased substantially; however, range of movement did not improve. Two of the 10 patients needed revision surgery. Neer[43] reported on his experience in this type of total shoulder arthroplasty. He used hooded glenoid components in 4 of 11 of the rotator cuff–deficient rheumatoid group and in 8 of 16 of the rotator cuff arthropathy group (Fig. 4). Specific conclusions as to the use of this semiconstrained construct were not made. Recently, Nwakama[44] reported on seven arthritic shoulders with massive rotator cuff tears that underwent total shoulder replacement using a semiconstrained hooded Neer prosthesis. Although pain level was improved in all patients, active motion was actually decreased. Five of six patients had anteroposterior instability and three of six had complete radiolucent lines on radiographic evaluation. Two shoulders necessitated revision surgery for subluxation or glenoid loosening. Orr and colleagues,[45] who analyzed the biomechanical behavior of this construct by using finite element analysis, presented additional support to the relatively high glenoid loosening rate. They found that the addition of superior constraint to the glenoid component generated increased stresses at the glenoid component–bone interfaces, making it more susceptible to early loosening. The lack of active motion improvement combined with early glenoid loosening has discouraged the use of this design.

Unconstrained Shoulder Arthroplasty

Two different types of unconstrained shoulder arthroplasty have been used for RCDA: total shoulder arthroplasty using an anatomic design with no additional built-in constraints and a replacement of the humeral head without use of a glenoid component (hemiarthroplasty).

Unconstrained Total Shoulder Arthroplasty

Neer et al.[43] published in 1982 their experience with prosthetic replacement of 273 shoulders, of which 16 were diagnosed as rotator cuff tear arthropathy. Eight RCDA shoulders received an unconstrained total shoulder prosthesis, while the rest were treated with a semiconstrained (hooded) implant. Eleven patients had a follow-up of more than 24 months. Using Neer's "limited goals rehabilitation" criteria,[43] 10 out 11 patients had successful results. Although 30% of shoulders and 33% of the rotator cuff tear arthropathy shoulders had glenoid lucent lines (less than 1 mm), clinically detected loosening was not found in any of these 273 shoulders. Lohr and Cofield[40] found unconstrained total shoulder replacements to provide better pain relief than hemiarthroplasty in 22 rotator cuff–deficient shoulders. However, a high rate of radiologic and clinical loosening was noted in their patients. Barrett et al.,[4] in a prospective study in 1987, reported on 44 patients who underwent total shoulder arthroplasty. Nine of the patients had a massive tear of the rotator cuff at the time of surgery. Glenoid loosening was found in four patients, all of which had previous massive rotator cuff tear. Three of these patients necessitated surgical revision. It was hypothesized that the unstable shoulder joint causes

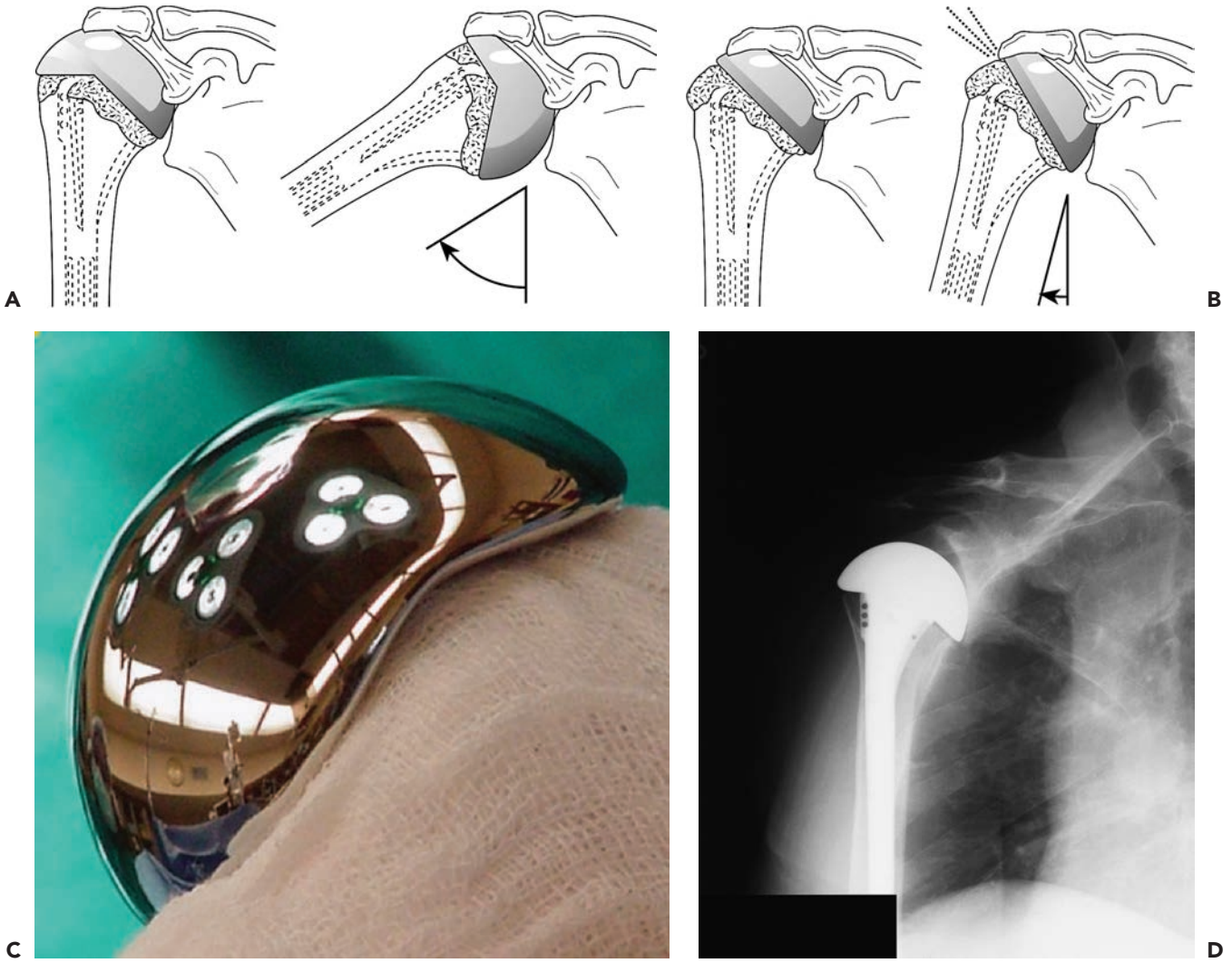


Figure 23-4 Hooded glenoid components.

repeated exaggerated superior translation of the humeral head on the glenoid component, allowing joint compression forces to be put on the superior glenoid margin rather than in the center of the glenoid, leading to loosening. Franklin et al.[26] tried to correlate different clinical and radiographic parameters with glenoid loosening in patients undergoing total shoulder replacement in the presence of massive rotator cuff tear. They found that the amount of superior migration of the humeral head was closely correlated with an increased glenoid loosening rate. They emphasized the eccentric forces applied by the unstable, translated humeral head on the glenoid component resembling the movement of a "rocking horse," leading to the loosening of the glenoid component. Due to these high loosening rates found relatively early in the postoperative course, the use of an unconstrained total shoulder implant has been disfavored.

Humeral Head Replacement

The relatively early loosening of the glenoid component has led surgeons to propose hemiarthroplasty with replacement of only the humeral head as a solution for RCDA shoulders. Using this approach, significant pain reduction in 47% to 86% of patients and significant, yet variable, gain in forward elevation have been reported at an average follow-up of 2 to 5 years.[24,48,51,68,72] Taking into account Neer's limited-goal criteria,[43] successful results have been achieved in 63% to 83% of patients. Pollock et al.[48] found similar pain relief and better forward elevation gains in hemiarthroplasty versus total shoulder replacement in 30 rotator cuff–deficient arthritic shoulders.

The inherent superior instability of hemiarthroplasty in these patients, where no significant soft tissue support is available, has nourished several debates concerning the

implant design and associated procedures needed to stabilize these shoulders.

Humeral Component Size

One of the first solutions to gain better stability was the use of larger humeral head components. The rationale was that a large humeral component can articulate better with the large "acetabulum" (consisting of the superior glenoid and undersurface of coracoacromial arch), therefore creating a relatively stable and more congruent joint. However, potential disadvantages to the use of large humeral components included overstuffing of the joint, which led to increased joint reaction forces, accelerated bone resorption of both the glenoid and the acromion, pain, and eventually further instability.[51] In addition, with the use of a larger humeral head, any attempt to augment or partially repair the deficient rotator cuff became much more difficult. The disadvantages of a large-diameter humeral component and the need for an additional prosthetic contact area with the acromion and coracoacromial arch have led to the use of a specially designed humeral component: the cuff tear arthropathy head (CTA head). This component has an anatomically sized head (lateral offset and radius of curvature) but adds a smooth extension of the articular surface that covers the greater tuberosity, which in RCDA does not have an attached cuff and has an irreparable cuff. This extension of the superior articular surface (Fig. 23-5) articulates with the coracoacromial arch. The CTA design enlarges the joint contact area without increasing the size of the head (lateral offset and radius of curvature) and thereby will not "overstuff" the joint. A recently published study evaluated the results of 60 rotator cuff tear arthropathy shoulders that have undergone humeral hemiarthroplasty using a CTA head prosthesis. None of the patients had an anterosuperior escape of the humeral head on forward flexion prior to surgery. Using Neer's limited-goals criteria, after 2 years of follow up, 89% had a successful result (i.e., significant improvement in range of motion and pain reduction).[63]

Reconstruction and Augmentation of the Rotator Cuff Elements

Pollock,[48] Cantrell and Burkhead,[10] and DiGiovanni[18] have advocated attempts to repair or augment the torn rotator cuff tendons. One of the options suggested is the transfer of the subscapularis tendon to a more superior position as a method for superior stabilization of the humeral head.[43] This option is limited obviously to those patients with a functional, unshortened subscapularis tendon. However, others reported satisfactory results without any attempt to repair or augment the torn supraspinatus and infraspinatus tendons. Williams and Rockwood[68] published satisfactory results with pain relief for 18 of 21 patients using no cuff reconstruction with balancing of the remaining cuff muscles by fine tuning the humeral head component size. Zuckerman et al.,[72] who also did not try to repair or

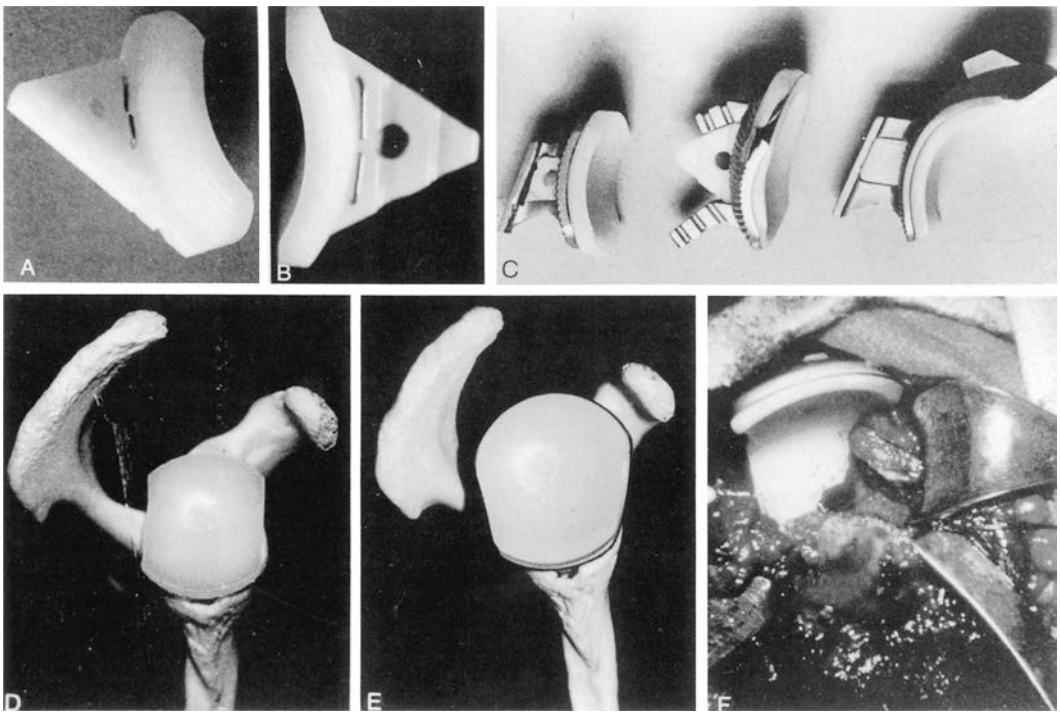


Figure 23-5 This extension of the superior articular surface articulates with the coracoacromial arch.

augment the torn supraspinatus and infraspinatus tendons, published similar results. Sanchez-Sotelo et al.[51] reported on 33 shoulders in 30 patients who received a hemiarthroplasty for the diagnosis of rotator cuff arthropathy. Medium-sized humeral head components were used in most shoulders. Statistically significant correlation was found between attempts to partially repair the rotator cuff tendons and postoperative, clinically significant, anterior instability. The authors explained this association with the creation of a muscle force imbalance by their repair attempts.

Augmentation of Superior Bone Elements

The importance of maintaining the coracoacromial arch in RCDA cannot be overemphasized.[2,51,68,72] The deficiency in superior bony restraints, secondary in most cases to previous surgical decompression of the subacromial space, creates an extremely difficult scenario. Sanchez-Sotelo et al.[51] reported poorer results and increased susceptibility to anterosuperior instability in a subgroup of RCDA patients that had had previous surgical coracoacromial arch decompression. Several solutions have been proposed for restoring these superior bony restraints. Wiley[67] described the use of an autologous tricortical iliac bone graft that was fixed between the coracoid and acromion in cases where the coracoacromial arch was compromised by previous subacromial decompression (Fig. 23-6). Engelbrecht and Heinert[21] introduced another technique in which augmentation of the glenoid rim was achieved by fixing an autologous bone graft to the superior glenoid rim, thus deepening

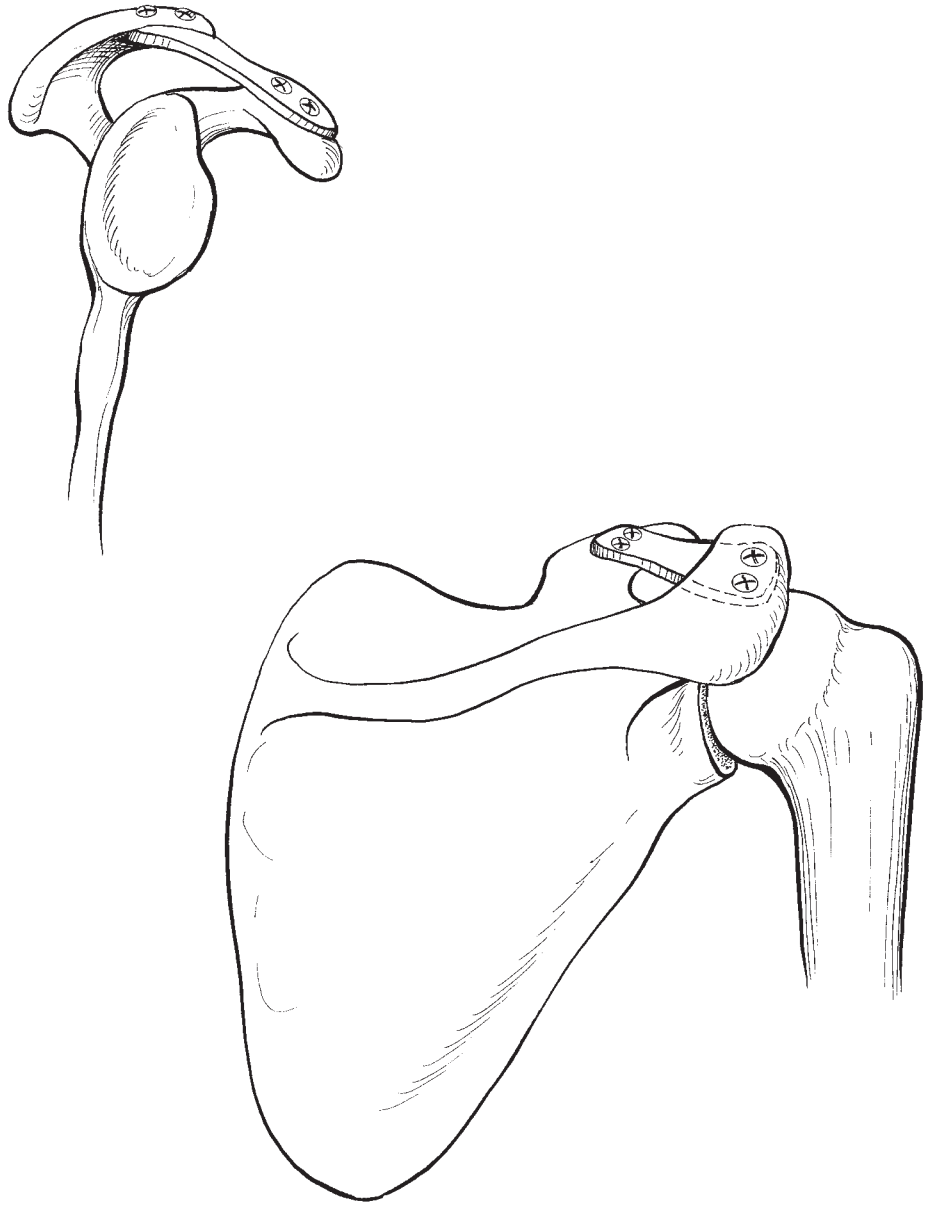


Figure 23-6 Wiley tricortical iliac bone graft that was fixed between the coracoid and acromion. (Reproduced with permission from Wiley AM. Superior humeral dislocation. A complication following decompression and debridement for rotator cuff tears. *Clin Orthop* 1991;Feb(263):135–141.)

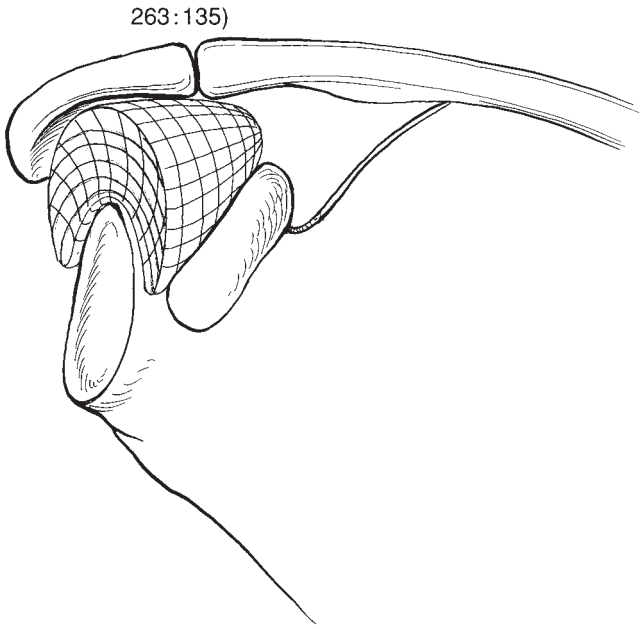


Figure 23-7 Engelbrecht augmentation of the glenoid rim autologous bone graft to the superior glenoid rim, thus deepening and widening the actual articulating surface of the glenoid. (Reproduced with permission from Engelbrecht E, Heinert K. More than ten years experience with unconstrained shoulder replacement. In: Kolbel, Helbig, Blauth, eds. *Shoulder replacement.* Berlin: Springer-Verlag, 1987:85–91.)

and widening the actual articulating surface of the glenoid and counteracting superior-directed forces applied by the humeral head (Fig. 23-7).

Bipolar Humeral Prosthesis

Bipolar humeral component design was presented by Swanson et al.[60] in 1975 as a solution for patients with advanced glenohumeral arthritis and poor rotator cuff tissue as part of a total shoulder design. The rational was to decrease prosthetic contact forces by a larger "articular" surface, and by the secondary motion possible within the bipolar humeral prosthesis itself. At a follow-up of 5 years, pain relief was rated good to excellent in 31 of the 35 shoulders operated. Arredondo and Worland[3,69] reported their results using a newer, lower-profile, bipolar design that allows for less bony resection during insertion. At a mean follow-up of 3.1 years (range 2 to 6 years), satisfactory results were found in 92% of their patients using the limited-goals criteria, including both pain relief and improved range of motion. The authors also reported prosthetic birotational motion (head-shell and shell-glenoid motion), which persisted in all shoulders. Vrettos et al.,[64] using the same bipolar prosthesis, reported less favorable results. Six of the seven patients reported moderate to severe pain and were unhappy with the results. No glenohumeral or intrinsic bipolar motion was found in radiographs taken in different angles of abduction. A recent

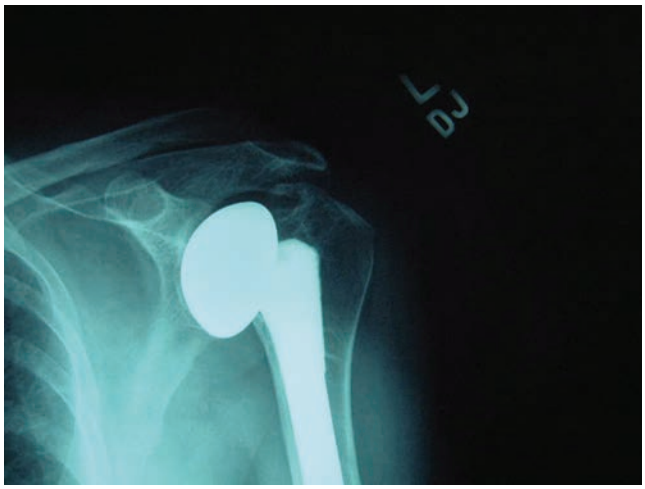


Figure 23-8 Bipolar hemiarthroplasty concerns of "overstuffing" the shoulder and secondary glenoid wear.

report by Duranthon et al.[19] on 13 RCDA shoulders with a mean follow up of 28 months revealed good pain relief with less than satisfactory range-of-motion improvement. Significant glenoid wear was found in three of the seven shoulders that were observed 2 years after surgery.

To date, bipolar hemiarthroplasty has not gained popularity. The reasons are the concerns of "overstuffing" the shoulder and secondary glenoid wear (Fig. 23-8), rupture of the subscapularis tendon due to the vertical orientation of the component (Fig. 23-9), the effect of polyethylene wear,[33] and the relatively short follow-up reported.

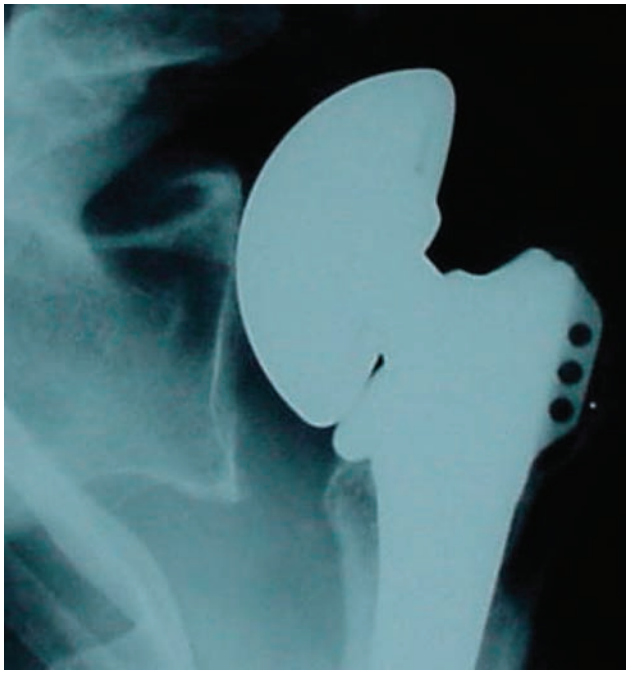


Figure 23-9 Vertical orientation of the bipolar prosthesis.

The inherent superior instability of hemiarthroplasty in RCDA shoulders has been at least partially counteracted using the aforementioned methods with relatively good clinical results. One of the still existing concerns is the durability of this "stability" achieved with the use of hemiarthroplasty in cuff-deficient shoulders. Sanchez-Sotelo et al.[51] reported on 33 shoulders in 30 patients that had a hemiarthroplasty for the diagnosis of rotator cuff arthropathy with an average follow-up of 5 years (range 2 to 11 years). Most shoulders received medium-sized humeral heads. Progressive superior migration and progressive glenoid bone loss were reported in 8 of 33 shoulders and acromial bone loss was reported in 16 of 33 shoulders, leading to acromion fracture in two patients. Poorer results and more severe anterosuperior instability were found in a subgroup of patients that had previous surgical coracoacromial arch decompression.

To summarize, hemiarthroplasty is a good surgical solution for a cuff-deficient arthritic shoulder with a contained humeral head having an intact "acetabularized" coracoacromial arch providing a stable fulcrum for rotation of the humeral head. Hemiarthroplasty is also ideally suited for a cuff-deficient arthritic shoulder with an irreparable rotator cuff but with the remaining cuff and deltoid being sufficient to have active elevation of the shoulder to approximately shoulder height prior to replacement. Hemiarthroplasty, although not a perfect solution, provides good pain relief and moderate range-of-motion gain for most patients, which is relatively long-lasting. Due to the high rates of glenoid component loosening, unconstrained total shoulder replacement should not be considered for rotator cuff–deficient arthritic joints. No study has demonstrated an advantage of rotator cuff partial repair or augmentation in these circumstances over simple débridement. A competent coracoacromial arch is a keystone for successful results and therefore should not be damaged during the surgical procedure. In cases of preexistent coracoacromial deficiency, the use of reversed shoulder prosthesis should be considered. Progressive bone loss and progressive anterosuperior instability could lead to a growing number of unsatisfactory results in long-term follow-up periods (more than 5 years).

In the authors' experience, the ideal surgical technique for hemiarthroplasty for RCDA preserves the coracoacromial arch, deltoid, and remaining cuff without detachment of any of these intact structures. The goals of the surgery are to:

1. Remove all inflamed and pathologic bursae tissues including an abnormal long head of the biceps
2. Remove all humeral osteophytes on the greater tuberosity
3. Replace the head with an anatomically sized humeral head if there is not significant medial glenoid erosion (less than 1 cm) or a larger head if there is significant glenoid bone loss
4. Correct eccentric glenoid bone loss of removal of the high side and create a smooth concave surface for the humeral head in the position that it will articulate
5. Not dissect the coracoacromial arch
6. Not dissect the deltoid origin
7. Not detach any of the remaining intact portion of the rotator cuff

To achieve these goals, a deltopectoral approach is used, keeping the coracoacromial ligament intact. The arm is placed in extension, adduction, and external rotation to dislocate the humeral head from the interval between the deltoid and pectoralis major. All abnormal bursae tissue is removed from around the humeral head and under the coracoacromial arch, taking care to preserve the coracoacromial ligament and remaining attached rotator cuff tendon (Fig. 23-10). A large Darrach is placed into the joint

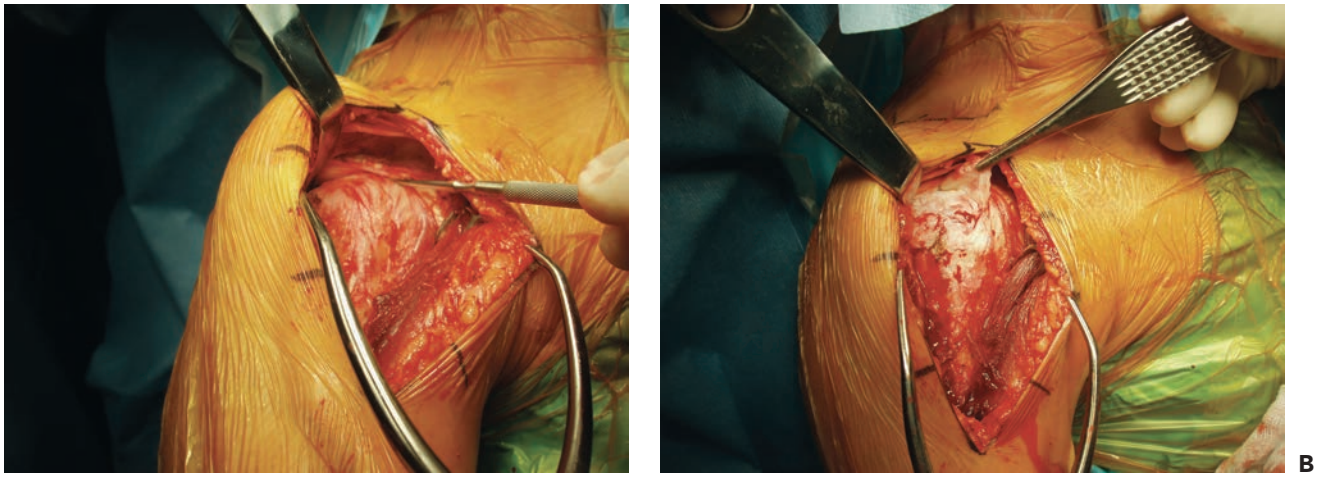


Figure 23-10 **(A)** All abnormal bursae tissue is removed from around the humeral head and under the coracoacromial arch, taking care to preserve the coracoacromial ligament and remaining attached rotator cuff tendon.

through the rotator cuff defect and between the humeral head and the glenoid to further lever the humeral head from the joint (Fig. 23-11). If the long head of the biceps is intact, then it is cut and tenodesed to local soft tissues distal to the bicipital groove. A narrow reverse Homans or bunion-type retractor is placed beneath the remaining intact portion of the anterior and posterior parts of the rotator cuff (Fig. 23-12). This exposes the humeral head from the superior aspect through the existing defect in the humeral head. Using a power saw, a free hand cut is made approximately 5 mm distal to the top of the greater tuberosity and perpendicular to the humeral shaft, thereby cutting off the top of the humeral head, exposing the cancellous bone and lending easy access to the humeral diaphyseal canal (see Fig. 23-12). Using the smallest humeral medullary canal reamers, the canal is broached, and with larger reamers the canal enlarged. A trial broach is then placed into the reamed canal until the collar of the broach

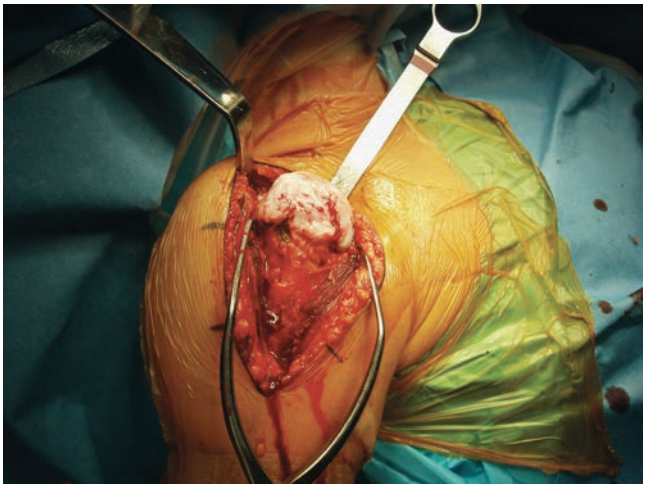
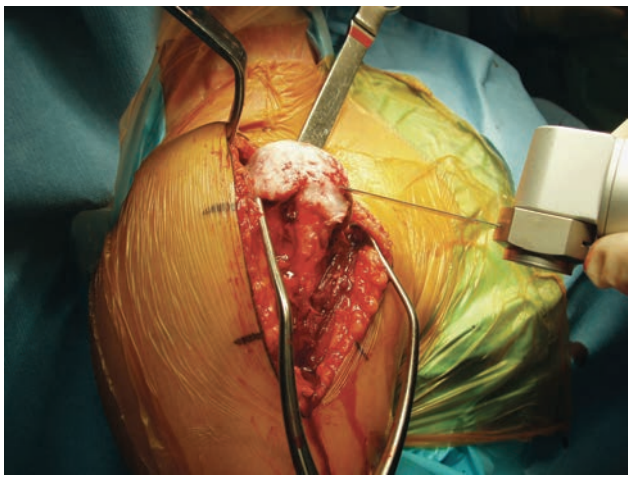
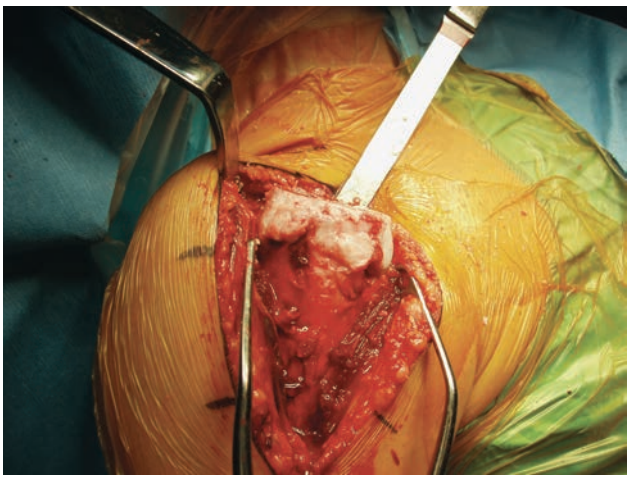


Figure 23-11 A large Darrach is placed into the joint through the rotator cuff defect and between the humeral head and the glenoid to further lever the humeral head from the joint.



A



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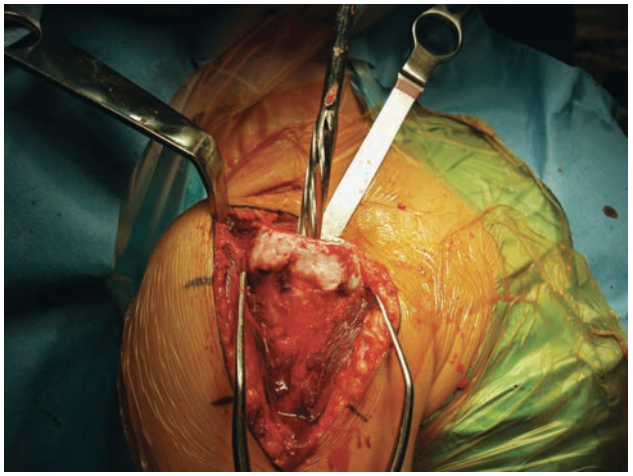


C

Figure 23-12 A narrow reverse Homans is placed under the remaining rotator cuff to expose the humeral head. **(A–C)** Using a power saw, a free hand cut is made approximately 5 mm distal to the top of the greater tuberosity and perpendicular to the humeral shaft, thereby cutting off the top of the humeral head, exposing the cancellous bone and lending easy access to the humeral diaphyseal canal.

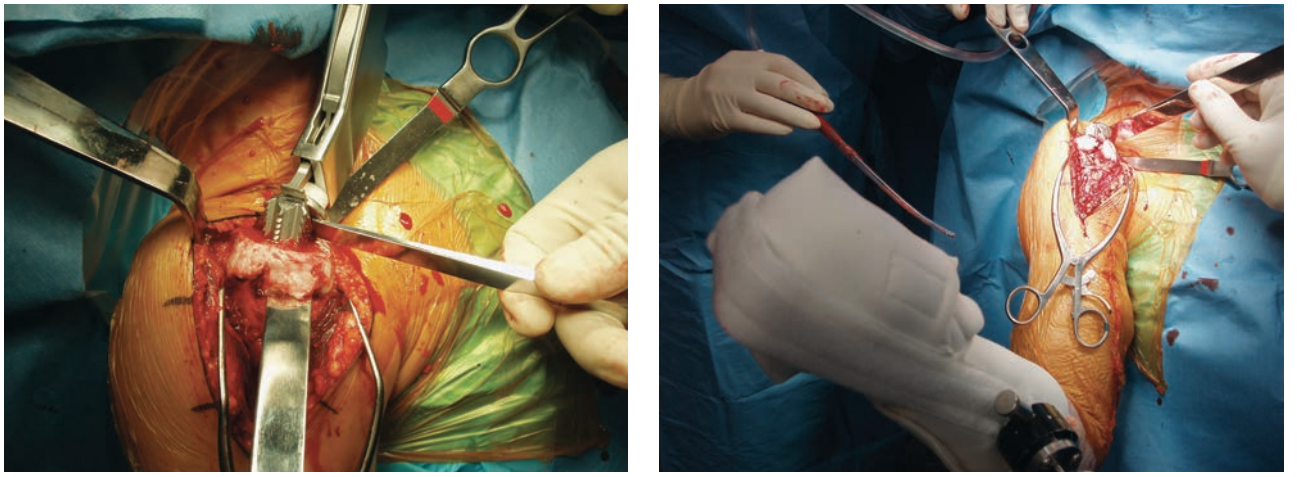


Figure 23-13 A trial broach is placed into the reamed canal until the collar of the broach hits the cut superior surface of the remaining humeral head. The version can be assessed by the plane of the back surface of the collar of the prosthesis and the long axis of the forearm as shown by the two osteotomes. Retroversion is measured from the flat back face of the broach collar to the long axis of the forearm with the elbow at 90 degrees of flexion.

hits the cut superior surface of the remaining humeral head. The broach is rotated to a retroverted position to be approximated 10 degrees more in retroversion than anatomic. Retroversion is measured from the flat back face of the broach collar to the long axis of the forearm with the elbow at 90 degrees of flexion (Fig. 23-13).

Alternatively or additionally, retroversion can be judged by the anatomic neck of the exposed humeral head. The blade of the oscillating power saw is then made parallel to the back surface of the broach collar and the remaining portion of the head is cut at the level of the anatomic neck, taking care to keep the head cut within the joint, thereby preserving the remaining intact cuff and protecting the axillary nerve (Fig. 23-14). The head fragment is removed and the inferior part of the head cut is palpated. Any remaining inferior humeral head osteophyte is removed with a half-inch curved osteotome and/or rongeur. In almost all cases, preoperative passive forward elevation is at least to 120 degrees and an inferior capsular release is not needed, nor is there much osteophyte present on the humeral head in classic CTA. If a capsular release is needed, then it is performed on the glenoid side of the capsule. A Fukuda or similar humeral head retractor is used to retract the humerus and the glenoid is inspected; if it is irregular it can be reamed with a smooth convex reamer or burred by hand to a smooth concave surface. The goal of glenoid preparation is to preserve the thickened cortical-type bone when possible and remove only irregular surfaces and prominent areas of bone that represent the high side of the glenoid when there is eccentric bone loss. All degenerative soft tissue or hypertropic synovium is removed and the joint is irrigated. The proximal humerus is again exposed as previously described. The humerus is then prepared as described by the manufacturer and the broach is inserted. It should be noted that all prosthetic instrument sets are designed for placement of the trials and final implant with a detachment of the subscapularis. When performing the surgery by the method described in this chapter, the instruments may not fit as well and may need to be modified for optimal use. When the trial stem is inserted, the trial heads may be placed and a trial reduction and range of motion with stability testing performed. If a DePuy CTA head is used, the additional cut in the tuberosity is made from the trial head or using the cutting guides

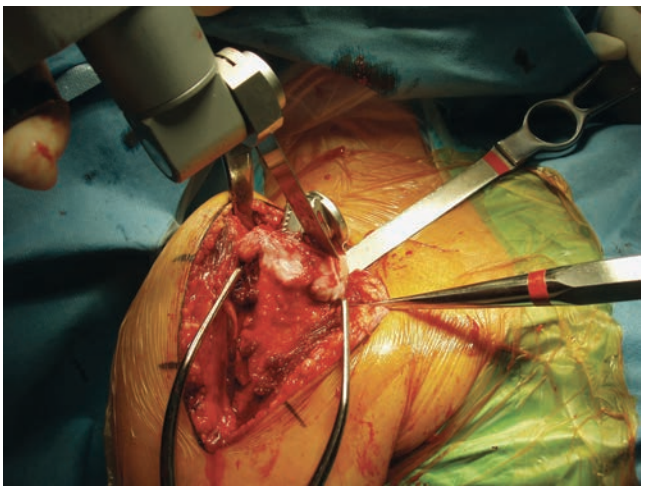


Figure 23-14 The blade of the oscillating power saw blade is made parallel to the back surface of the broach collar and the remaining portion of the head is cut at the level of the anatomic neck, taking care to keep the head cut within the joint, thereby preserving the remaining intact cuff and protecting the axillary nerve.

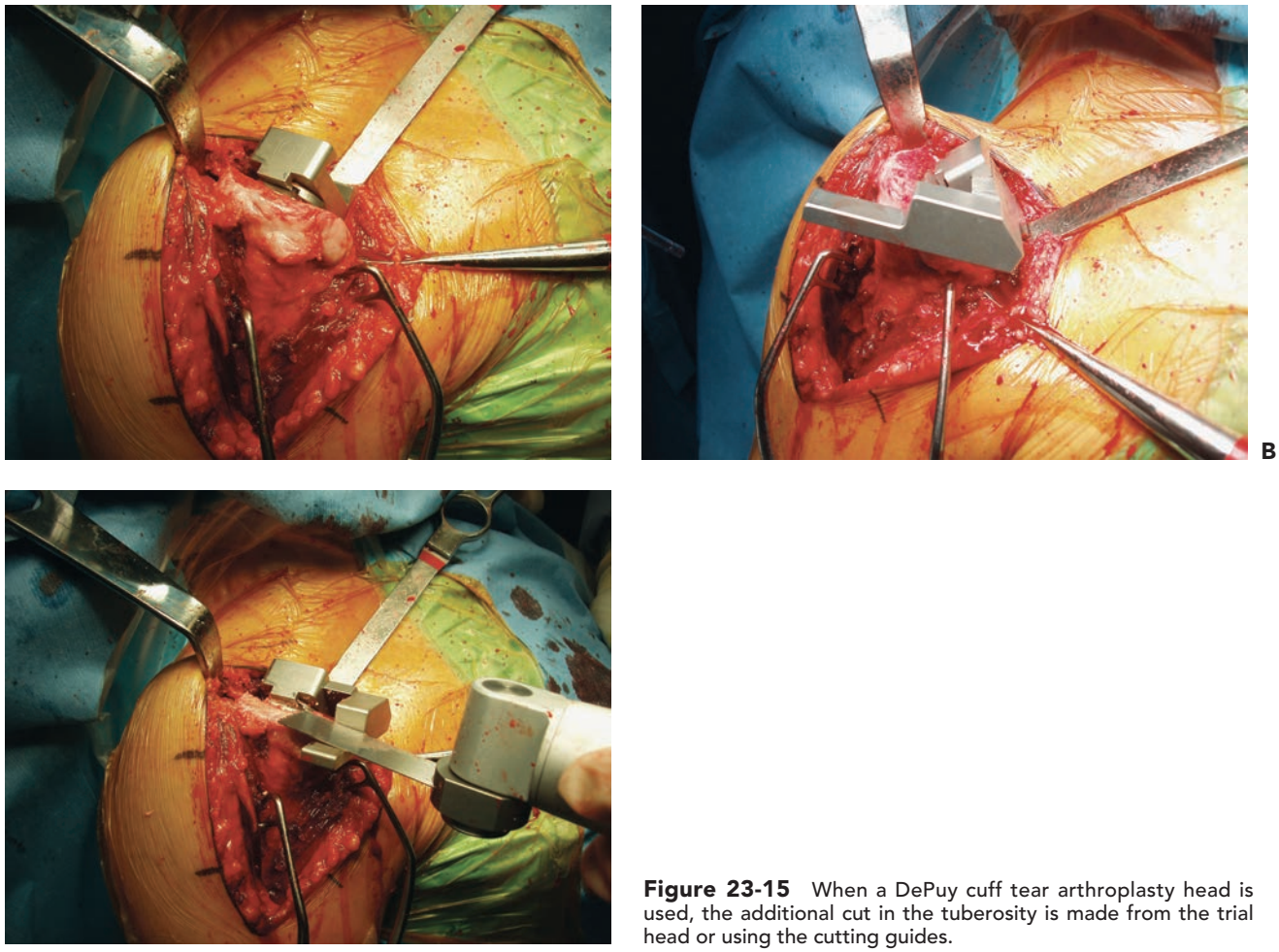


Figure 23-15 When a DePuy cuff tear arthroplasty head is used, the additional cut in the tuberosity is made from the trial head or using the cutting guides.

(Fig. 23-15). After final preparation of the bone cuts and selection of the prosthetic, the final components are assembled and inserted with or without cement depending on the stem design, bone quality, and stability of the stem. Impaction bone grafting can in some cases be performed using either allograft cancellous bone or the patient's resected humeral head in the case of primary replacements. Impaction grafting can increase the stability of the uncemented stem and avoid the use of bone cement in many cases. The final prosthetic should sit directly and precisely on the cut surface of the humerus (Fig. 23-16). With proper surgical technique and rehabilitation, good results can be anticipated in most patients (Fig. 23-17).

Reversed Shoulder Arthroplasty

The term "reversed shoulder" describes a ball-and-socket arthroplasty design where the convex surface is on the glenoid side and the concave surface is on the humeral side. The reverse shoulder systems currently used have a complete conformity of the articular surfaces (equal radii of curvature) and a high degree of constraint (high wall height), thereby enforcing ball-in-socket kinematics by virtue of the design of the prosthetic. The use of reversed shoulder arthroplasty was suggested and tried almost three decades ago.[9,23,46] However, the use of a small glenoid ball with a center of rotation lateral to the glenoid component fixation on the bony glenoid vault in a highly constrained prosthesis resulted in a high rate of prosthetic failure due to loss of glenoid component fixation (Fig. 23-18). Grammont,[29] approximately 15 year ago, renewed the interest in the reversed design construct, especially for use in rotator cuff–deficient arthritic shoulders. In contrast to previous reversed prostheses, Grammont designed a more shallow humeral socket that articulated with a larger radius of curvature hemispherical glenoid component. In this design the center of rotation of the hemisphere was near the component's interface with the bony glenoid. This design produced less interprosthetic constraint, and the small lateral offset (absence of neck) reduced the shear forces at the point of fixation of the glenoid component to the bone, making failure of glenoid fixation minimal. Shifting the glenohumeral center of rotation medially and distally to its original position in the natural shoulder creates a stable

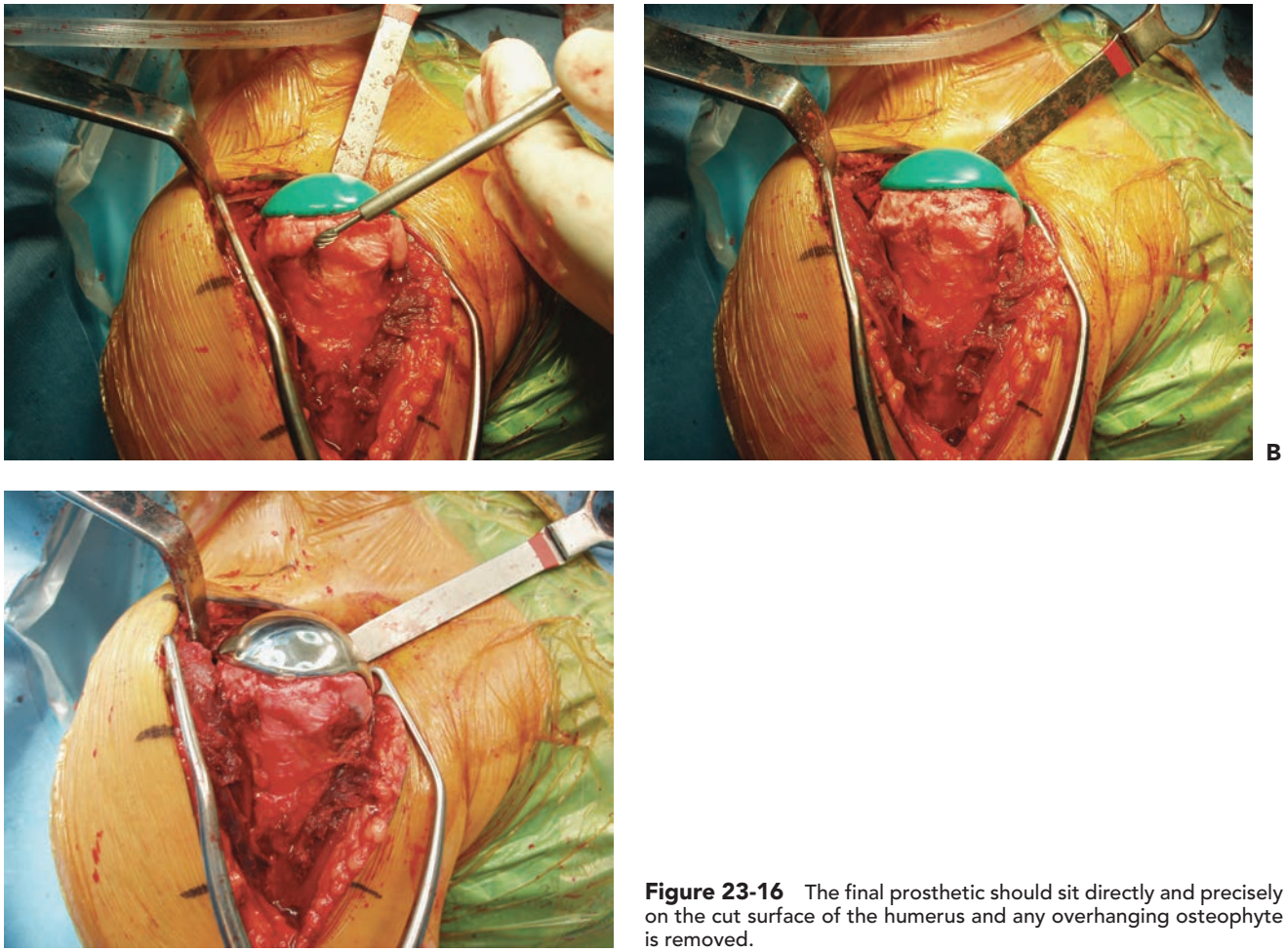


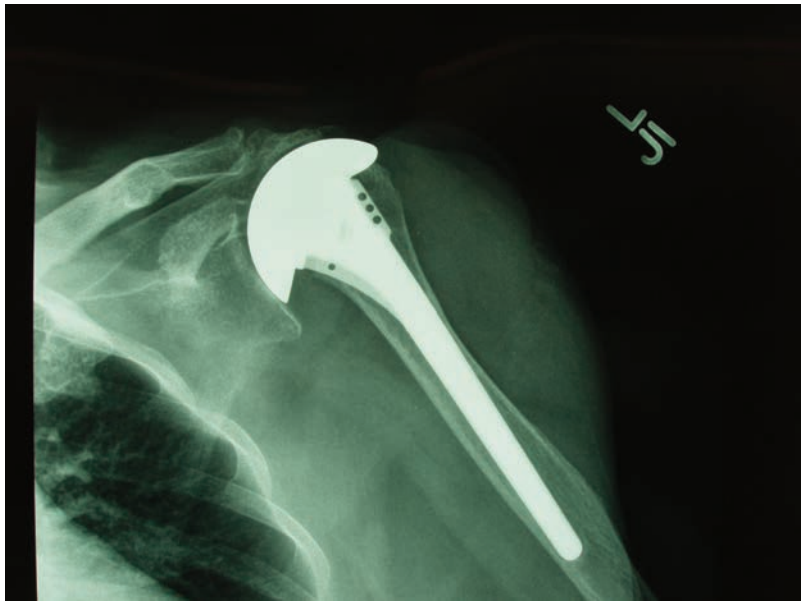
Figure 23-16 The final prosthetic should sit directly and precisely on the cut surface of the humerus and any overhanging osteophyte is removed.

fulcrum and longer moment arm for the deltoid muscle, making it more efficient.[14] The larger spherical glenoid prevents the "rocking horse" effect of the humeral head on the glenoid (Fig. 23-19).

The inherent stability and mechanical advantage of the reversed shoulder prosthesis was compared to the unconstrained shoulder hemiarthroplasty in the absence of functional rotator cuff tendons. Using a computerized model, De Wilde et al.[16] found that the reversed shoulder prosthesis design medializes the rotation center, stretches the deltoid muscle, increases the deltoid lever arm, and results in a significantly more powerful abduction of the shoulder than an anatomic hemiarthroplasty.

The Grammont Delta reverse shoulder prosthesis has been used for almost 13 years in Europe. Several studies have evaluated this prosthesis for the use of rotator cuff–deficient arthritic joint. Bouttens and Nérot[8] reported an average 5-year follow-up on 39 patients with rotator cuff tear arthropathy, Favard et al.[22] on 15 patients with a 25-month follow-up, Sirveaux et al.[58] on 80 shoulders with a 44-month follow-up, Boileau et al.[6] on 21 patients with a 40-month follow-up, and Werner et al.[66] on 58 patients with a 38-month follow-up, demonstrating a significant improvement in pain and significant improvement in active motion. In addition, other surgeons[7,32,50] showed significant pain reduction with no or only minimal pain in most patients and a significant improvement of active forward flexion.

Seebauer et al.[52–55] presented their results with reversed prosthesis Delta3 in 57 patients with RCDA with a mean age of 71 years. Thirty-six patients were available for complete follow-up. The surgical approach used was anterosuperior in 90% and deltopectoral in 10%. After a mean follow-up of 42 months (range 27 to 68), they found that 98% reached Neer's limited-goals criteria. An age- and sex-correlated mean Constant score[13] reached 94% at last follow-up. Nearly all patients reported complete pain relief at rest and for minor activities. Sirveaux et al.[58] retrospectively studied 80 Grammont reversed shoulder prosthesis with a mean follow-up of 44 months. The mean Constant score had increased from 22.6 to 65.6. Ninety-six percent had no or only minimal pain, and the mean active forward elevation increased from 73 to 138 degrees.



A

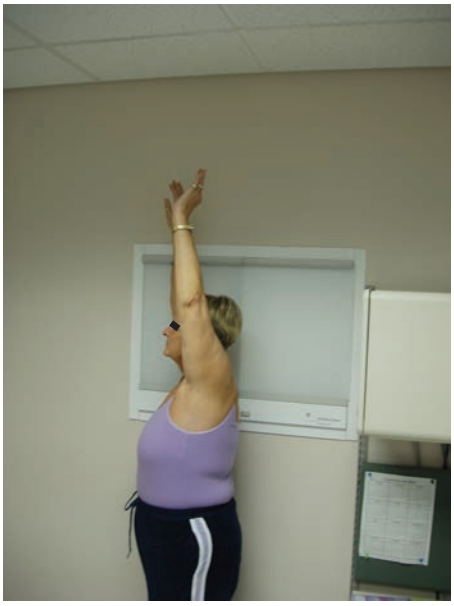


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Figure 23-17 With proper surgical technique and rehabilitation, good results can be anticipated in most patients.

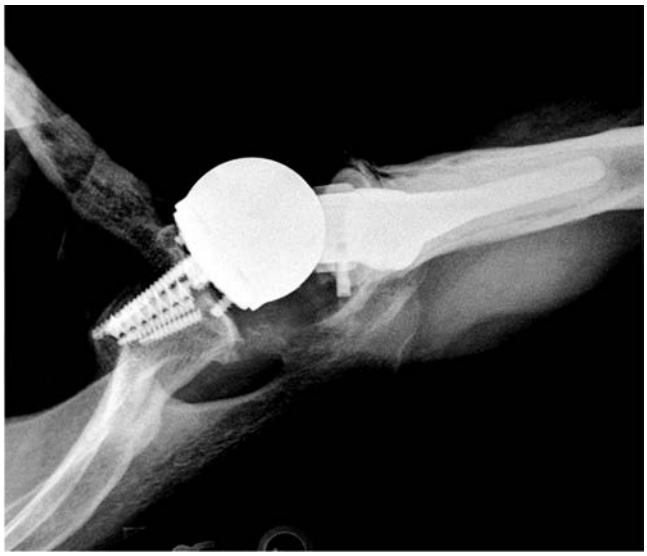


Figure 23-18 The use of a glenoid ball with a center of rotation lateral to the glenoid component fixation on the bony glenoid vault in a highly constrained prosthesis can result in prosthetic failures due to loss of glenoid component fixation. (From Frankle et al. The reverse shoulder prosthesis for glenohumeral arthritis with severe rotator cuff deficiency. *J Bone Joint Surg* 2005;87A:1702.)

There are, however, several unsolved problems that need to be addressed with the Grammont reverse prosthetic:

1. It is difficult to assess the correct deltoid tensioning during surgery. Undertensioning (using undersized components) may lead to instability of the prosthesis and dislocations in up to 10% of cases[6,58,66] (Fig. 23-20). Overtensioning may lead to restricted range of motion and fatigue fractures of the acromion[6,66] (Fig. 23-21).
2. Inferior scapular notching has been shown to occur in 50% to 96% of cases.[6,7,15,50,58,66] It is a common radiographic finding at early follow-up. Boileau et al.[6] have performed fluoroscopic examinations that showed that scapular notching is a result of impingement of the medial aspect of the polyethylene humeral cup on the scapular neck inferiorly with the arm in the adducted position. It is probably the direct consequence of the absence of a prosthetic neck on the glenoid side and the lowering of the humerus. In many cases (as high as 45%) the notching can involve one or more of the glenoid fixation screws (Figs. 23-22 and 23-23). Werner et al.[66] showed that the notching stabilized in 50% of shoulders in 1 year. They also showed no correlation between notching and the clinical result. However, Sirveaux et al.[58] showed poorer clinical results when significant notching occurred. Due to the relative short follow-up periods in the studies published, the true significance of the notching still unclear.
3. Active external rotation range of motion or strength has not gained from the use of the reversed shoulder prostheses.[6,58,66] There are a couple of explanations: The amount of posterior deltoid that can be used to compensate for the absent external rotators is decreased

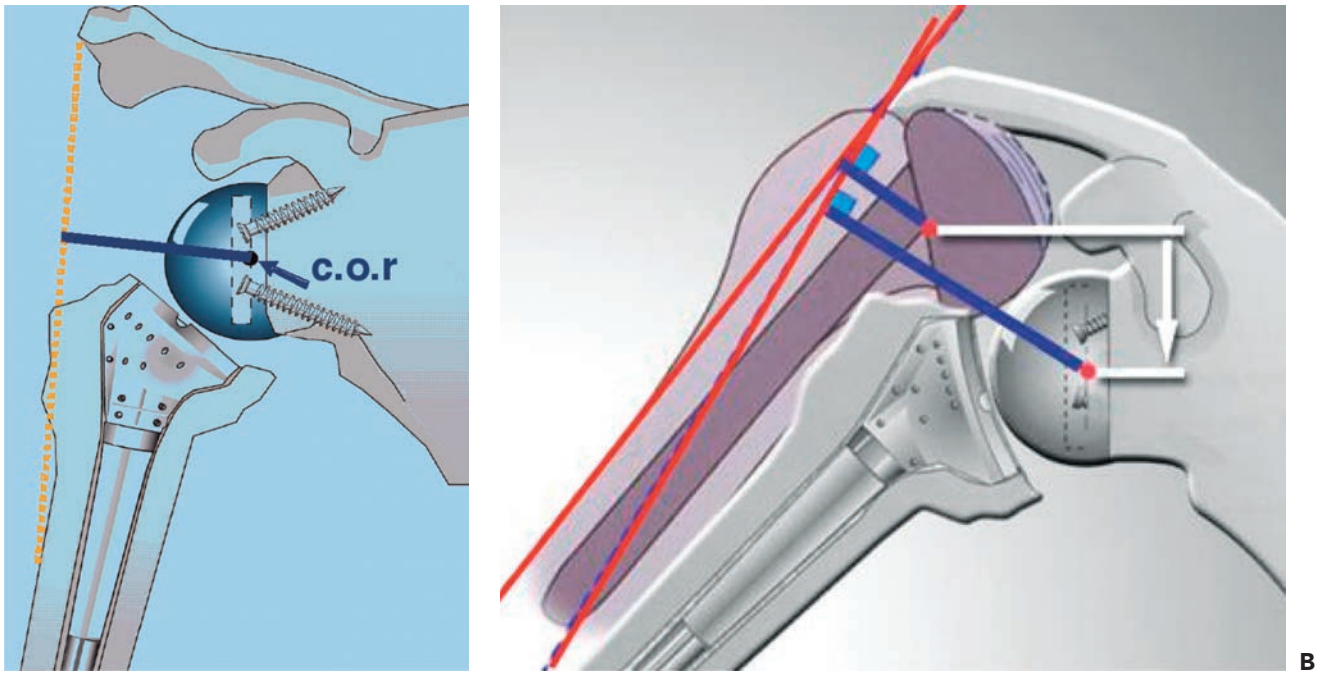


Figure 23-19 Shifting the glenohumeral center of rotation medially and distally to its original position in the natural shoulder creates a stable fulcrum and longer moment arm for the deltoid muscle, making it more efficient.

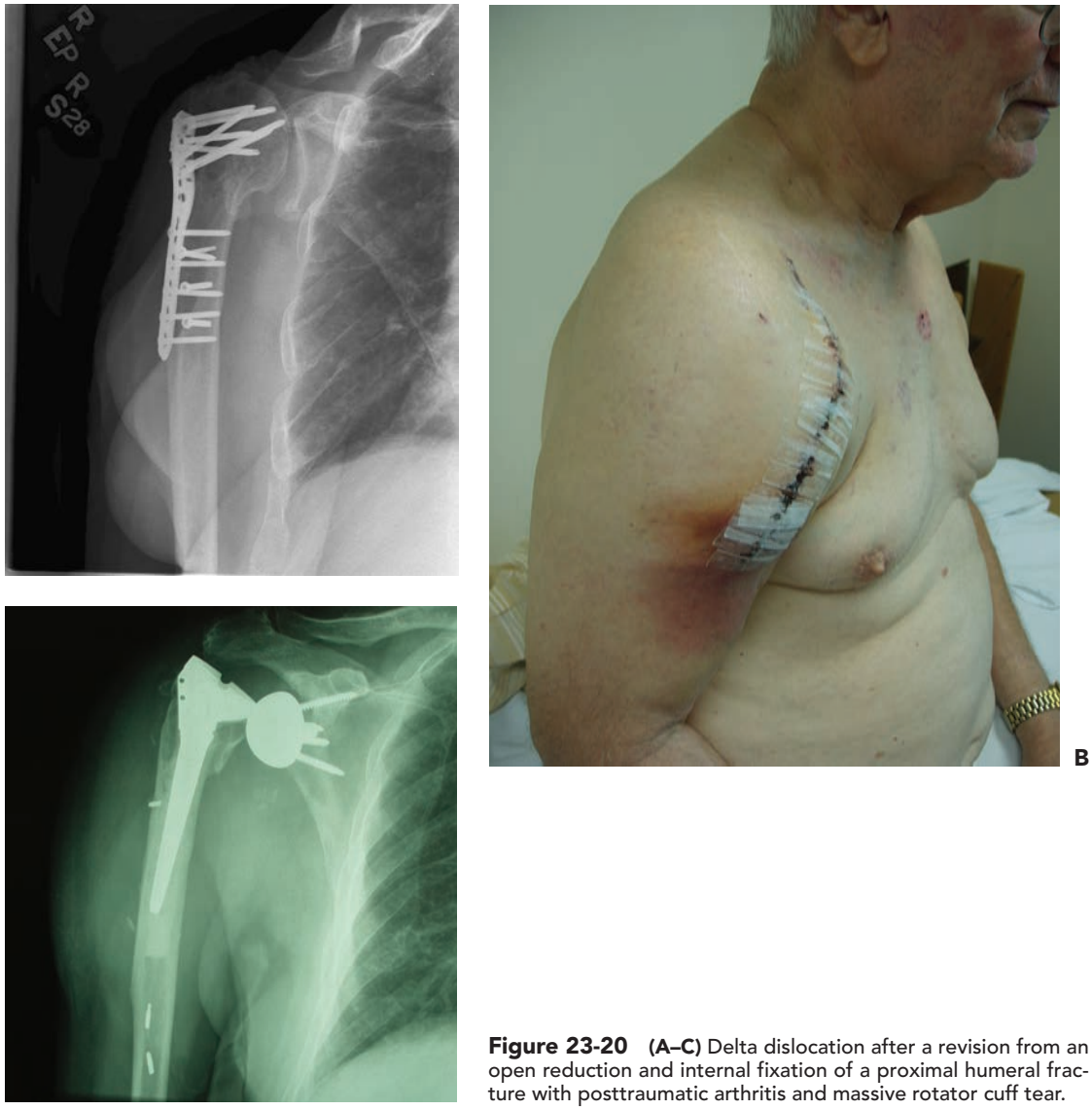


Figure 23-20 **(A–C)** Delta dislocation after a revision from an open reduction and internal fixation of a proximal humeral fracture with posttraumatic arthritis and massive rotator cuff tear.

because of this humeral medialization; and the remaining external rotators (i.e., teres minor) may also be slackened and less efficient because of this humeral medialization. The most important factor in the active external rotation range of motion is the status of the teres minor and any remaining portions of the infraspinatus. The extent of preoperative external rotation strength is an important factor associated with postoperative function and active forward elevation. The active external rotation after a reverse prosthesis is significantly better when the teres minor is intact than when it is absent or has fatty infiltration.[6,7,58,66]

4. There are relatively short follow-up periods. As stated above, most studies of the newer designs are of less than 4 years' follow-up. Larger patient number published studies with longer follow-up periods are necessary to evaluate the rate, progressiveness, and significance of glenoid loosening[20] and glenoid notching.

Other complications reported with the reverse shoulder prosthesis are glenoid and humeral radiolucent lines, glenoid component dissociation, hematoma formation, and infection. Reoperation rate was reported to be as low as 0% and as high as 33% in a recently published study.[66] The reverse shoulder prosthesis by Encore has less notching and a higher incidence of loss of glenoid component failure (12%) due to the presence of a lateral offset to the glenoid component.[25]

The reverse shoulder arthroplasty is a valuable procedure for the treatment of severe dysfunction of the shoulder due to rotator cuff tear arthropathy or in patients with severe humeral bone loss and cuff deficiency. However, because of the relatively high complication rate and the fact that there may be long-term complications that are not yet known, arthroplasty with this implant should be reserved as a salvage procedure for situations in which an acceptable clinical outcome cannot be expected with another treatment modality.

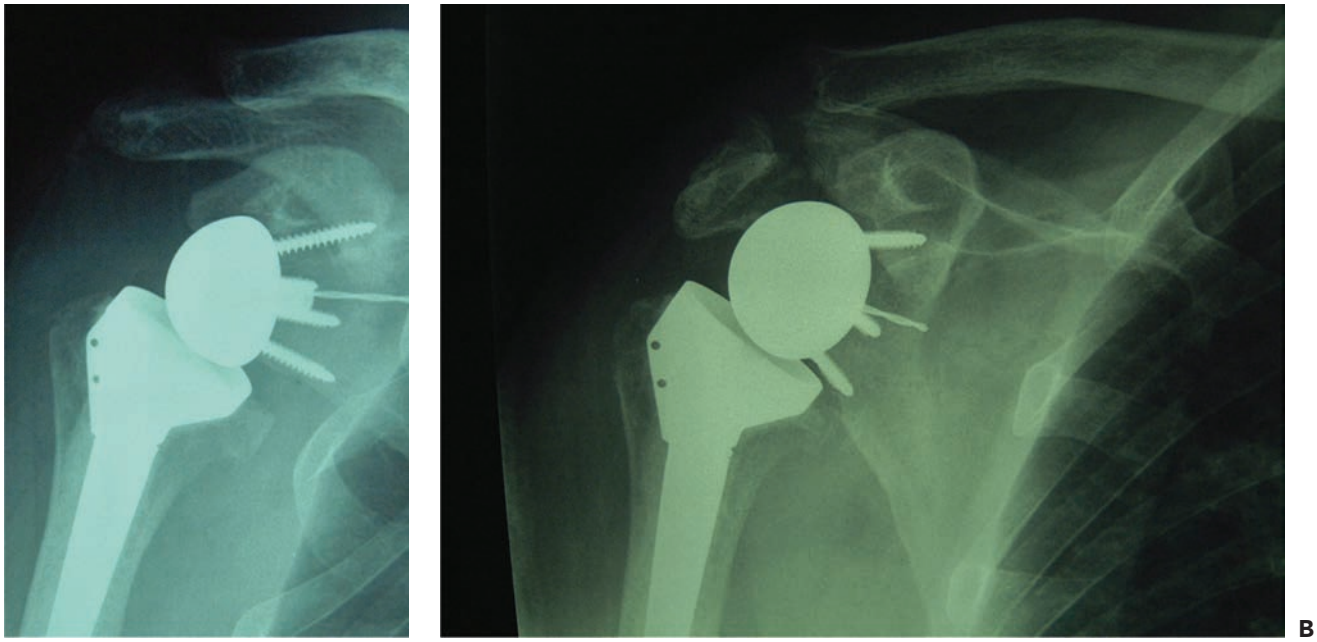


Figure 23-21 **(A,B)** Fatigue fracture of the acromion and the scapula body with a reverse total shoulder arthroplasty.

SURGICAL TECHNIQUE

This prosthesis can be placed using either the superior deltoid splitting approach, which is most popular in Europe and is recommended for the primary arthroplasty for its excellent exposure of the glenoid, or through a standard deltopectoral approach, which is preferred in revision surgery, particularly when removing as stemmed humeral implant. In these cases the deltopectoral approach is more extensile than the superior deltoid splitting approach. An extensile approach may be required to remove a secure humeral stem or other humeral hardware. A deltopectoral approach can be used in primary reverse shoulder replacement. Glenoid exposure is facilitated by absence of a superior and in many cases a posterior rotator cuff. When the subscapularis tendon is detached with a deltopectoral approach, the glenoid exposure is much better than with standard arthroplasty with an intact rotator cuff. In all cases, regardless of the approach used, there needs to be sufficient glenoid bone to allow for secure fixation of the glenoid component.

As the superior approach for the surgical technique using the superior approach for the reversed prosthesis, Delta3 has not been widely described in the English literature and will therefore be described in some detail in this chapter.

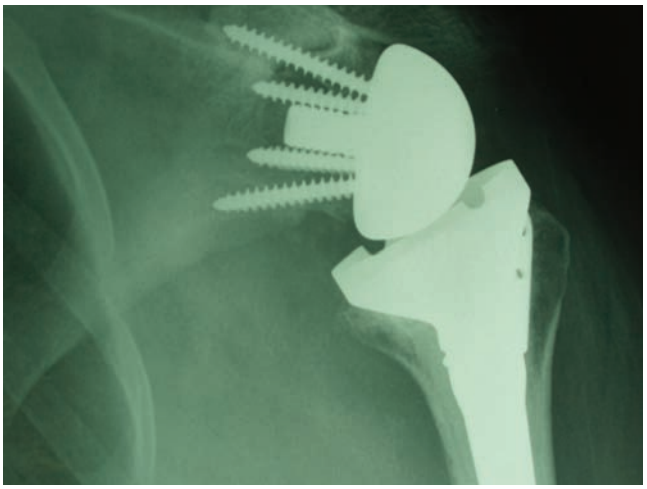


Figure 23-22 Mild inferior scapular notching.

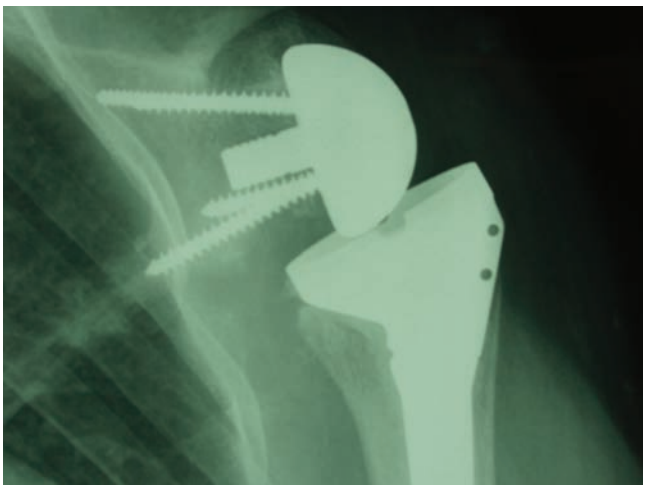


Figure 23-23 Moderate inferior glenoid notching.

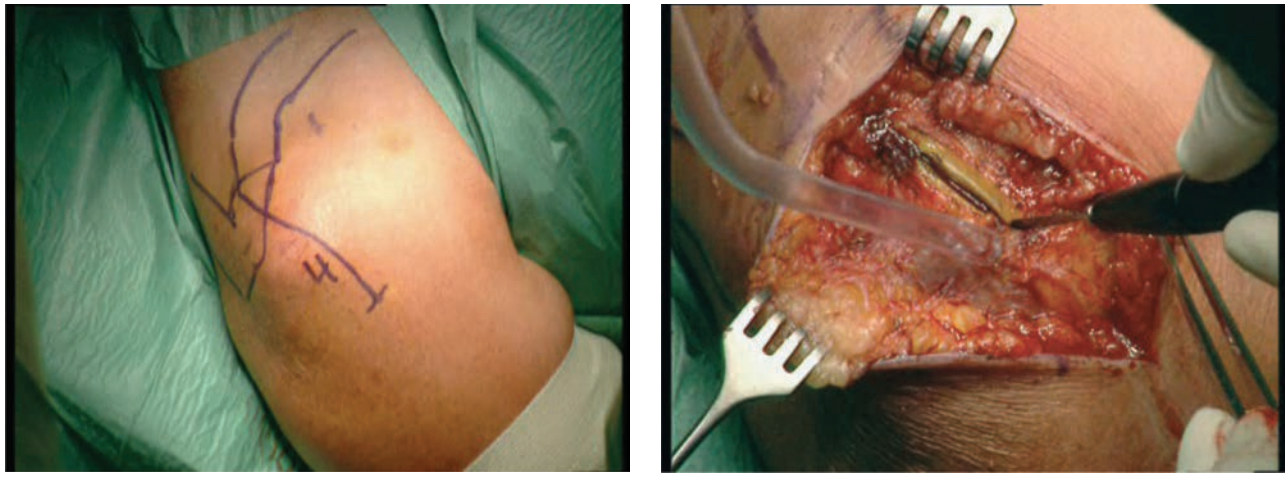


Figure 23-24 **(A,B)** A skin incision is made perpendicular to Langer's lines from the level of the acromioclavicular joint posterior to the joint over the midacromion and over the middle deltoid for a distance of 5 to 6 cm. The anterior deltoid is sharply dissected from the distal 2 cm of the clavicle and the acromion, leaving a tendon and fascia attached to the deltoid muscle for later secure attachment.

Positioning

The patient is positioned in a beach-chair position.

Surgical Exposure

An anterior–superior approach (McKenzie) is generally utilized unless a previous surgery dictates otherwise (deltopectoral, anterolateral).[41] A skin incision is made perpendicular to Langer's lines from the level of the acromioclavicular joint posterior to the joint over the midacromion and over the middle deltoid for a distance of 5 to 6 cm (Fig. 23-24). The anterior deltoid is sharply dissected from the distal 2 cm of the clavicle and the acromion, leaving a tendon and fascia attached to the deltoid muscle for later secure attachment (see Fig. 23-24). The coracoacromial ligament is cut sharply from the acromion and left attached to the undersurface of the deltoid. If there is a prominent acromion or spur, an osteotomy of the acromion is performed to the level of the anterior border of the clavicle. The humeral head is then dislocated superiorly into the wound by extension of the arm, slight external rotation, and axial load at the elbow (Fig. 23-25). The degenerative hypertrophied bursa is removed. Humeral osteophytes are removed with a rongeur.

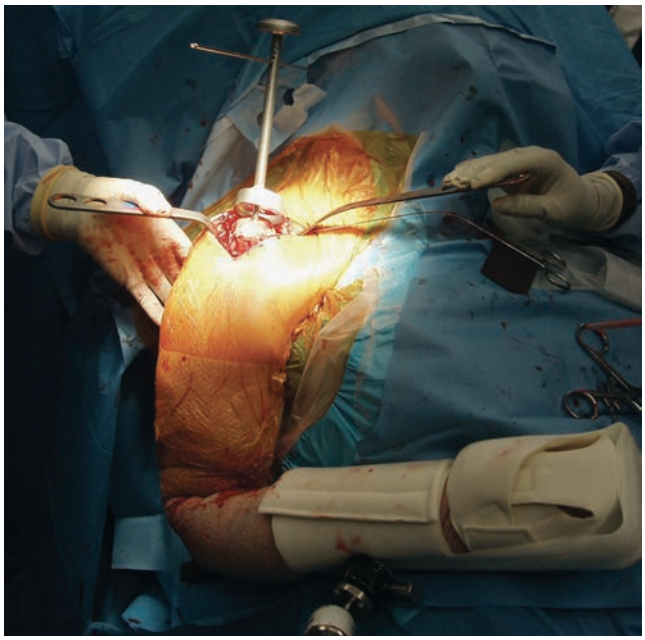


Figure 23-25 The humeral head is dislocated superiorly into the wound by extension of the arm, slight external rotation, and axial load at the elbow.

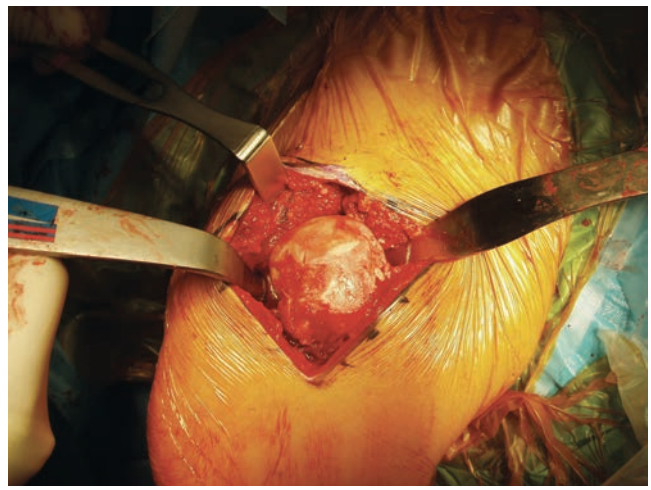


Figure 23-26 Small Homans retractors are placed beneath the intact cuff to expose the humeral head.

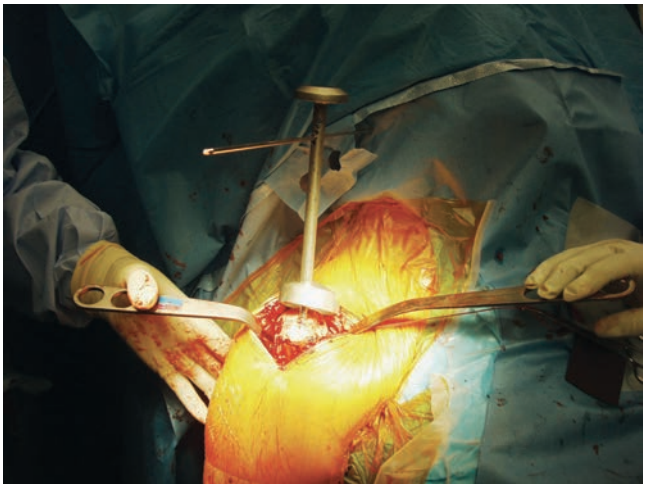


Figure 23-27 The humeral canal is broached with an intramedullary cutting guide and the head is cut in approximately 10 degrees of retroversion.

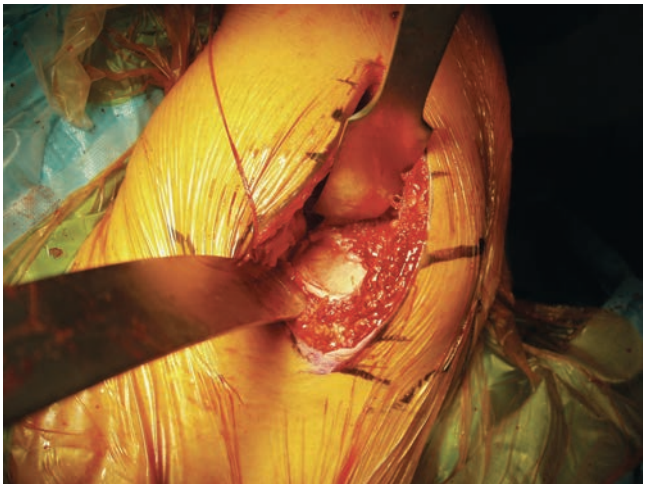


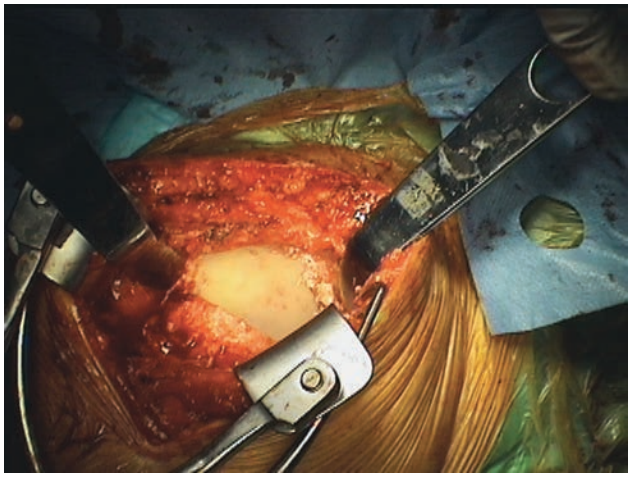
Figure 23-29 A humeral head retractor is placed to expose the glenoid and to be able to palpate the inferior scapula neck.

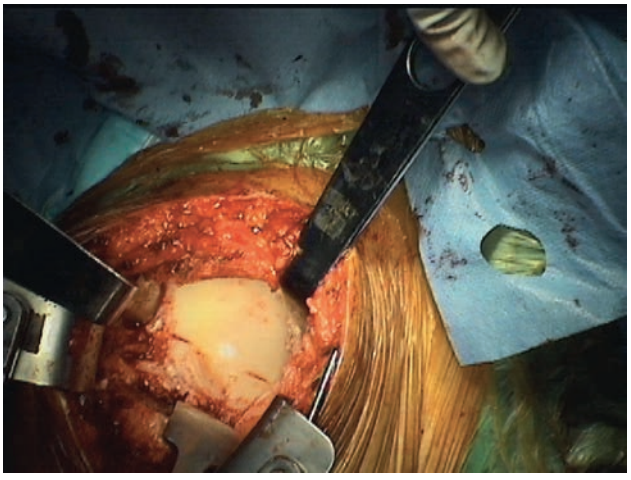
A large Darrach is placed through the cuff defect into the joint and the head is levered away from the glenoid. Any cuff tissues covering the top of the head are removed and care is taken to preserve anterior or posterior cuff tissue when it is present. Small Homans retractors are placed beneath the intact cuff to expose the humeral head (Fig. 23-26). The humeral canal is broached with an intramedullary cutting guide and the head is cut in approximately 10 degrees of retroversion (Fig. 23-27). The height of the head cut should be such that with axial distraction of the humerus, the cut surface of the humeral osteotomy is at the inferiormost portion of the glenoid (Fig. 23-28a). A humeral head retractor is placed to expose the glenoid (Fig. 23-28b). A complete capsulectomy is carried out anteriorly, posteriorly, and inferiorly. It is important to preserve the intact rotator cuff that remains anteriorly and posteriorly and expose the entire glenoid as well as be able to palpate the inferior scapula neck (Fig. 23-29).

Glenoid Reaming

Glenoid reaming is carried out in a slightly anterior and inferior orientation, if allowed by the available glenoid bone stock. This provides more articular contact and stability with the arm in internal rotation, and when the arm is in the adducted position it reduces polyethylene liner contact with the scapula neck, which minimizes inferior glenoid notching (Fig. 23-30). The reaming is preformed for the selected size glenosphere with a goal of inserting the largest component that will be accepted by the soft tissue and bone (glenoid) size. When reaming it is best, when possible, to preserve subchondral bone.

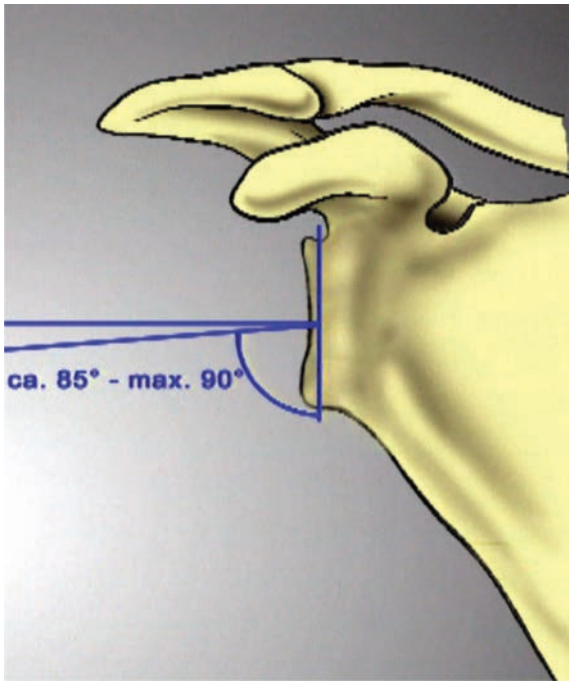
A





B

Figure 23-28 **(A,B)** The height of the head cut should be such that with axial distraction of the humerus, the cut surface of the humeral osteotomy is at the inferiormost portion of the glenoid.



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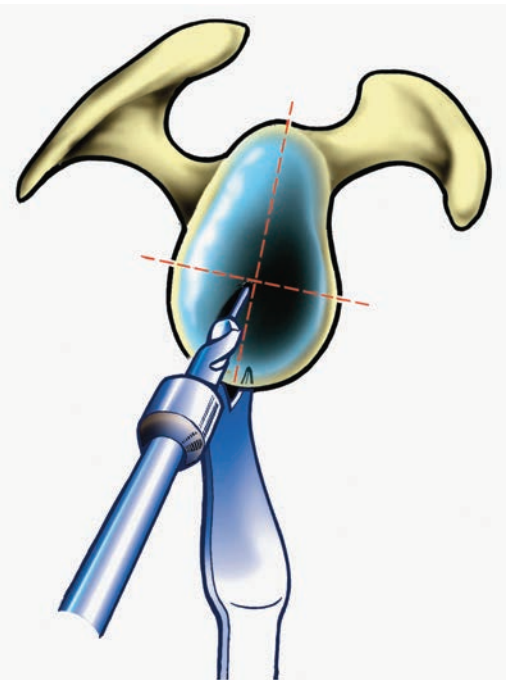

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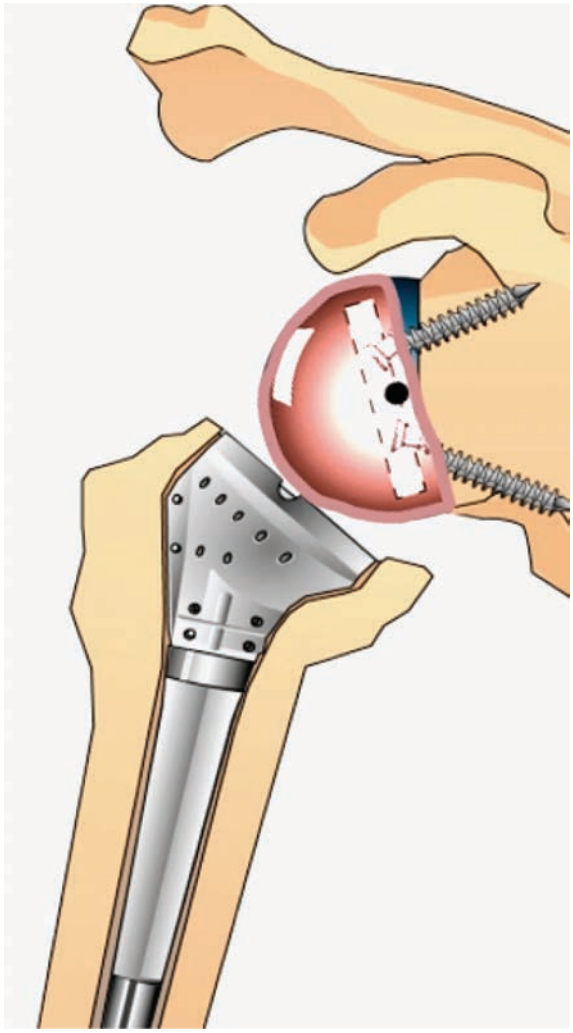


C

Figure 23-30 The glenoid reamer is placed so that the glenosphere is inferior to the glenoid rim and is tilted interiorly.

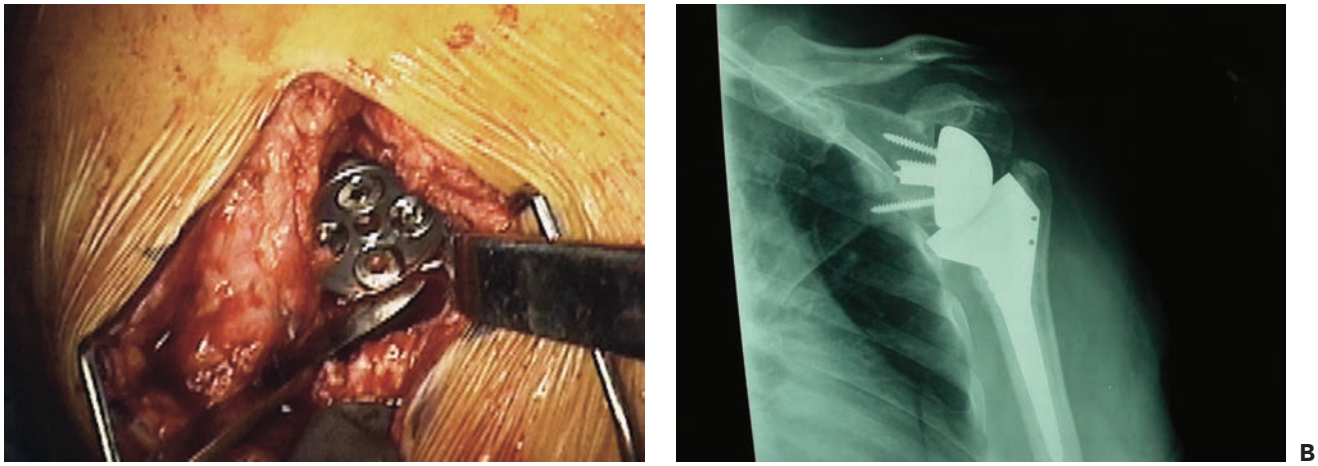


Figure 23-31 **(A,B)** It is important to have the inferior screw in the thick bone of the axillary boarder of the scapula.

Glenoid Assembly

The hydroxy-appitite–coated glenoid base plate ("metaglené") is then connected and secured to the glenoid bone using two divergent locking screws (superior and inferior) and two convergent compression nonlocking screws. It is important to have the inferior screw in the thick bone of the axillary boarder of the scapula (Fig. 23-31). The metaglené is rotated so that the inferior hole in the base plate is in line with the inferior scapula neck and scapula body. This is the first screw to be placed. Orientation of this hole is facilitated by palpation of these bony landmarks, which in turn is facilitated by inferior capsular excision. Currently, two sizes of glenospheres (both fitting onto the same size metaglené) are available (36 and 42 mm in diameter). The size to be used is defined by the diameter of the proximal humerus. When possible, it is preferable to use the larger-diameter glenosphere, which improves stability and range of motion. After secure placement of the definitive metaglené, a trial glenosphere is placed and the humeral shaft is again exposed into the wound as described above.

Humeral Side

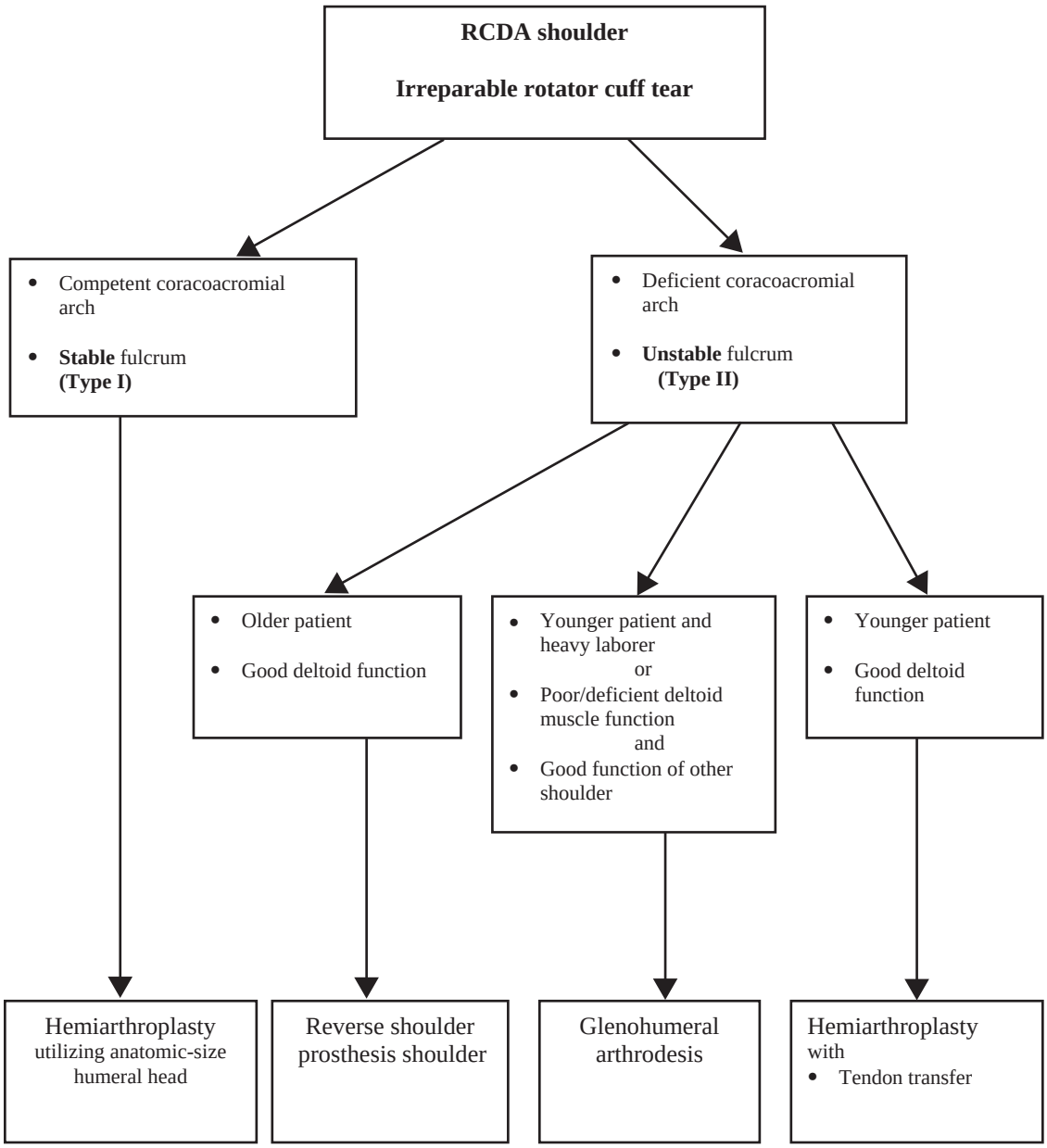
The humeral shaft and the metaphysis are reamed to cortical bone using hand reaming. Using trial components, the stability and range of motion are tested and final prosthetic height and version noted for placement of the final components. The humeral component is cemented. Three thicknesses of polyethylene inserts are available in the standard design. These thicknesses can help adjust for tissue tension at final prosthetic selection. A 9-mm metal extension component that screws into the final (and provisional) implant can also allow for increasing tissue tension after placement of the final component. Ideally, cementing the stem in the proper height is determined by tissue tension of the trial components, which makes routine use of the metal extension piece infrequently necessary. When using the trial component, the tissue tension should be judged with removal of self-retaining retractions with the arm in different positions of flexion and extension and rotation. There should not be any toggle between the components, and in most cases reduction will require firm axial distraction of the humerus to engage the components. Lift-off of the humeral component with the arm in adduction with the arm by the side of the body is not acceptable and requires correction before placement of the final components. When this occurs it is most often due to impingement of the humeral liner on the scapula neck or glenoid rim. If the glenosphere is of the proper size and placement (at or slightly below the inferior glenoid rim with a 10-degree inferior tilt), then this is usually secondary to prominent bony ridge at the glenoid rim or near the glenoid neck, which should be removed with a rongeur or burr. Similarly, if there is component lift-off with internal or external rotation, then removal of any overhanging bone on the glenoid or humeral side is often necessary. Use of the retentive polyethylene component to improve component stability should be avoided, if possible, as it has a greater tendency for notching.

Wound Closure

After thorough irrigation, the deltoid muscle is repaired side to side and with transosseous sutures to the acromion using #2 nonabsorbable braided suture.

Postoperative

Postoperatively, the shoulder is supported with a small abduction pillow for 3 to 4 weeks. Full active assisted



ALGORITHM 23-I
Algorithm for surgical management of rotator cuff–deficient arthritic (RCDA) shoulder.

range-of-motion exercises are immediately started, excluding extension or internal rotation behind the body.

TREATMENT ALGORITHM

Patients with mild symptoms and mild limitation in functional range of motion and activities of daily living should be treated nonsurgically. Painful shoulders with a stable fulcrum should be considered for a hemiarthroplasty. A competent coracoacromial arch is a keystone for successful results, and therefore, should not be damaged during the surgical procedure. Due to the high rates of glenoid component loosening, anatomic (as opposed to reverse shoulder prostheses) total shoulder replacement should not be performed for rotator cuff–deficient arthritic joints. For older patients with a painful, anterosuperiorly unstable shoulder, a reverse shoulder arthroplasty should be considered. For younger patients with an incompetent coracoacromial arch but a good functional deltoid, a combination of hemiarthroplasty and tendon transfer can be considered. For younger heavy laborers and those with a

nonfunctional deltoid muscle, an arthrodesis is a better option than prostheses.

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24 Complications of Shoulder Arthroplasty

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While there has been discussion in the literature concerning complications of shoulder arthroplasty, they are often addressed obliquely in the materials presented with only a few articles concentrating on a specific complication. By collating this literature, one can evaluate the types and frequency of complications but is less informed about the nuances of each type of complication and treatment recommendations. Therefore, we have felt it important to not only detail the information in various patient series and display the lesser number of articles addressing a specific complication, but also to present the materials developed over time at our institution that can be analyzed in some depth.

TYPES AND FREQUENCIES OF COMPLICATIONS

Literature

Previously, we identified 22 patient series published since 1980 on unconstrained total shoulder arthroplasty (TSA).[1–22] Combined, these series have reported on 1,183 total shoulder arthroplasties. The authors identified 23 different complications. The total number of complications was 123 for a relative percentage of complications of 10.4%, assuming one complication occurred in each of the shoulders having a complication. Certainly some shoulders had two or more complications, so the number of shoulders having a complication would be somewhat less than 10%. Only rotator cuff tearing, instability, and glenoid loosening occurred in more than 1% of shoulders reported (Table 24-1).

We identified 20 patient series reporting on hemiarthroplasty of the shoulder that have been published since 1980.[5,6,19,21,23–38] These included reports on 498 shoulders. Nineteen different complications were identified and 78 total complications were reported for a relative frequency of complications of 15.7%, assuming only one complication occurred in each shoulder (Table 24-2). Six complications occurred at a frequency of 1% or greater. These included glenohumeral instability, painful glenoid arthritis, humeral tuberosity nonunion, rotator cuff tearing, nerve injury, and infection.

The Mayo Clinic Experience

Total Shoulder Arthroplasty

The relative incidence and type of complications at our institution have changed over time. In 1999, we reported on 419 unconstrained total shoulder arthroplasties performed by the senior author (RHC) between December 1975 and December 1989.[39] The complications were defined according to time of occurrence, with early complications occurring within 90 days of surgery and late

TABLE 24-1
COMPLICATIONS OF TOTAL SHOULDER ARTHROPLASTY[a]

Complication	No. (%)[b]
Rotator cuff tear	23 (1.9)
Instability	18 (1.5)
Glenoid loosening	15 (1.3)
Intraoperative fracture	15 (0.9)
Malposition	7 (0.6)
Nerve injury	7 (0.6)
Infection	5 (0.4)
Humeral loosening	4 (0.3)
Postoperative fracture	4 (0.3)
Wound problem	4 (0.3)
Wire breakage	3 (0.3)
Impingement	3 (0.3)
Tuberosity nonunion	3 (0.3)
Pain, unexplained	3 (0.3)
Reflex dystrophy	2 (0.2)
Hematoma	2 (0.2)
Component dissociation	2 (0.2)
Extruded cement	2 (0.2)
Heterotopic ossification	1 (0.1)
Stiffness	1 (0.1)
Spacer dislocation	1 (0.1)
Intraoperative death	1 (0.1)
Pulmonary embolism	1 (0.1)
Totals = 23	**123 (10.4)**

[a] Twenty-two series since 1980; 1,183 shoulders studied.
[b] Relative percentages of each complication, assuming one complication occurred in each of the shoulders having a complication.

TABLE 24-2
COMPLICATIONS OF HUMERAL HEAD REPLACEMENT[a]

Complication	No. (%)[b]
Instability	14 (2.8)
Glenoid arthritis	12 (2.4)
Tuberosity nonunion	9 (1.8)
Rotator cuff tear	9 (1.8)
Nerve injury	8 (1.6)
Infection	5 (1.0)
Intraoperative fracture	3 (0.6)
Humeral loosening	3 (0.6)
Wound problems	2 (0.4)
Tuberosity malposition	2 (0.4)
Hematoma	2 (0.4)
Perioperative death	2 (0.4)
Postoperative fracture	1 (0.2)
Heterotopic ossification	1 (0.2)
Impingement	1 (0.2)
Reflex dystrophy	1 (0.2)
Acromioclavicular pain	1 (0.2)
Pain, unexplained	1 (0.2)
Stiffness	1 (0.2)
Totals = 19	**78 (15.7)**

[a] Twenty series since 1980; 498 shoulders studied.
[b] Relative percentages of each complication, assuming one complication occurred in each of the shoulders having a complication.

complications occurring after this period of time. Surgical complications were also classified as minor when there was no compromise of outcome and little or no treatment was required. A major complication occurred when the final result was compromised or reoperation was required.

Among these shoulders, 130 (31%) had a major surgical complication, with 95 of these shoulders requiring reoperation (Table 24-3). The most frequent complication requiring reoperation was joint subluxation followed in order of frequency by rotator cuff tearing, glenoid loosening, dislocation, humeral loosening, and infection. One can see that the variety and aggregate number of complications in the early experience with the shoulder arthroplasty was quite high. Therefore, we reviewed the outcome of a more contemporary group of patients to determine whether the complications have changed or the complication rates have lessened over time.

The complications of 431 total shoulder arthroplasties performed in patients by the senior author (RHC) between December 1990 and December 2000 were reviewed.[40] Fifty-three surgical complications occurred in 53 patients. Thirty-two were considered to be major (7.4%), with 17

TABLE 24-3
SEVERITY OF SURGICAL COMPLICATIONS

Complication	Minor[a]	Major	Reoperation
Subluxation	1	11	32
Rotator cuff tear	5	11	17
Glenoid loosening		2	17
Brachial plexopathy	3	8	
Dislocation			10
Humeral loosening			9
Infection (deep)			6
Impingement	5		
Dysesthesias	5		
Infection (superficial)	2		2
Fracture	3	1	
Hematoma	1		1
Reflex dystrophy		2	
Nerve laceration			1
Tuberosity nonunion	1		
Long head of biceps rupture	1		
Totals	**27**	**35**	**95**

[a] See text for definitions.

TABLE 24-4
SEVERITY OF COMPLICATIONS

Complication	Minor	Major	Major with Reoperation
Rotator cuff tear	3	8	6
Fracture	7	5	1
Brachial plexopathy	8		
Subluxation		1	4
Dislocation		1	3
Humeral loosening	1		
Humeral and glenoid loosening			1
Infection (deep)			1
Hematoma	1		1
Long head of biceps rupture	1		
Totals	**21**	**15**	**17**

(3.9%) of these requiring reoperation (Table 24-4). Thirty-two occurred early and 21 occurred late. Rotator cuff tearing was the most common complication encountered. Of the 17 symptomatic cases of rotator cuff tearing, eight patients had a preoperative rotator cuff tear. There were four postoperative subscapularis tears and all four patients experienced anterior instability. When comparing the five main categories of diagnosis (osteoarthritis, rheumatoid arthritis [RA], posttraumatic arthritis, cuff tear arthritis, and osteonecrosis) there was no statistically significant difference ($P = 0.088$) in the complication rates observed in each of these groups—when comparing each of the last four diagnostic categories relative to the primary diagnosis of osteoarthritis. Risk factors for any complication and for specific complications were carefully assessed. The frequency of complications was not affected by age, gender, previous surgery, humeral head size, or whether the humeral component was cemented or uncemented.

When comparing this with the prior study, one can clearly see that the frequency of complications has dramatically decreased as has specifically the number of major complications and the need for reoperation. In addition to the general lessening of complications, there is the noticeable striking diminution of component loosening.

Hemiarthroplasty

We have analyzed our hemiarthroplasties in two groups. The first group includes those with glenohumeral arthritis—either osteoarthritis or RA. Between July 1977 and March 1983, 77 shoulders were so treated in 74 patients.[41] Six patients with six operated shoulders were lost to follow-up and four others died before evaluations were complete. This resulted in 67 shoulders in 64 patients forming the basis of the review. There were 35 shoulders in 35 patients with osteoarthritis and 32 shoulders in 29 patients with RA. Follow-up evaluation averaged 9.3 years and ranged from 2 to 14.1 years. There were three complications: One patient developed a hematoma requiring surgical evacuation; the second had a humeral shaft fracture at the time of surgery, which was treated with internal fixation using a long-stemmed component and cerclage (the fracture healed); and the third complication was a brachial plexus traction injury that recovered without residual symptomatology.

The outcome of humeral head replacements performed for acute proximal humeral head fractures and chronic proximal humeral head fractures between 1979 and 1995 was also reviewed, focusing on complications.[42] Twenty-eight replacements were performed in 26 patients with acute fractures and 55 hemiarthroplasties were performed in 55 patients for problems that arose following initial treatment for a proximal humeral fracture or fracture-dislocation. Again, all patients were included to be sure that every complication was recognized during the follow-up period. In the acute group follow-up averaged 56 months, ranging from 2 to 184 months, while in the chronic group follow-up averaged 57 months, ranging from 5 to 156 months. The complications identified for these acute and chronic fracture groups are displayed in Table 24-5. One is immediately impressed that these are much greater in number and many more types of complications arose when compared with hemiarthroplasty done for the elective treatment for shoulder arthritis.

SPECIFIC TYPES OF COMPLICATIONS

The six significant and most common complications will be individually presented. Material will generally be arranged in the following sequence: the recognition and evaluation of the complication with a classification system if one is applicable, reference to the frequency of the complication as presented in the literature, identification of specific literature for the complication, notation of materials included in pertinent review articles, the experience with this complication at the Mayo Clinic, methods to prevent the complication, and treatment for the complication.

Nerve Injury

Physical examination of the operated upper extremity is the key to diagnosis following surgery. It is quite practical to ask the patient to perform active movement of the hand and wrist and to test for isometric contractions of the elbow flexors and the posterior portion of the deltoid muscle. This usually can be accomplished on the day of surgery; however, interscalene block is now commonly used, making it sometimes necessary to perform the neurologic examination the

TABLE 24-5
COMPLICATIONS OF HEMIARTHROPLASTY FOR FRACTURES[a]

Complication	Acute (No. = 28)	Chronic (No. = 55)	Totals (No. = 83)
Instability	2	10	12
Glenoid arthritis	2	7	9
Rotator cuff tear	2	4	6
Infection	3	3	6
Tuberosity nonunion	2	2	4
Fracture		3	3
Implant malposition		3	3
Implant loosening	1	2	3
Tuberosity malunion		2	2
Reflex dystrophy	1		1
Totals	**13**	**36**	**49**

[a]Adapted from Muldoon MP, Cofield RH. Complications of humeral head replacements for proximal humeral fractures. *Instr Course Lect* 1997;46:15–24.

day following surgery. Should a nerve injury be identified, the common peripheral nerve injury classification that is defined by Seddon or Sunderland is probably useful in retrospect but has less value in the acute setting.[43,44] Weber et al. developed a post-hip arthroplasty nerve palsy severity scale based on symptoms, physical examination, electromyographic findings, and the compromise of postsurgical rehabilitation,[45] and it is useful as a grading system. This scale, though, as with the more standard classification scheme, is only fully applicable over time, as the nerve injury evolves, and less helpful in the acute setting. Electromyographic testing may be useful after the initial 3 weeks, but as will be explained below, is probably more practical at 4 to 6 weeks, should neurologic recovery not occur in the interim.

In the 22 patient series encompassing 1,183 operated shoulders that were reported since 1980 and defined in Table 24-1, seven nerve injuries (0.6%) were identified following TSA. In the 20 patient series involving 498 shoulders reported since 1980, eight nerve injuries (1.6%) were identified following hemiarthroplasty. The slightly higher frequency following hemiarthroplasty may be attributed to this surgery often occurring in the acute setting where some degree of nerve injury might complicate the initial fracture and yet not be fully defined because of the inability to perform a complete examination before surgery. Only one article has been published on neurologic complications after TSA.[46] These authors identified eight neurologic deficits reported in the literature. Of these five were axillary nerve palsies, only one of which completely resolved and two partially recovered. One musculocutaneous nerve palsy did not resolve. One radial nerve palsy responded completely to removal of cement that had extruded through a humeral defect during revision surgery. The final injury produced ulnar nerve dysesthesias, which resolved.

Nerve injuries have received comment in review articles of shoulder arthroplasty. Miller and Bigliani mentioned that nerve injuries are uncommon, that they most often represent a neurapraxia, and that the axillary nerve is the most likely to be injured.[47] They are of the opinion that if the initial lesion is partial and improving, observation is indicated. If there is a suspicion that the nerve was lacerated at surgery and electromyography at 6 weeks reveals a complete lesion with no improvement at 12 weeks, exploration and surgical repair are suggested. Wirth and Rockwood were able to identify 14 reported nerve injuries following total shoulder replacement.[48] Again, they felt most of the injuries represented a neurapraxia with nonoperative treatment being appropriate. Six lesions involved the axillary nerve, three the ulnar nerve, two the musculocutaneous nerve, and one the median nerve, and two were a more general brachial plexus injury. Resolution was complete in seven, was incomplete in two, and did not occur in one, and in four the extent of recovery was not defined. Importantly, in two shoulders there was a laceration of the axillary nerve occurring in a heavily scarred operative field. Soghikian and Neviaser[49] presented a thorough discussion of complications of hemiarthroplasty, and similarly Muldoon and Cofield[42] presented material on complications of hemiarthroplasty for proximal humeral fractures; in neither of these reviews was nerve injury identified as a complication.

Lynch et al. reported the Mayo Clinic experience with neurologic complications after TSA.[46] Four hundred seventeen arthroplasties were studied. Seventeen patients with 18 operated shoulders had a neurologic deficit after surgery (4.3%). All appeared to be traction injuries; 13 involved the brachial plexus. The upper and middle trunk were involved in six, the upper trunk in three, the lateral

cord in two, the lower trunk in one, and all trunks in one. Interestingly, three were thought to represent the initiation of an idiopathic brachial neuritis. One patient with dysesthesias after earlier radiation therapy had an increase in the level of dysesthesias in the lower trunk and one patient developed median neuropathy at the wrist. The quality of recovery in the first 16 patients was graded as good in 11 and fair in five. Time to recovery was less than 3 months in eight, 3 to 6 months in four, 6 to 12 months in one, and greater than 12 months in three. Numerous patient factors were studied including diagnosis, age, sex, height, weight, use of corticosteroids, the presence of diabetes mellitus, preoperative range of motion, the presence or absence of rotator cuff disease, previous surgery, and the use of interscalene block. None of these was found to be related to a nerve injury. However, exposure through the slightly more demanding deltopectoral approach ($P = 0.003$) and the use of methotrexate in patients with RA ($P < 0.0001$) were statistically associated with the development of a postoperative nerve palsy. Thus, this series of a large number of shoulder arthroplasties defines that brachial plexus stretch injuries are by far the most frequently recognized neurologic deficits following prosthetic shoulder arthroplasty, that when searched for nerve injuries are more common than has been recognized, and thankfully that recovery is the rule, without significant compromise to the arthroplasty per se.

As identified by the above information, prevention of nerve injuries at surgery is usually, but not always, possible. Certainly, locating the axillary nerve at the inferior aspect of the subscapularis and near the posterolateral aspect of the humerus is useful, and careful retraction of the conjoined group is important. Dissection on the undersurface of the superior and posterior aspects of the rotator cuff should not extend more than 1 cm medial to the glenoid rim, to avoid injury to the suprascapular nerve. Positioning of the arm in extension and abduction with external rotation should be limited in extent and time as much as possible. To facilitate all of these protective measures, it is important to have a dry operative field and to be especially cautious when there is distortion of anatomy such as following an old fracture or fracture-dislocation.

Concerning treatment, definition of the injury by careful physical examination is important. If there is weakness of hand, wrist, or elbow function, splinting may be necessary. Swelling should be minimized by elevation and use of compressive dressings. If active motion is not possible for the hand, wrist, or elbow, passive motion should be used. Passive motion of the shoulder should be commenced during the early postoperative period (within the limits determined at surgery) with active assisted motion initiated as the return of strength will allow.

If there is no improvement in neurologic function by 4 to 6 weeks, electromyography with nerve conduction should be performed to determine more precisely the localization and extent of the nerve injury. If the lesions are diffuse and incomplete, one would suspect a brachial plexus stretch–type lesion and conservative measures would continue. If a focal complete nerve injury is identified such as to the axillary nerve, one would be more concerned about a significant adverse intraoperative event. Quite likely in this setting, continued observation would occur. Further examination would be performed at 3 months. If there was no apparent recovery, electromyography would be repeated at that time and more serious consideration would be given to operative intervention to address the isolated nerve lesion. There is an important caveat, however: So few nerve lesions have occurred due to trauma to a specific nerve that it is hard to be concrete about the recommendations for surgical exploration, other than those indications that apply in general to focal peripheral nerve injuries associated with surgical intervention.

Periprosthetic Fractures

Fractures can occur both intraoperatively and postoperatively. Intraoperatively, humeral shaft fractures are most common. Fractures can also involve the proximal humeral metaphysis, the humeral tuberosities, the glenoid, and the coracoid process. Postoperatively, humeral shaft fractures are also most common. Fractures can involve other areas including the acromion process and the coracoid process. The recognition of a fracture intraoperatively may be obvious, but sometimes undisplaced cracks develop in the bone that are only detected on subsequent radiographs. Postoperative humeral shaft fractures have been classified by Wright and Cofield.[50] Type A extends proximally from the tip of the prosthesis and may create stem loosening (Fig. 24-1). A type B fracture is centered at the tip of the stem with minimal to no proximal extension (Fig. 24-2), and the type C fracture involves the humeral shaft distal to the tip of the prosthesis and usually includes fracturing into the distal humeral metaphysis (Fig. 24-3).

All of these fractures following TSA are relatively uncommon, as can be seen from Table 24-1. Intraoperative fractures occur in less than 1% of cases and postoperative fractures occur in less than one-half of 1% of cases. Following humeral head replacement (Table 24-2), intraoperative fractures and postoperative fractures are even less common. However, it is not unusual for a number of arthroplasty series to report one or two humeral shaft fractures, and such is the case with reports by Kelly et al.,[16] Boyd et al.,[51] Barrett et al.,[3] Hawkins et al.,[15] and Faludi and Weiland.[52] The fractures have occurred during varying maneuvers during surgery including exposure, humeral preparation/reaming, and humeral implant insertion. Additionally, three glenoid fractures were described, one by Kelly et al. and two by Hawkins et al. Similarly, a number of these series have described the occasional postoperative fracture including the report of one humeral shaft

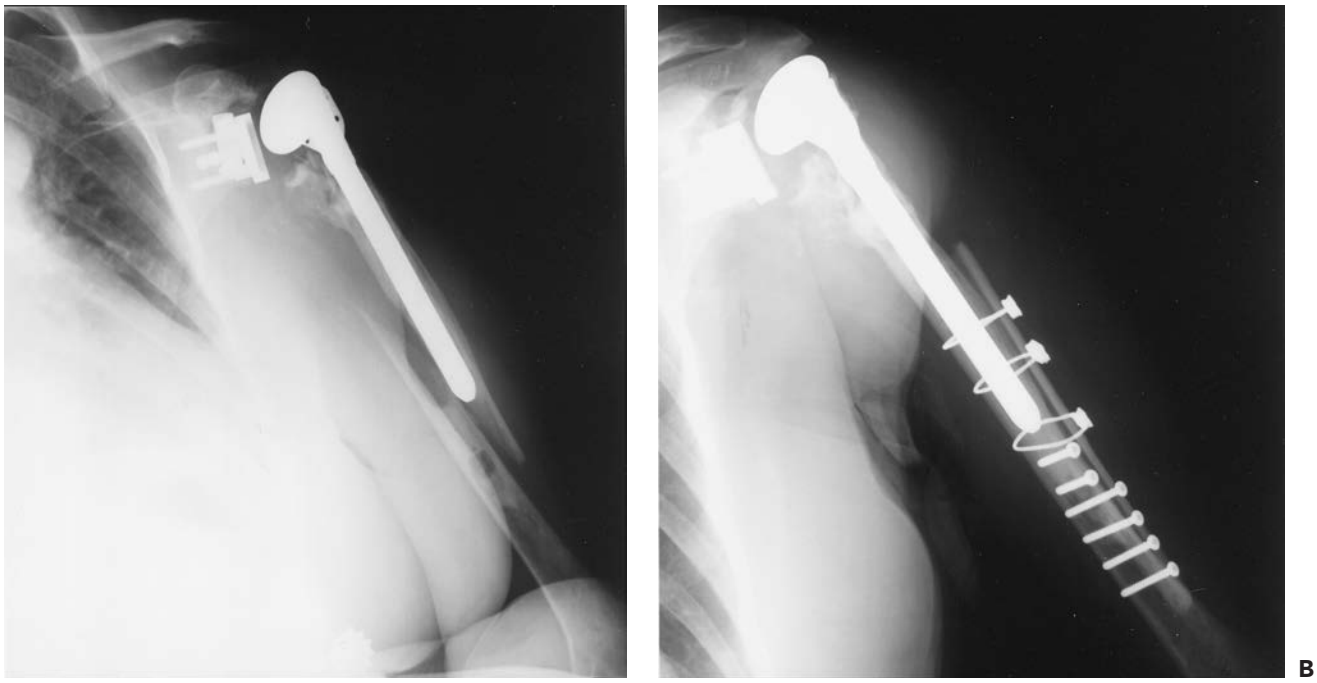


Figure 24-1 **(A)** Example of a type A fracture through the bone and cement mantle. The humeral component was well fixed. The periprosthetic fracture was treated with a cortical strut, cables, and screws. **(B)** Radiograph taken at 4 months demonstrates a healed fracture; however, the strut graft did not appear to be incorporated. (Reproduced with permission from Kumar S, Sperling JW, Haidukewych GH, Cofield RH. Periprosthetic humeral fractures after shoulder arthroplasty. *J Bone Joint Surg* 2004;86A:680–689.)

fracture by Barrett et al.,[3] one humeral shaft fracture by Neer et al.,[18] one humeral shaft fracture by Brenner et al.,[7] three humeral shaft fractures by Post et al.,[53] one humeral shaft fracture by McElwain and English,[54] and one acromial fracture by Arnst et al.[23] and another by Thomas et al.[55] Thus, although fracturing is indeed uncommon, it in fact does occur, and many of the authors reporting on shoulder arthroplasty have experienced this complication at some point in time.

Only a few reports have specifically addressed this problem. Boyd et al. identified seven humeral shaft fractures after shoulder arthroplasty.[56] Treatment was complicated, and five fractures did not unite until surgery was performed. Fractures that were treated operatively healed. In five of the six patients who had union of their fracture, shoulder motion was lost compared to prefracture levels. The authors of this study identified many associated factors including injury to the radial nerve, the high frequency of delayed union or nonunion, the question of mechanical interference with fracture healing by the prosthesis or cement, the advanced age of many patients, the presence of osteopenia or systemic disease, the questionable effectiveness of immobilization, the extensive time to union, and the compromise of function of the shoulder arthroplasty. Because of the small size of the patient group, the many diverse factors that need to be considered, and the variation in outcome, it was difficult for the authors to reach clear recommendations relative to fracture treatment.

Bonutti and Hawkins identified four patients with fracture of the shaft of the humerus.[57] They recognized the historical success with treatment of humeral shaft fractures by conservative means but felt that aggressive treatment was needed for fracture of the shaft of the humerus associated with shoulder arthroplasty. Groh et al. identified 11 fractures adjacent to humeral prostheses.[58] Four fractures extended from the proximal portion of the humeral shaft to beyond the distal tip of the prosthesis, and two occurred immediately proximal to the tip of the prosthesis. Three proximal fractures were managed with an orthosis. The remainder were treated with the combination of a long-stem prosthesis and cerclage wires. All fractures healed and function of the shoulder arthroplasty was maintained.

Campbell et al. identified 16 intraoperative and five postoperative periprosthetic humeral fractures.[59] Fracture outcome was analyzed according to three groups: cast or brace immobilization, intramedullary fixation with a humeral stem plus cerclage wiring, and plate or screw fixation. Stable intramedullary fixation provided superior results in time to union, had less effect on rehabilitation of the shoulder, and demonstrated a trend toward fewer complications. They felt that cast or brace immobilization

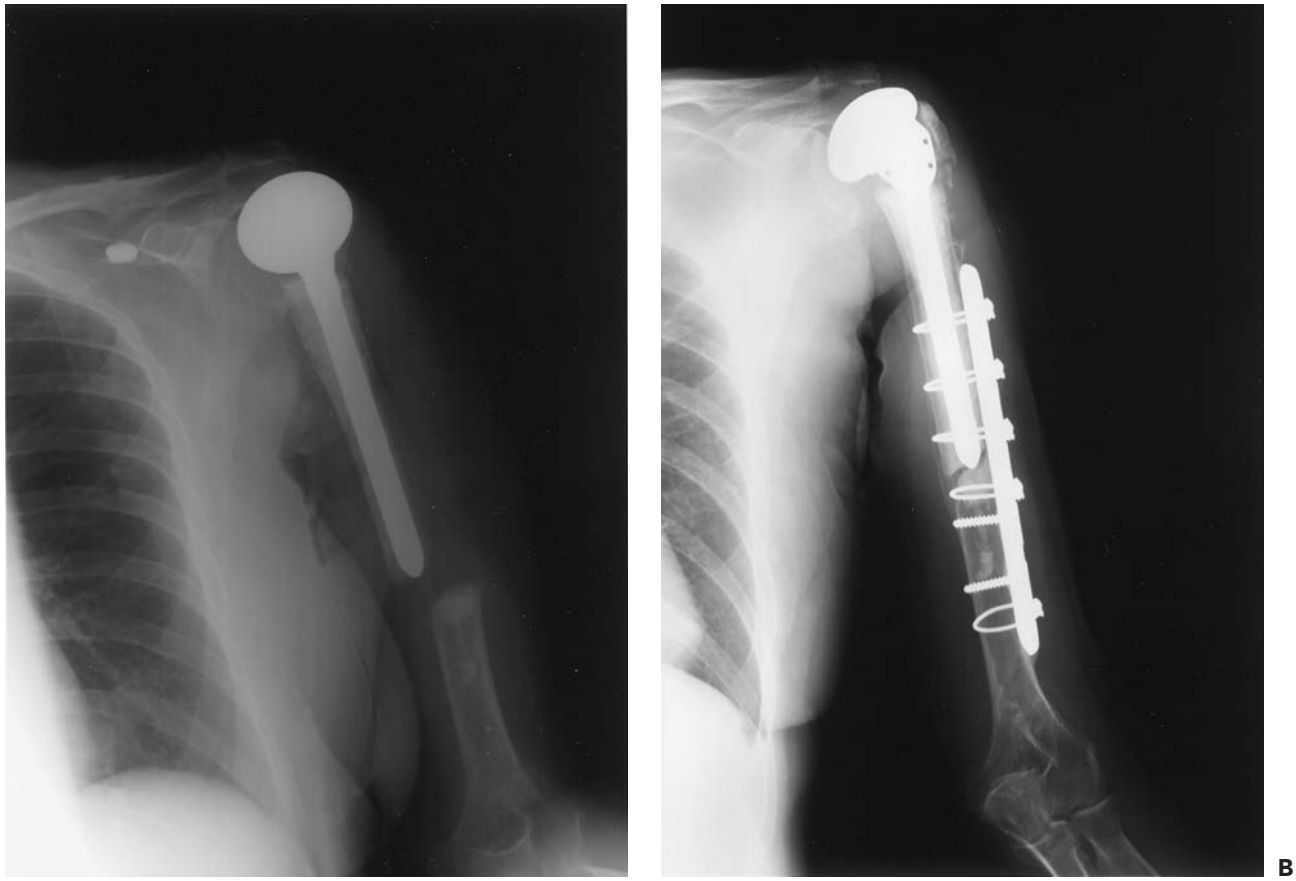


Figure 24-2 **(A)** This patient sustained a type B fracture that subsequently went on to a nonunion with nonoperative treatment. The humeral component was well fixed. Therefore, the patient was treated with open reduction and internal fixation with iliac crest bone graft. **(B)** Radiographs taken 27 months later demonstrate that the fracture healed. (Reproduced with permission from Kumar S, Sperling JW, Haidukewych GH, Cofield RH. Periprosthetic humeral fractures after shoulder arthroplasty. *J Bone Joint Surg* 2004;86A:680–689.)

could be acceptable for postoperative fractures distal to a well-fixed prosthetic stem. The authors noted that more proximal fractures should be fixed with a stem of appropriate length and cerclage wiring.

Three reviews on periprosthetic fractures have been performed at the Mayo Clinic.[50,60,61] Krakauer and Cofield's report included the outcome of intraoperative periprosthetic fractures in total shoulder replacement.[60] Intraoperatively, over an 18-year period, there were nine humeral shaft fractures, five greater tuberosity fractures, two proximal humeral metaphyseal fractures, four glenoid fractures, and two coracoid fractures. Of the intraoperative humeral shaft fractures, stability was achieved with a long-stem implant in six and with cerclage fixation in one. Two fractures were undisplaced and no fixation was performed; healing occurred in one but nonunion developed in the other. Three of the greater tuberosity fractures were sutured. The other two were undisplaced fractures and treated without fixation. One of the sutured greater tuberosities went on to nonunion and compromised active shoulder function. The proximal humeral metaphyseal and coracoid fractures created no adverse effect on arthroplasty outcome. The four glenoid fractures precluded placing an implant, and two of these patients continued to have moderate pain with their hemiarthroplasty.

Between 1976 and 2001, 19 postoperative periprosthetic humerus fractures occurred among 3,091 patients who underwent shoulder arthroplasty at the Mayo Clinic.[61] The outcome of 16 patients who had a complete series of radiographs were included in the study. The mean time from the arthroplasty to the fracture was 49 months (range 1 to 146 months). The indication for the arthroplasty was osteoarthritis in five patients, RA in five patients, failed arthroplasty in two, avascular necrosis in one, posttraumatic arthritis in one, and proximal humeral nonunion in two patients. There were seven patients with severe osteopenia. Twelve fractures occurred at the prosthesis tip, of which six extended proximally (type A) and six did not (type B). There were three fractures distal to the implant and extended into distal humeral metaphysis (type C).

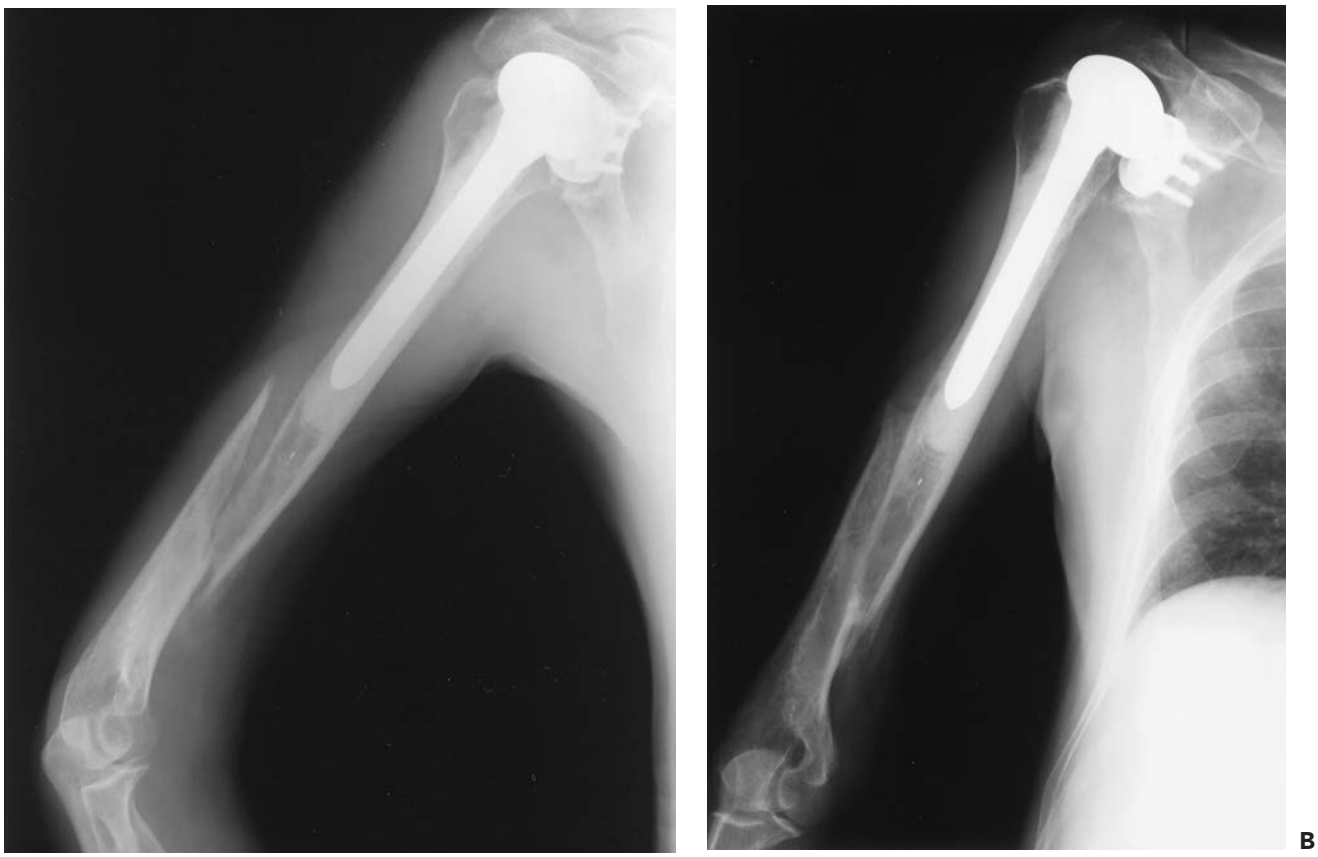


Figure 24-3 Example of a type C periprosthetic fracture **(A)** that was treated nonoperatively and went on to heal in 239 days **(B).** (Reproduced with permission from Kumar S, Sperling JW, Haidukewych GH, Cofield RH. Periprosthetic humeral fractures after shoulder arthroplasty. *J Bone Joint Surg* 2004;86A:680–689.)

One fracture occurred in the proximal metadiaphyseal region due to osteolysis.

There were six fractures that healed with nonoperative treatment at a mean of 180 days. Five of 10 fractures requiring operative management had failed to heal at a mean of 123 days with nonoperative care. The remaining five fractures underwent immediate operative intervention. All of the fractures healed. One patient required multiple procedures including a free fibula transfer. This patient took 1,116 days from the time of first surgery to heal. Except for this case, it took a mean of 230 days after the first surgery for the fractures to heal.

The authors reported that the data from this study did not clearly indicate the need for surgery in the case of a type A fracture. Type B fractures should be treated with surgery. Open reduction and internal fixation should be performed if the component is well fixed. Revision with a long-stem component is recommended if the component is loose. A trial of nonoperative treatment was recommended among those with type C fractures.

The majority of intraoperative fractures can be prevented. As a part of preoperative planning, any stress-risers in the bone should be identified and osteopenia should be noted. One must be careful twisting the humerus during exposure, bone preparation, or seating of the implant. Forces across the humerus or other bones can be diminished by appropriate release of scar and capsular contractures. For patients with extreme osteopenia, one should consider the anteromedial surgical approach with release of the deltoid origin on the clavicle and anterior acromion rather than the deltopectoral approach. This greatly lessens the forces needed for exposure.

Even with the small experience reported by each individual author, the accumulating experience suggests that recommendations for treatment can be made. If an intraoperative humeral shaft fracture occurs, it should almost always be internally fixed; typically this would be with a long-stem humeral implant and wire cerclage. For very distal fractures, plate fixation may be preferred. Similarly, greater tuberosity fractures should almost always be sutured with heavy, nonabsorbable suture or wire. Postoperative humeral fractures may be considered in three categories in the manner of Wright and Cofield. For proximal fractures that involve the implant and are associated with

implant loosening, revision arthroplasty with insertion of the long-stem humeral implant and cerclage fixation should be strongly considered. For those fractures centered around the tip of the implant without a great deal of proximal fracture extension, external support can be undertaken if skeletal alignment is adequate. If skeletal alignment cannot be achieved or delayed union occurs, open reduction and internal fixation (ORIF) with a plate, screws, and cerclage would be indicated. For fractures involving the distal humerus, treatment would be dictated according to the guidelines for distal humeral fractures. This might include external support in the presence of excellent skeletal alignment or ORIF for displaced, unstable fractures.

Infection

Evolution in thinking regarding infections around the prosthetic joint arthroplasty has been directed toward four categories of infections. The first is a positive culture obtained at the time of the arthroplasty, perhaps associated with previous internal fixation. The second is an acute infection developing within 2 to 3 months following the arthroplasty. The third is an apparently acute, probably hematogenous, infection developing quite typically a year or several years following the initial arthroplasty, and the fourth is a chronic infection, sometimes associated with sinus tract formation.

The recognition of a latent, low-grade infection at the time of the arthroplasty can be somewhat difficult. The joint fluid may be slightly opaque. The synovial lining may include scattered areas of mild erythema, small foci of granulation tissue may be present on the eburnated bone surfaces, and the reactive fibrosis may be somewhat softer and slightly more pink than white. However, all these changes may be absent, or if not, quite subtle. A frozen section may show a few foci of polymorphonuclear leukocytes or in fact may show a mild, rather nonspecific low-grade inflammatory response with lymphocytic cells—not atypical of many patients with arthritis. Occasionally in this setting, particularly in individuals who have had previous surgery and internal fixation, it may be prudent to remove the internal fixation to débride any abnormal tissue, to close the wound, and to await cultures before proceeding with the arthroplasty. Judgment in these cases can be extremely difficult, and it seems there are no clear-cut guidelines for these borderline situations.

When infection develops in the acute phase, within the first months following surgery, distinction between a superficial infection and a deep infection can be difficult. The presence of a postoperative hematoma may also cloud the issue. The patient often has the symptoms and signs of an acute infection, but they may be muted. There may be some malaise of varying degree; a fever may or may not be present; the wound can be slightly more swollen than usual, particularly in the presence of a focal hematoma, or the wound may be slightly to markedly red. There may be drainage, or there may not be. The erythrocyte sedimentation rate will be elevated and probably won't be of much diagnostic value. Serum C-reactive protein holds some promise in identifying patients with subacute infections. Similarly, the white blood count can be abnormal in the acute postoperative phase, but abnormalities in the subacute phase will be suggestive of the presence of an infection. Standard radiographs may show gas in the tissues, but this would be unusual. Scintigraphic techniques are probably of little value. Needle aspiration is usually indicated. In the end, débridement with culture of the deep tissues may be the only way to diagnose an infected shoulder arthroplasty in the immediate postoperative period.

An acute, presumably hematogenous infection developing in a previously well-functioning arthroplasty is usually diagnosed clinically, in association with laboratory studies and joint aspiration.

Distinguishing a late, indolent, chronic infection from a mechanical problem can be easily accomplished or may be extraordinarily difficult. Certainly in a patient with a painful arthroplasty, infection should always be considered. The external examination may be normal. The radiographs, however, may show lucencies surrounding the components. In the humerus there may be resorption of bone, particularly in the area of the humeral calcar; laminated periosteal new bone formation; scalloping of the endosteal bone surface; or evident sinus formation through bone. The more chronic and low-grade the infection is, the more likely it is that the laboratory studies will be normal. Scintigraphy can be quite revealing. Usually the technetium-labeled scan will be positive throughout the arthroplasty region if infection is present. However this can also be active in the presence of noninfectious inflammation or mechanical problems. We prefer to supplement that scan with an indium labeled leukocyte scan. This will not always be clearly abnormal, but when positive, it is highly suggestive of infection. Joint aspiration should be performed, and arthrography should not be forgotten. Arthrography can be quite useful at showing irregularities, that is, hypertrophic changes within the synovium, fistula formation from the joint perhaps communicating with a pseudo-bursa, and dye tracking along the interface of the implant or implant and cement with the bone in an irregular fashion. Despite these tests, we have found preoperative studies to be very ineffective in determining the presence of infection.[62]

Referring to Table 24-1, infections reported in the recent total shoulder arthroplasty literature occurred in 0.4% of cases. Infections are reported somewhat more commonly following humeral head replacement. In the recent literature, Table 24-2, infections are reported in 1% of cases. When we reviewed the literature specifically for complications of humeral head replacement for proximal humeral fractures, delayed wound healing or infection occurred in 6 of 203 acutely treated cases and in 3 of 100 shoulders having

delayed treatment for chronic fracture-related problems.[42] Thus, the literature suggests that the infection rate is less than 1% for elective shoulder arthroplasty and may well be several times that for arthroplasty used for the treatment of complex fractures.

There is surprisingly little literature directed specifically toward infections associated with shoulder arthroplasty. Codd and coauthors reported their experience with 16 patients.[63] They recognized, as formulated earlier in this chapter, that there is no single preoperative study that is consistent for the diagnosis of infection, and they suggested relying quite strongly on the operative histology. Interestingly, their group included three patients who had positive cultures while undergoing revision surgery but negative intraoperative histology. These patients were treated with antibiotics, and none of them developed infection following this treatment. Eight patients presented with infection of their arthroplasty, four underwent resection arthroplasty, and four had reimplantation of a prosthesis with antibiotic-containing bone cement. The patients undergoing reimplantation had better shoulder function. In another subgroup, five patients apparently had earlier shoulder infection, which was treated, and then had humeral head replacement at a later date. This report typifies the information on infected shoulder arthroplasties. There is information available, but the numbers of patients are small and one hesitates to be too forceful in recommendations regarding treatment.

Coste et al. reported on the outcome of 49 shoulders with an infected shoulder arthroplasty in a multicenter study.[64] Radiotherapy and previous surgery were found to be significant risk factors for the development of infection. The authors reported that antibiotics or débridement alone were not effective treatments. They recommended in acute infection that immediate revision should be undertaken with extensive débridement and prosthesis exchange with appropriate antibiotic therapy. Jerosch and Schneppenheim reported on their experience with 12 infected shoulder arthroplasties. Among this group, 10 patients presented with late infection. The authors recommended two-stage revision in this group with placement of a temporary spacer.[65]

Three review articles have additional, useful information on the treatment of infection associated with shoulder arthroplasty. Wirth and Rockwood in 1996 reviewed 1,615 total shoulder arthroplasties in 32 scientific reports.[48] Infection was identified as the fifth most common complication. Reported associations included host-related risk factors of diabetes mellitus, RA, systemic lupus erythematosus, previous shoulder operations, remote sites of infection, compromise of the immune system, and the speculation as to whether or not previous steroid injections predispose to infection in this circumstance. The value of preoperative laboratory testing is difficult to ascertain because of inconsistent reporting methods. Suggested treatment options include antibiotic suppression, incision and drainage, removal of the implant with reimplantation, resection arthroplasty, arthrodesis, and amputation. The specific treatment selected depends on the timing of the infection, the pathogen, and the stability of the implant. For early infections with a Gram-positive organism exploration with incision, drainage, and débridement, the use of antibiotics and retention of the prosthesis is recommended. For late infections and those with Gram-negative organisms, removal of the implant, intravenous antibiotics, and then later consideration of reimplantation is suggested.

Miller and Bigliani in 1993 identified four infections in 1,168 reported cases.[47] They commented that the excellent vascularity and abundant soft tissue coverage is one reason for the low rate of infection. There should always be a high suspicion of the possibility of a low-grade infection at the time of revision surgery. One should be particularly wary of infection in an immunocompromised host. Suggested treatment for early infections included débridement, wound closure with drains, an initial broad-spectrum antibiotic with a high sensitivity to Gram-positive organisms, and more specific antibiotics as the culture results become apparent. For delayed infections, removal of the implant is suggested along with the use of intravenous antibiotics and immobilization for 3 months. If the patient is dissatisfied at 1 year, the authors discussed the possibility of performing a fusion if there is no osteomyelitis. It is possible to reinsert a hemiarthroplasty or TSA with antibiotics impregnated in the cement, but osteomyelitis should certainly not be present in this circumstance.

Soghikian and Neviaser in 1993 reviewed complications of humeral head replacement.[49] They recommended that one be quite suspicious of infection in the presence of previous failed ORIF, that perioperative antibiotics be used, that hemostasis be meticulous, and that intraoperative cultures be obtained. When late infection develops, the implant should be removed, there should be antibiotics for a minimum of 6 weeks, and reimplantation should be considered at that time or later.

Sperling et al. reviewed the results of infected shoulder arthroplasties at the Mayo Clinic.[66] Between 1972 and 1994, 2,512 primary shoulder arthroplasties and 222 revision shoulder arthroplasties were performed at the authors' institution. Among these shoulder arthroplasties, 19 primary shoulder arthroplasties and seven revision shoulder arthroplasties were diagnosed with deep periprosthetic infection. Additionally, during this time period, seven primary shoulder arthroplasties and one with a previously revised shoulder arthroplasty were referred for treatment of deep periprosthetic infection. Two shoulders were excluded because of incomplete medical records and with component removal performed elsewhere. The mean time from arthroplasty to the diagnosis of infection was 3.5 years.

The patients were divided into four groups on the basis of treatment. Group I comprised 21 shoulders that underwent

resection arthroplasty. Six of these shoulders had additional episodes of infection. Group II comprised six shoulders that underwent débridement and prosthetic retention. Three shoulders failed this treatment with subsequent reinfection and underwent a resection arthroplasty. Group III comprised two shoulders that had removal of the prosthesis, débridement, and immediate reimplantation. There was one resection arthroplasty 9 months after direct exchange because of reinfection. Group IV comprised three shoulders that had removal of the prosthesis, débridement, and delayed reimplantation. Reinfection did not occur in any of these patients. At the most recent follow-up, patients with a prosthesis had better pain relief and shoulder function than patients treated with resection arthroplasty. The authors concluded that delayed reimplantation may offer the best hope for pain relief, eradication of infection, and shoulder function (Fig. 24-4).

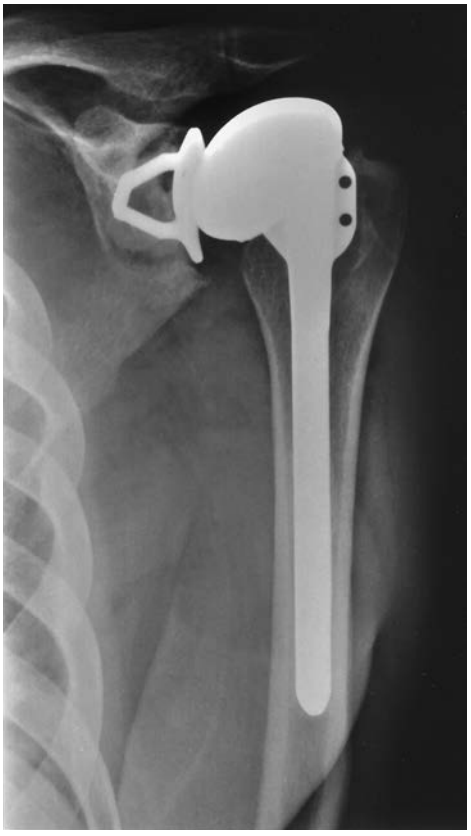
In summary, there are many variables involved with infections surrounding arthroplasties including host factors, the timing and nature of the infection, and the type of organism. For the acute or delayed acute infection, arthrotomy and débridement and the use of intravenous antibiotics should be strongly considered, but this advice must be tempered given the reinfection rate, which is difficult to ascertain from the literature but is moderate, perhaps somewhere between 30% and 60%. In the past, resection arthroplasty was the standard. It is successful for eradication of the infection and usually provides pain relief. Shoulder function is often unsatisfactory if rated on one of the usual shoulder rating systems. Direct exchange of the prosthesis can be considered for lower-grade infections, but the frequency of success is uncertain, due to the limited collective experience with this type of treatment in the shoulder. Delayed reimplantation has been successful for eradication of infection. It is more consistent in offering pain relief and affords better function than resection arthroplasty. The timing to reimplantation is uncertain. The suggested times extend from 6 weeks to 1 year.

Rotator Cuff Tears and Impingement Syndrome

Problems with rotator cuff healing or subsequent rotator cuff tearing following arthroplasty are probably substantially underreported, being identified instead as poorer outcomes and included in the lower portions of result rating scales or detailed in association with instability rather than specifically identified as a rotator cuff stretching or tearing. Certainly, management of the rotator cuff is an important part of shoulder arthroplasty. Preexisting rotator cuff disease and tearing is common. We carefully studied 156 consecutive total shoulder arthroplasties for rotator cuff disease. Overall, 48 (27%) had tears of the rotator cuff present at surgery. Tears did vary in size, being small (less than 1 cm in greatest length) in 10, medium (1 to 3 cm) in 10, large (3 to 5 cm) in seven, and massive (greater than 5 cm) in 21. By disease category, in osteoarthritis there was no tearing in 46 shoulders and the rotator cuff was thin in one and torn in six. In RA there was no tearing in 11 and the rotator cuff was judged to be thin and scarred in 37 and was torn in 18. In old trauma there was no abnormality in six, the rotator cuff was thinned and scarred in two and was torn in three, and in 10 there was tuberosity malunion requiring osteotomy. In rotator cuff tear arthritis the rotator cuff was of course torn in all 14. In other miscellaneous diagnoses there was no rotator cuff tearing in 13 and the rotator cuff was thin and scarred in two and torn in seven. Thus, preexisting rotator cuff disease is an important consideration, may well alter the approach to shoulder arthroplasty, and certainly if tearing is present would predispose the patient to having a complication related to the rotator cuff postoperatively.

The classification of rotator cuff tearing is probably no different with arthroplasty than without arthroplasty. The classification can be by location, tear size, and tear shape. Location identifies the tendon or tendons affected. Size is conveniently recorded in centimeters and is typically measured following the initial débridement before mobilization of the tendons themselves. Many cuff tears are in the impingement area or slightly posterior to this, associated with upward subluxation of the humeral head that may occur in many patients with arthritis. Occasionally longitudinal tears occur in line with the tendon fibers, but most often transverse tears exist. These may be essentially linear or they may include an area defect that has an oval, triangular, or trapezoidal shape, prior to mobilization of the tendon into the defect. Additionally, shoulder arthroplasty has made surgeons acutely aware of the possibility for interstitial tendon tearing, particularly in RA, where the tendons are quite thin and scarred, perhaps one-half to one-quarter of their normal thickness. Certainly if these tendons were examined histologically there would be disruption of tendon fiber continuity; thus, a microscopic rotator cuff tear exists in the absence of a gross full-thickness tear. It has been shown that the outcome of arthroplasty in these patients is strongly influenced by this compromise of rotator cuff function, although stability of the implant can usually be obtained.

The diagnosis of rotator cuff tearing following arthroplasty follows the scheme of diagnosis for rotator cuff tearing in the absence of arthroplasty. Symptomatically there is often an increase in pain, recognition of a loss of active motion, and weakness. Physical examination defines this diminution in active motion relative to passive motion and identifies weakness in the torn cuff components. Additionally, following arthroplasty, rotator cuff tearing may present most dramatically as instability, usually superiorly or anteriorly but occasionally posteriorly. Certainly when instability occurs following shoulder arthroplasty, disruption of the surgical arthrotomy or rotator cuff tearing



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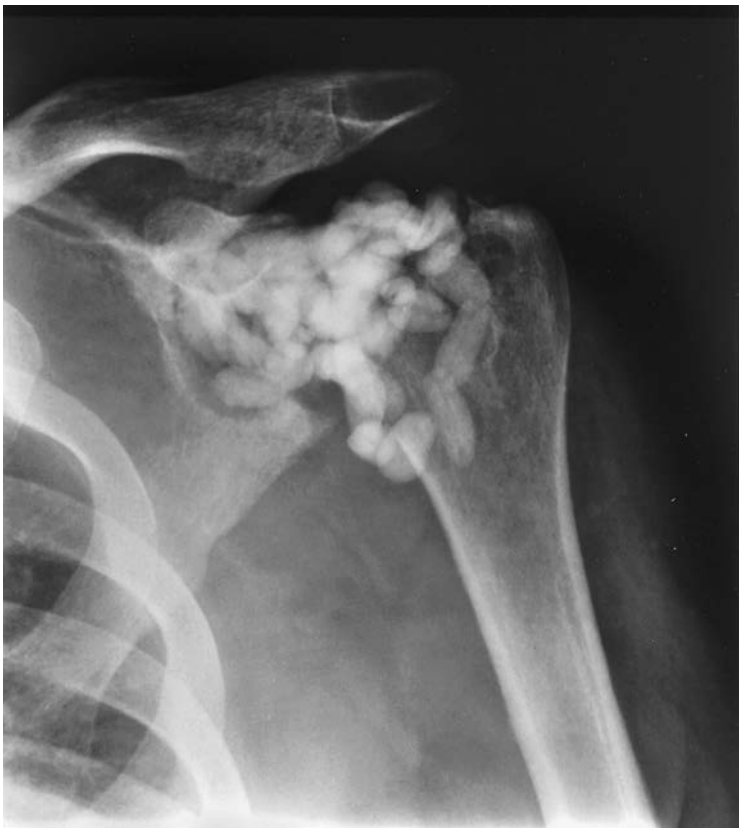

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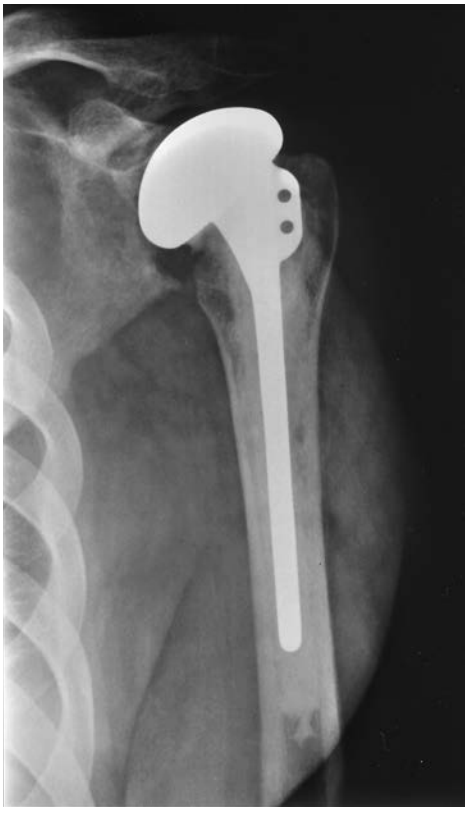

C

Figure 24-4 Example of a 60-year-old man who underwent shoulder arthroplasty for osteoarthritis. He presented 6 years following surgery with increasing pain and erythema. Radiograph demonstrates glenoid component loosening with proximal humeral osteolysis **(A).** He underwent component removal, débridement, and placement of antibiotic impregnated beads **(B).** Two months after component removal, he underwent delayed reimplantation. At the most recent follow-up, 2 years following surgery, he had no pain, 140 degrees of active abduction, and 45 degrees of external rotation **(C).** (Reproduced with permission from Sperling JW, Kozak TKW, Hanssen AD, Cofield RH. Infection after shoulder arthroplasty. *Clin Orthop* 2001;382:206–216.)

should always be suspected. Plain radiographs may show a severe upward subluxation diagnostic of rotator cuff tearing. In the absence of this but with clinical evaluation suggesting rotator cuff tearing, an arthrogram can be performed. With a tendency for fibrosis surrounding implants there is concern that the arthrogram may be false negative; however, practically this seldom seems to be an important issue, with the arthrogram typically defining the rotator cuff tear much as it would in a shoulder without prosthetic arthroplasty. Recently, the technique of performing magnetic resonance imaging (MRI) in a patient with a shoulder arthroplasty has become available.[67] This may be a valuable tool to determine the size of the tear, location of the tear, and degree of fat infiltration of the tendon.

A special note relative to rotator cuff tearing in the postoperative condition relates to healing of the subscapularis. We have recognized that a number of patients following arthroplasty have on physical examination somewhat excessive external rotation and slight weakness in internal rotation. They must in fact have stretched their anterior shoulder capsule and rotator cuff repair or have disrupted it to some degree. In many of these patients there is no patient-identified adverse response with excellent pain relief and motion and maintenance of glenohumeral stability. As such, one cannot assume that disruption of the rotator cuff is the source of pain following shoulder arthroplasty. Other possible sources of pain must also be explored and identified, as apparently rotator cuff tearing can exist following shoulder arthroplasty, and as in the nonarthroplasty state can be minimally symptomatic or asymptomatic.

Although problems with the rotator cuff following arthroplasty are seldom at the forefront of one's thinking when considering complications of shoulder arthroplasty, if one tabulates all the complications of TSA as defined in Table 24-1, rotator cuff tearing is the most common complication in this collection of reported series, having a rate of 1.9%. Following humeral head replacement, rotator cuff tearing occurs in a similar frequency (1.8%) (Table 24-2). Tuberosity problems also occur in fracture patients, with nonunion developing in 1.8% and tuberosity malunion in 0.4% in this group of 20 patient series.

Neer and Kirby, when discussing revision of humeral head and total shoulder arthroplasties, mentioned the possibility of impingement of the rotator cuff in the postoperative state.[68] They said that scar requires release and impingement by the anterior acromion, the coracoacromial ligament requires anterior acromioplasty, and impingement by the acromioclavicular joint requires distal clavicle excision to free the rotator cuff. Miller and Bigliani, in presenting the complications of total shoulder replacement, mentioned a postoperative rotator cuff tear frequency of 3% to 4%.[47] They commented that repair is not always required but recalled the admonition of Franklin et al.'s report[69] that there is an increased frequency of glenoid loosening when there are large rotator cuff tears; this might support repair of a rotator cuff tear in the postarthroplasty patient. Wirth and Rockwood estimateed a 2% frequency of rotator cuff tearing following shoulder arthroplasty and found that both operative and nonoperative treatment have been employed with lack of clear definition of the benefits of one form of treatment over the other.[48]

Arthroscopic acromioplasty has been used for the treatment of impingement following shoulder arthroplasty.[47] Freedman and colleagues reported on the results of six patients who underwent arthroscopic acromioplasty for chronic impingement syndrome following shoulder arthroplasty.[70] The results were excellent or good in five patients and unsatisfactory in one patient.

Probably in no area is the comprehensive approach to shoulder arthroplasty more important than in the prevention of postoperative rotator cuff tearing. Balancing the joint is key to preventing large repair-threatening forces on the arthrotomy site or a repair of a preexisting rotator cuff tear. Component positioning must be carefully performed, the correct size of the component must be selected, unbalanced capsular tightness must be adjusted by appropriate releases or adjustments in component size, and impingement should be eliminated if it exists. Following implantation of the prosthetic parts, the glenohumeral joint should be stable and in fact will usually be stable even prior to the arthrotomy repair. The arthrotomy repair should then be secure. Range of motion of the shoulder should be assessed to define the limits determined by the arthrotomy repair. Sufficient postoperative limb support should be provided, as should careful patient instruction in protecting the limb. The physiotherapy program takes into consideration the limits of motion as defined at the time of surgery and the surgeon's recognition of the rate of soft tissue healing.

There does not seem to be a yes-or-no answer in the recommendations for treatment of rotator cuff tearing following TSA. Certainly, as mentioned above, there are a number of patients who seemingly have compromised their arthrotomy repair, yet are comfortable and have good motion and the arthroplasty is stable. Further surgical treatment would almost certainly not be considered in these patients. Other patients express some weakness of their shoulder following arthroplasty, yet have good pain relief and fair active movement. Again, in many of these older patients surgical repair of their rotator cuff stretching or tearing would not be undertaken. On the other hand, there are some patients who have been doing well with their arthroplasty who have an adverse event or an accident and have a dramatic change in shoulder function, or those who without apparent reason or following an injury develop acute instability of their arthroplasty. In these patients with a rather acute and dramatic change in their arthroplasty with substantial compromise of function, rotator cuff repair would be undertaken. Finally, there are a few patients who

seem to have chronic deterioration of function in their shoulder arthroplasty. Usually this can be attributed to other problems (such as component loosening), but occasionally this is related to a degenerative or impingement-engendered attrition and tearing of the rotator cuff. In these patients, as in patients without shoulder arthroplasty, the situation should be defined and a discussion held with the patient about the benefits and limitations of rotator cuff repair. In this setting, it seems that the benefit/risk ratio is rather close, and typically, continued conservative treatment measures are recommended.

Instability

It is easiest to organize thinking about shoulder instability by utilizing the established categories for shoulder instability in the absence of shoulder arthroplasty. Instability following shoulder arthroplasty does occur acutely. Chronic dislocations can occur and, in fact, so do recurrent dislocations. The instability can be of any degree—mild subluxation or complete dislocation—and it can be in any direction—superiorly, anteriorly, posteriorly, or inferiorly. Perhaps the most common instability situation that occurs following shoulder arthroplasty is some degree of superior humeral subluxation. Boyd et al. commented on this and recognized it in 29 of 131 shoulders (22%) having a Neer prosthesis.[51] In their patient group, even in the presence of this situation, all patients had decreased pain compared to their preoperative state and improved range of motion. This upward subluxation seemed to be caused by a combination of factors and was not due to rotator cuff tearing alone, as they recognized major rotator cuff tears in 21% of patients with a normal glenohumeral relationship and in 24% of patients with this proximal or superior subluxation.

The presence of instability is usually readily recognized by physical examination and radiography. However, there may be associated abnormalities that require more in-depth study including the presence or absence of rotator cuff tearing, component malposition, a component size that is either too large or too small for the soft tissue envelope, or component loosening with a change in component position.

In the literature instability is one of the most common complications seen following TSA, occurring in 1.5% of shoulders (Table 24-1). It may even be the most common complication following humeral head replacement, with 2.8% of the shoulders developing this complication in the 20 series reported since 1980.

There have been two scientific articles directly addressing postoperative instability. Moeckel et al. reported on 10 shoulders developing instability following 236 consecutive arthroplasties.[34] The instability was anterior in seven and posterior in three. They recommended correction of the soft tissue imbalance and prosthetic revision as necessary. With particular reference to disruption of the subscapularis tendon, mobilization and repair of the tendon was recommended, but three of seven continued to have instability and required re-repair supplemented with an allograft of tendo Achilles. This latter technique proved successful in these three shoulders. Wirth and Rockwood reported on 18 shoulders developing instability, 11 posterior, six anterior, and one inferior.[48] For posterior instability, the treatment was directed at restoring appropriate retroversion of the humeral component, performing a posterior capsulorrhaphy, and achieving appropriate position of the glenoid component or in the absence of a glenoid component reaming the glenoid surface to a neutral angle. For anterior instability, component version was again corrected when necessary. The anterior structures were repaired and sometimes reinforced with the pectoralis major tendon. If the coracoacromial ligament could be reconstructed, this was also done. For inferior instability, humeral length was restored.

Several review articles comment on shoulder instability. In Neer and Kirby's 1982 article on revision of humeral head and total shoulder arthroplasties, they recognized loss of humeral height as extremely common following fractures or tumors and, indeed, that it occurred in 18 of 27 failed arthroplasties in this series.[68] At revision surgery the height was restored and comment was made that bone grafting might be needed. Miller and Bigliani identified instability in 1% to 2% of cases.[47] He also recommended restoring humeral length, careful attention to appropriate glenoid positioning when using a glenoid component, or appropriate reaming of the glenoid surface when a glenoid component is not used. At revision surgery it would usually be necessary to correct prosthetic position and to perform soft tissue reconstruction. Wirth and Rockwood's 1996 review of complications of total shoulder replacement noted that anterior instability was associated with dysfunction of the anterior deltoid, malrotation of the humeral component, and disruption of the subscapularis tendon.[48] This latter problem was attributed to poor tissue quality or technique, inappropriately oversized components, or aggressive physical therapy. Superior instability was associated with dynamic muscle dysfunction, attenuation of the supraspinatus muscle–tendon unit, or rotator cuff tearing. This type of instability might be associated with an increased frequency of glenoid loosening. Posterior instability was associated with a retroverted glenoid component, an excessively retroverted humeral component, and soft tissue imbalance. If the glenoid component was not used, posterior instability was associated with posterior glenoid erosion. They recommended being aware of asymmetrical glenoid wear and compensating for this with careful reaming or bone grafting of the glenoid. Also, the surgeon must alter the position of the humeral component to include somewhat less retrotorsion. Treatment for posterior instability would include restoring normal humeral component version, reestablishing proper glenoid compo-

nent version, and addressing soft tissue imbalance, including consideration of posterior capsulorrhaphy. Inferior instability was seemingly always associated with humeral shortening. The treatment was to re-establish humeral length. Soghikain and Neviaser, in reviewing the complications of humeral head replacement, identified superior subluxation as an issue and recommended careful assessment of the rotator cuff during the initial surgery and carefully positioning the height of the humeral component.[49] Adequate postoperative protection is necessary to allow soft tissue healing. Should a postoperative dislocation occur, closed reduction should be performed, followed by immobilization and delayed physiotherapy.

In the original Mayo Clinic series analyzing postoperative surgical complications between 1979 and 1989, subluxation was the most common complication identified (11%). Subluxation was usually a devastating complication. As this complication was the most common one in our patients in the original series and was quite significant, a separate analysis of instability following shoulder arthroplasty was undertaken.

The results of surgical treatment for the unstable shoulder arthroplasty at the Mayo Clinic were recently reviewed.[71] Between 1985 and 1999, 33 shoulders (seven hemiarthroplasties and 26 total shoulder arthroplasties) underwent surgical treatment for instability. There was anterior instability in 19 shoulders and posterior instability in 14 shoulders. The primary arthroplasty had been performed for the treatment of osteoarthritis in 16 shoulders, arthritis of dislocation in six, acute fracture in four, RA in three, and other conditions in four.

Prosthetic instability was attributed to abnormal capsular tension and/or rotator cuff dysfunction in 21 shoulders, malposition of the components in one shoulder, and a combination of both in 11 shoulders. One shoulder was treated with resection of the components. In the remaining 32 shoulders, each of the contributing elements to instability was specifically addressed at the time of revision surgery. Revision surgery was successful in restoring stability in only 9 of the 32 shoulders. Anterior instability had a higher failure rate than posterior instability ($P = 0.04$). Although 11 shoulders had additional surgery to treat recurrent instability, only 14 of the 33 shoulders were stable at the time of the most recent follow-up. Based on a Neer rating system, there were four excellent, six satisfactory, and 23 unsatisfactory results.

The authors noted that soft tissue imbalance was present in most cases of instability following shoulder arthroplasty, and component malpositioning played an additional role in some cases (Fig. 24-5).[72] Unfortunately, more than one-half of the shoulders in the study remained unstable despite attempts at revision. As such, preventing subluxation and dislocation is the key. Identifiable risk factors for postoperative shoulder instability would apparently include an older patient, preexistent rotator cuff disease, preoperative subluxation, or dislocation. Additional factors would include irregular (i.e., asymmetrical) glenoid wear (usually posterior), the use of prosthetic parts that are too small for the surrounding soft tissue envelope, or the use of prosthetic parts that are too large, creating undue stress on the arthrotomy repair with a potential for disruption of the repair early in the postoperative period. Of course, component malposition should be avoided and rotator cuff and capsule repair should be secure with appropriate postoperative external support and a carefully designed therapy program.

The treatment plan includes identifying all factors associated with the instability (involving the bone, the implants, and the soft tissues), and surgery is directed toward addressing all of these abnormalities. Postoperative support should then be ample, and the therapy program would be modified to maintain stability.

Component Problems

Component loosening remains one of the central concerns in shoulder arthroplasty, particularly in regard to the glenoid component. Unfortunately, radiographs about the shoulder can be quite poor and not show the interfaces well, particularly if there is metal backing to the glenoid component, for only slight change in angle of the x-ray beam will obscure interface changes. The 40-degree posterior oblique view is an improvement over the standard anterior–posterior view in assessing the interface between the glenoid implants and the bone of the scapula. We have found fluoroscopically positioned spot views to be most consistent for evaluating not only the glenoid component, but also the humeral component.[73] It is also useful to have a sequence of x-rays because a change in component position may be apparent with a series of films when it is not clearly demonstrated on a film taken at one point in time.

By recognizing the characteristic patient presentation and having high-quality x-rays, over time it is usually possible to diagnose component loosening without needing to resort to more complex studies; however, occasionally these might be necessary and could include arthrography. Arthrography in addition to outlining the rotator cuff tendons will display synovitis, which is often present in situations where component loosening occurs. Also, dye can track between the bone-cement interface. However, the accuracy of this test for the diagnosis of component loosening has not been fully assessed. Shoulder arthroscopy has been suggested as one means to diagnose glenoid loosening.[74]

In the series of total shoulder arthroplasties published since 1980, glenoid loosening was the third most common complication (1.3%), and humeral loosening was recognized to occur in 0.3%. In these series the problem was treated with component revision slightly more than one-half the time and with removal of the loosened glenoid component, generally retaining the humeral head replacement, in

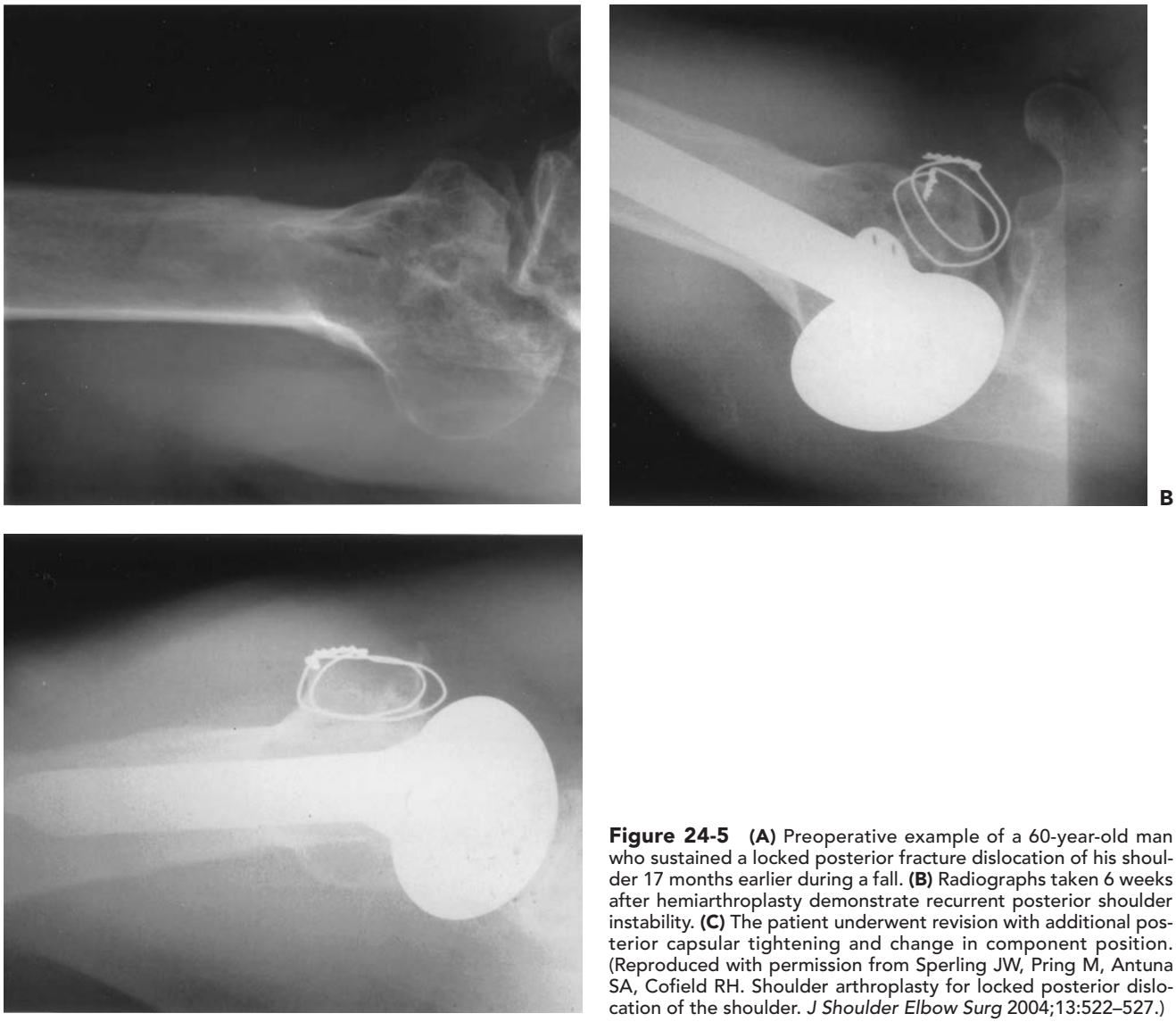

Figure 24-5 **(A)** Preoperative example of a 60-year-old man who sustained a locked posterior fracture dislocation of his shoulder 17 months earlier during a fall. **(B)** Radiographs taken 6 weeks after hemiarthroplasty demonstrate recurrent posterior shoulder instability. **(C)** The patient underwent revision with additional posterior capsular tightening and change in component position. (Reproduced with permission from Sperling JW, Pring M, Antuna SA, Cofield RH. Shoulder arthroplasty for locked posterior dislocation of the shoulder. *J Shoulder Elbow Surg* 2004;13:522–527.)

slightly less than one-half the time. There is little information on the treatment of loose humeral components. One of the authors in this series of reports identified two loose humeral components, but no treatment was undertaken as the patients had no pain. In another series, revision was performed because of symptomatic humeral loosening in one shoulder.

In the recent series on humeral head replacement, humeral loosening was again recognized in less than 1% of shoulders (0.6%). These cases of humeral component loosening were only recognized in a series of isoelastic prostheses and no treatment was mentioned.

Glenoid component loosening has been identified in conjunction with severe incompletely reconstructible rotator cuff tears or following the development of rotator cuff tearing in the postoperative period. Franklin et al.'s seven cases occurring in this setting all exhibited large glenoid bone-cement lucent lines, often in association with a shift in component position.[69] This article was published to remind us of the importance of the rotator cuff in maintaining joint position and in preventing eccentric and excessive component loading and presumably subsequent component loosening.

Review articles are more ample in their discussion of glenoid component loosening. Miller and Bigliani in 1993 described that radiolucent lines were common but that there was apparently no direct correlation to the level of clinical symptoms.[47] They felt that lucent lines may reflect the surgical technique, the quality of the glenoid bone, and changes affected by stress shielding or disuse osteoporosis. Also, they recognized that radiographic technique must be excellent to detect these lucencies in this anatomic region. They felt that loosening could be minimized by careful bone preparation at the time of surgery, fitting the implant

closely to the bone, and using a minimal amount of cement. Humeral loosening was recognized to be rare. Subsidence of the prosthesis can occur in osteoporotic bone, and the use of bone cement to fix the humeral component was suggested. Wirth and Rockwood, in reviewing 32 reports of TSA, identified that loosening represented nearly one-third of all complications and was in fact the most frequent complication reported in those studies they reviewed.[48] They stated that methods to enhance component fixation included concentric spherical reaming of the glenoid surface, preservation of the majority of the subchondral plate, a slight mismatching of the radii of the glenoid and humeral heads, and the consideration of press-fit or tissue ingrowth implants.

Brems evaluated the relationship between lucent lines and revision surgery.[75] In a review of 20 reported series on TSA, there were lucent lines present in 38.6% of all total shoulder arthroplasties at a mean follow-up of 5 years. Among the shoulders with periprosthetic lucency, the rate of revision surgery was 7.7%.

Longer-term follow-up of total shoulder arthroplasties and humeral head replacements is more informative concerning component loosening. Of 113 total shoulder replacements performed at the Mayo Clinic between 1975 and 1981, the probability of implant survival was 93% at 10 years and 87% at 15 years. Seventy-nine of these patients with 89 arthroplasties were available for follow-up an average of 12.2 years following surgery, with a range of follow-up from 5 to 17 years. Seventy-five glenoid components developed bone-cement radiolucencies, and 39 glenoid components (44%) were considered to have radiographic evidence of definite glenoid loosening. This loosening was statistically associated with an increase in pain. Additionally, a change in position of the humeral component occurred in 49% of press-fitted stems but in none of the shoulders with cemented humeral stems. Humeral component loosening was not associated with pain. In this group of patients, glenoid component revision was carried out in four, so although the radiographic frequency of glenoid loosening was quite high, the clinical need for revision surgery remained low even with long-term follow-up. Two humeral components required a revision because of loosening. One humeral component was found to be loose at the time of revision of a loose glenoid component and was revised at that time. A second patient developed humeral component loosening in association with a humeral shaft fracture and revision was undertaken using a long-stemmed humeral component and fracture fixation.

In the series at Mayo of 67 humeral head replacements performed for osteoarthritis or RA, follow-up was possible for an average of 9.3 years. Only one of these prostheses was fixed in place with bone cement. All the remaining Neer implants were press-fitted. Six of the 35 shoulders with osteoarthritis had a lucent zone 1.5 mm or greater surrounding the humeral component, and in three of these there was a change in humeral component position downward in the humeral canal. Three of the 32 shoulders with RA had a 1.5-mm or greater lucent line surrounding the majority of the humeral stem. Nine of the components in rheumatoid patients had a change in component position with the component shifting downward in the humeral canal. In these patients with substantial radiographic changes relative to the humeral component, only one in the osteoarthritic group had severe pain and two in the rheumatoid group had moderate or severe pain. Thus, there was no clear relationship between the development of symptoms and the radiographic appearance of the component. But, of course, it must be clearly noted that the frequency of radiographic changes surrounding these press-fit Neer components was quite high and suggests that fixation with bone cement for this type of implant would be preferred.

Glenoid arthritis has evolved to become the most predominant reason for revision of hemiarthroplasties at our institution (Fig. 24-6).[76,77] Sperling and Cofield reported on the outcome of revision of hemiarthroplasty to TSA for glenoid arthritis at the Mayo Clinic.[78] Between 1983 and 1992, 22 hemiarthroplasties underwent revision for glenoid arthritis. Eighteen shoulders with a minimum 2-year follow-up were reviewed. Thirteen of the hemiarthroplasties were performed at our institution and five were performed elsewhere. There was significant pain relief with revision to TSA. However, 7 of 18 had an unsatisfactory result rating due to limitation of motion or need for an additional procedure.

Given the limitations of the current technology, it may be impossible to fully eliminate glenoid loosening; however, as is outlined in the above series, glenoid loosening that is clinically symptomatic enough to require revision surgery is uncommon, but all attempts to reduce the incidence of component loosening will be quite worthwhile in improving the consistency of shoulder arthroplasty. The surgeon can do little, if anything, to address the bone quality that the individual has; however, the amount of bone in the glenoid is quite small and certainly the bone should be preserved as much as possible during insertion of an implant. This includes preserving much of the subchondral plate of the glenoid and very meticulous removal of only that quantity of bone necessary to insert the implant. Certainly, very little reaming should be performed on the glenoid surface. Only the amount of subchondral plate needed to insert the columns or keel of the component should be removed. Further preparation would include pulsatile water lavage, a thorough drying of the glenoid bone, mixing the bone cement to diminish porosity using a vacuum system or centrifugation, pressurization of the bone cement, impaction of the component into position, and maintaining the stability of the component against the bone until the cement hardens.

The choice of a glenoid component remains controversial. There continues to be significant debate concerning

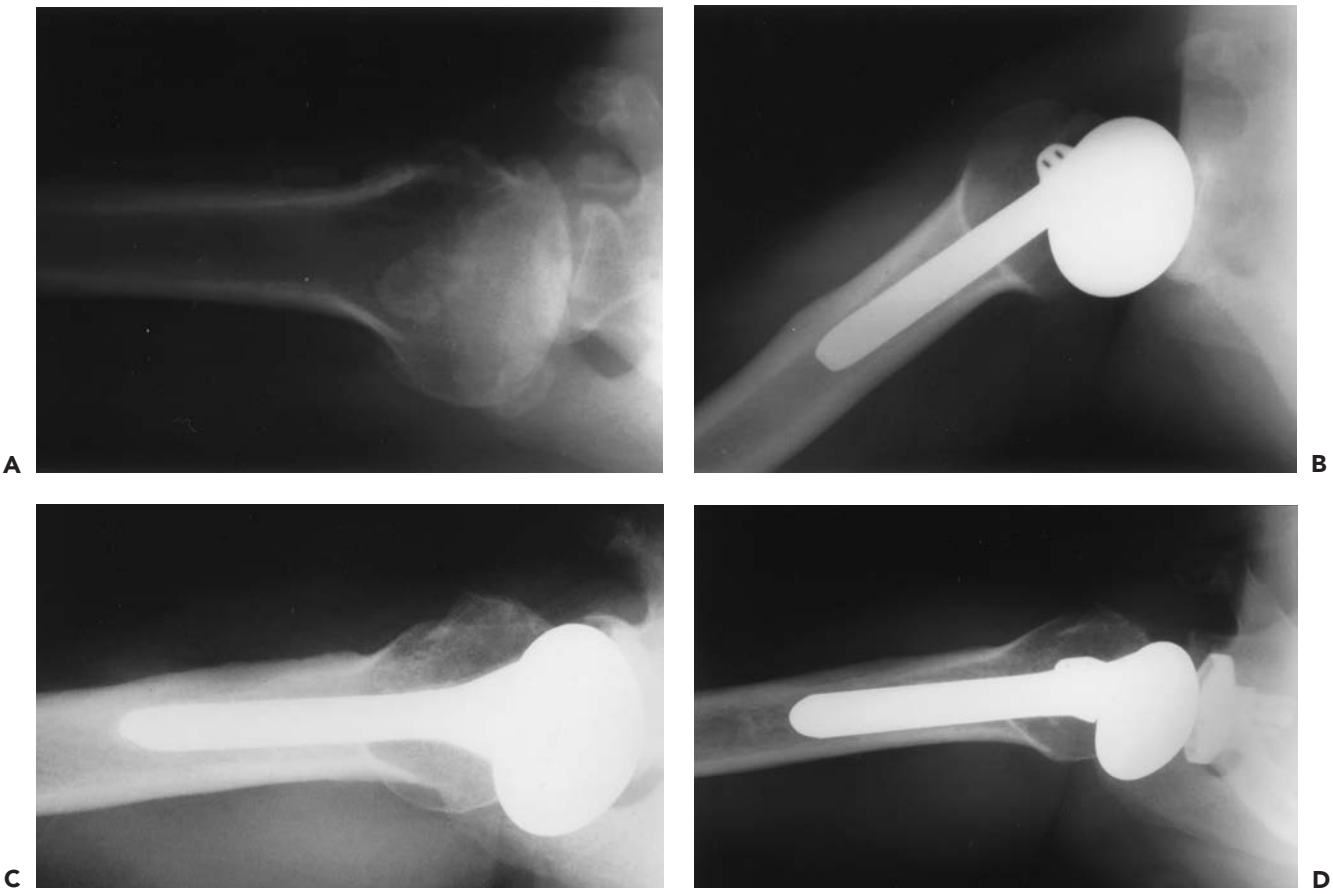

Figure 24-6 **(A)** Example of a 41-year-old man with osteoarthritis. **(B)** Radiograph taken immediately after surgery. **(C)** Radiograph taken 17 years following hemiarthroplasty demonstrates severe glenoid arthritis. **(D)** The patient underwent revision to total shoulder arthroplasty. (Reproduced with permission from Sperling JW, Cofield RH, Rowland CM. Minimum fifteen-year follow-up of Neer hemiarthroplasty and total shoulder arthroplasty in patients aged fifty years or younger. *J Shoulder Elbow Surg* 2004;13:604–613.)

the shape of the glenoid component as well as mismatch between the radius of the glenoid component and humeral head.[79–82] Additionally, the choice of a humeral component also remains controversial and is beyond the scope of this chapter.

Clearly, the joint must be well balanced. Tight capsular structures should be lengthened or divided to lessen unnecessarily high compressive forces across the joint. The rotator cuff should be repaired as best possible and the humeral head should be positioned against the glenoid to eliminate subluxation, as that may create loading of the edge of the glenoid component and subsequent component loosening.

It seems apparent that many glenoid components that have loosened do not require revision surgery. If a patient becomes substantially and continually symptomatic, it is important to exclude other problems as a cause of the symptoms, such as an occult low-grade infection, rotator cuff tearing, or humeral loosening. All of these things, of course, may occur independently or in association with glenoid loosening. Recent data from our institution indicate that satisfactory pain relief is obtained in 86% of patients who have placement of a new glenoid component at the time of revision compared to removal alone in 66%.[83]

Less common complications related to shoulder components include dissociation and component fracture. There have been reports of dissociation of modular humeral head components as well as fatigue fractures of the humeral stem.[84,85] Blevens and colleagues examined the conditions that may affect the Morse-taper interface strength in humeral components. They reported that as little as 0.4 mL of fluid could significantly prevent fixation of the modular head.[86]

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25 Techniques for Revision Arthroplasty: Management of Bone and Soft Tissue Loss

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INTRODUCTION

Failure of a total shoulder arthroplasty is one of the most difficult problems to surgically manage, and most circumstances result in a complex reconstruction. Case complexity is due to a wide variability and sometimes unpredictability of the pathology, often resulting in severe loss of soft tissue and bone. The literature has few studies devoted to revision shoulder arthroplasty; therefore, most of this chapter will convey the authors' approach based on experience treating the most difficult cases. The first critical decision for the surgeon is deciding which patients need revision surgery and which goals are reasonable for each case. Once the problem is identified, the surgeon will have to

anticipate common problems encountered during revision surgery, which requires meticulous preoperative planning and a thorough understanding of the normal shoulder anatomy. Most importantly, the surgeon should expect to improvise and deal with soft tissues or bone that cannot be restored to normal.

PATIENT COMPLAINTS

The most common presenting complaint of a patient with a failed shoulder arthroplasty is pain, which can occur at rest, with activity, or during sleep (Table 25-1). Loss of function is the next most common symptom that the patient describes, and this can be in the form of stiffness or loss of strength. These complaints are usually described together, but it is important for the surgeon to prioritize the complaints from the patient's perspective to assess patient expectations and better define reasons for pain and functional limitation. It is often useful for the surgeon to define the patient's functional limitations as they relate to stiffness, weakness, and instability. It is also beneficial to define the anatomic reasons for each of these complaints: pain, stiffness, weakness, and instability. Once the anatomic causes are defined it is then recommended to make a mental list of how each of these factors can be reversed or corrected. The ability to correctly define and correct each anatomic factor will define the likelihood and level of success that can be achieved with revision surgery. The most experienced surgeons have the ability to make an accurate assessment of the factors and know their own ability and success rate for reversal of these processes.

Pain can be caused by loose implants, stiffness, infection, implant malposition, instability, implant wear, and impingement. Loss of strength can be caused by rotator cuff tears, muscle atrophy, deltoid insufficiency, and nerve injury. Stiffness can be caused by capsular contracture, malposition of implants, pain, and bony impingement. All three primary symptoms are interrelated, and determining how they are interrelated can be challenging.

Documenting the chronology of the patient's pain, strength, and motion in relation to their postoperative recovery can be helpful. Pain that persists immediately after surgery is consistent with infection and malpositioned implants that overstuff the joint. Stiffness immediately after surgery is consistent with malpositioned implants, persistent capsular contracture, and persistent bone impingement. Stiffness that develops over time after surgery can be due to poor patient compliance with physical therapy and excessive pain that limits full participation in rehabilitation. Loss of strength should not be confused with stiffness. Patients are frequently diagnosed with rotator cuff tears by physical examination because they cannot raise their arms above shoulder level, but if the surgeon takes the time to examine the patient in the supine position, the arm may be limited to only 90 degrees of passive motion. Loss of strength that occurs immediately after surgery may be due to nerve injury or intraoperative muscle injury. More commonly, patients complain of loss of endurance or weakness that occurs secondary to poor muscle rehabilitation or rotator cuff tears. Further investigation with laboratory studies and radiographs will also help diagnose the primary cause of failure.

TABLE 25-1
CAUSES FOR FAILED ARTHROPLASTY

Symptom	Cause
Loss of motion	Bony block malunion
	Prosthetic position
	Prosthetic size
Instability	Incorrect glenoid version
	Glenoid wear
	Rotator cuff deficiency
	Coracoacromial arch deficiency
Weakness	Rotator cuff tear
	Deltoid atrophy
	Neurologic injury
	Loss of tuberosities
Pain	Infection
	Glenoid arthritis
	Stiffness
	Loose components

PHYSICAL EXAMINATION

The physical examination of the failed shoulder arthroplasty should not be different from the examination of a normal shoulder. Careful assessment should include the passive and active arcs of motion; visual inspection of prior incisions and muscle atrophy; strength of the deltoid, external rotators, and subscapularis; and pain-inciting maneuvers. Particular attention should be given to possible superior escape of the humeral head during active elevation of the arm, false-positive belly press test secondary to internal rotation stiffness, anterior deltoid deficiency, and anterior or posterior subluxation of the humeral head that is either static or dynamic.

It is important to remember that patients with pain and limitations of function after prosthetic arthroplasty do not always have a problem with their prosthetic or the surgery initially performed. Patients with well-functioning implants and an anatomic reconstruction (x-ray findings) can have good range of motion, stability, and strength but have problems associated with a painful acromioclavicular joint, subacromial impingement, or rotator cuff or biceps tendon pathology. It is important to consider these diagnoses and evaluate these on physical examination and radiographic studies, particularly when

the joint on examination seems to function well but the patient complains of persistent pain.

DIAGNOSTIC WORKUP

Patients who have a failed shoulder arthroplasty should have a sedimentation rate sent to the laboratory, and if it is elevated an infection should be suspected. In all cases the shoulder should be aspirated to evaluate for an infection. Routine radiographs including a true anteroposterior (AP) radiograph of the shoulder and an axillary lateral of the shoulder should be scrutinized for lucent lines around the implant, version of the humeral and glenoid components, relationship of the humeral head to the glenoid, bone loss, bony impingement from the glenoid or tuberosities, and humerus abnormalities that could interfere with long-stem implants. Comparison to recovery room films or any postoperative films can show obvious changes that could not otherwise be appreciated (Fig. 25-1).

Computed tomography (CT) scans should also be obtained to define the glenoid for possible bone loss that often cannot be appreciated on plain radiographs. Specific instructions for the radiologist should be requested so that the appropriate protocols can be used to subtract the artifact created by metal implants. Also, if the patient's arm cannot be held in neutral rotation during the CT scan, simultaneous images of the humerus epicondyles should be taken for accurate assessment of humeral head version.

Close study of the CT scan during surgery often helps us reconstruct the glenoid with the appropriate version, because we cannot rely on the distorted anatomy during revision surgery.

Finally, a magnetic resonance imaging (MRI) scan should be obtained if there is a suspicion of the significant full-thickness rotator cuff tear. Some may argue that the MRI is not necessary because the best evaluation of the cuff is made intraoperatively, but getting a full preoperative evaluation can help minimize unexpected findings during these complex procedures. Sperling et al. reported their results of a modified MRI protocol for the diagnosis of rotator cuff tendon tears in shoulders with metal prostheses.[5] Twenty-one MRIs were performed with a limited pulse sequence for the evaluation of shoulders before their revision arthroplasties. Of the 11 rotator cuff tears found in the cohort, MRI correctly predicted 10 of them. Subscapularis tears were the most common type of tear found in eight patients. Ten patients did not have a tear at the time of their revision, and MRI predicted that eight of them had an intact cuff. MRI also predicted glenoid wear in eight of the nine hemiarthroplasties in the group.

Bone scans can be used to assess implant loosening, although we rarely obtain this study because the radiographs often show lucent lines around the glenoid implant, and isolated humeral loosening is rare.

Arthroscopy of a total shoulder prosthetic can in some cases give the surgeon a better understanding of the cause of failure or pain. This can be related to glenoid component loosening or failure. Arthroscopy can define biceps tendon damage, rotator cuff damage, and subacromial impingement. In some cases the arthroscopic approach can be used to remove an all polyglenoid component, release or tenodese the long head of the biceps, repair a small to medium rotator cuff tear, perform a subacromial decompression, or remove a symptomatic acromioclavicular joint.

MODES OF FAILURE

To elucidate the common causes of unsatisfactory shoulder arthroplasties, Hasan et al. analyzed 139 consecutive patients who presented to the shoulder service because they were not satisfied with their surgery.[2] Seventy-four percent of the shoulders were stiff, 35% were unstable, and 59% of the

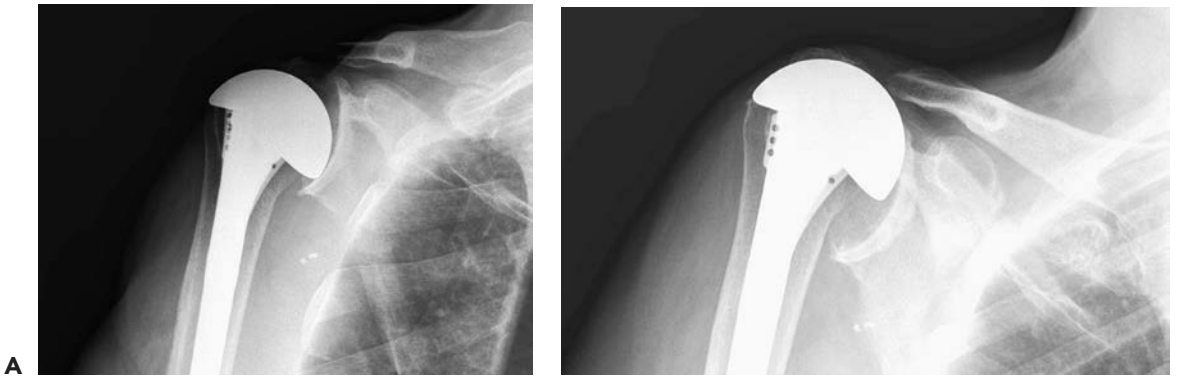


Figure 25-1 **(A)** This x-ray shows a hemiarthroplasty with a cuff tear arthropathy head that is centered in the glenoid. **(B)** One year postoperatively, the head has migrated superiorly in the glenoid. Clinically, the patient has superior escape of the humeral head when he tries to elevate his arm. Patients with superior escape require a reverse shoulder prosthesis to regain active shoulder elevation.

patients with a total shoulder had loose glenoid implants. Implants were malpositioned in 23% of the cohort. In the patients with hemiarthroplasty, 42% had glenoid erosion, and in patients who underwent hemiarthroplasty for a fracture, 43% had nonunion of the tuberosities. The average patient could only perform 2 out of 12 functions on the simple shoulder test. Based on these findings, the authors recommended greater attention to proper implant positioning, postoperative motion, and tuberosity fixation, all of which are directly impacted by the shoulder surgeon.

During the initial evaluation of a failed shoulder arthroplasty, it is often helpful to review the possible modes of failure according to their anatomic origin. The humeral component can cause pain or stiffness because it is loose. Excessive version of the humeral head can cause instability or limit motion or failure of the tuberosity fixation. Incorrect sizing of the humeral head, varus stem positioning, improper stem height, or a combination of all three can lead to an "overstuffed shoulder," resulting in altered shoulder mechanics that cause pain and stiffness. Glenoid loosening can cause pain. Improper glenoid version or excessive glenoid wear can cause instability. Bone loss in either the humerus or glenoid that was present preoperatively could have doomed the implants to failure, and more importantly, will affect the surgical options for revision.

The soft tissue integrity around the shoulder arthroplasty is arguably the most important consideration for the surgeon, even though the bone and implant are so often considered first. Bone defects can often be replaced with bone graft or modified implants with the use of cement, but rotator cuff defects cannot be easily repaired with grafts. Even if the tendon heals appropriately or a tendon graft material is used to deal with a tendon defect, function will not return because the muscle atrophy and fatty fibrous changes are often irreversible. If the rotator cuff cannot be salvaged, then the traditional unconstrained shoulder arthroplasty will not reliably restore shoulder function. A muscle transfer does not restore normal glenohumeral kinematics and a glenoid component should not be replaced because of its propensity to loosen. A constrained reverse total shoulder prosthesis could be used with or without a muscle transfer procedure. A muscle transfer may be indicated in a younger active person if the supraspinatus is not repairable or if there is an additional tear of the infraspinatus and teres minor. A reverse shoulder replacement requires a functioning deltoid and sufficient glenoid bone to allow for secure fixation of the metaglen and glenosphere.

INDICATIONS FOR REVISION

Just as the primary indication for the initial shoulder arthroplasty was pain relief, it should also be the primary reason for performing any revision procedure. Patients should be told to expect less improvement with strength and motion than with pain relief. Our experience has shown that patients who expect to have normal shoulder function after a revision procedure feel unsatisfied with their revision procedure, despite our own satisfaction with the patient's function and radiographs. Also, the surgeon should not have unreasonable expectations of their own surgical ability. Rotator cuff deficiency with muscle atrophy cannot be reversed, proximal bone loss cannot be simply replaced without a strategy to reattach the rotator cuff, and stiffness not caused by bone impingement will likely recur because of patient factors, not surgical technique.

The cause of the failed arthroplasty must be identified and considered surgically correctable (partially correctable) with a low potential of a major complication resulting in increased pain for less function (Fig. 25-2). For instance, a well-fixed humeral implant should not be removed to correct varus positioning to place another unconstrained prosthetic if the resulting proximal bone loss does not allow reattachment of the rotator cuff. Failed shoulder arthroplasties that do not have an identifiable anatomic cause for pain and dysfunction should not undergo revision. The most common indications for

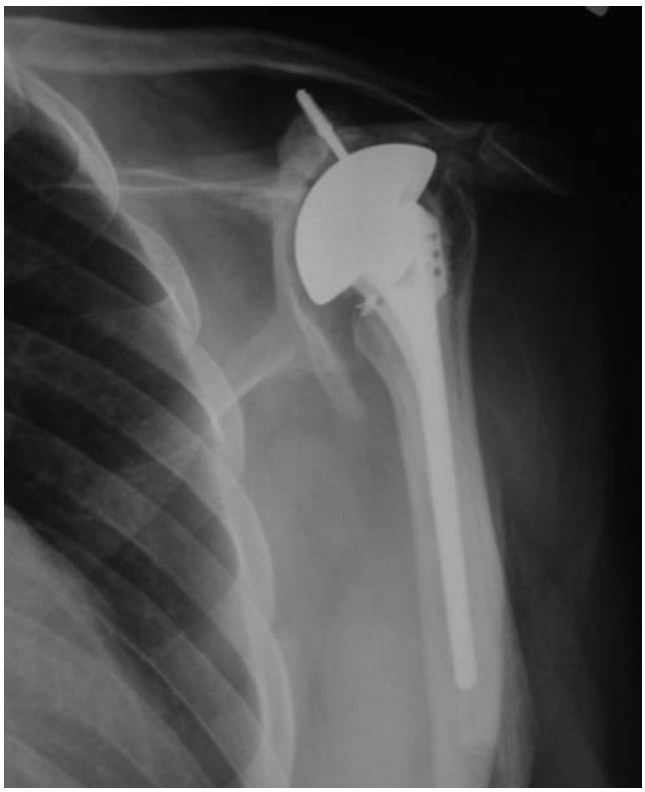


Figure 25-2 This hemiarthroplasty in a rheumatoid patient demonstrates severe medial migration. If glenoid resurfacing is attempted, restoring normal lateral offset should be avoided because the rotator cuff will be too contracted. In revision surgery, soft tissue deficiency may limit reconstructive options more than bone deficiency.

revision unconstrained arthroplasty include loosening of the implants, instability, an unresurfaced glenoid, and implant malpositioning. Revision arthroplasty with a muscle transfer or with a constrained implant is indicated if the patient has superior escape of the humeral head associated with a chronic rotator cuff tear.

MUSCLE TRANSFER OR CONSTRAINED ARTHROPLASTY?

The shoulder surgeon has three surgical treatment options to offer a patient who has a failed shoulder arthroplasty with an unrepairable rotator cuff. First, if the patient's only goal is pain relief and above-shoulder function is not important, then performing a hemiarthroplasty with a cuff tear arthropathy humeral head should provide satisfactory results. A glenoid should not be implanted in these patients because of the high risk of loosening. Second, if the patient would like to regain above-the-shoulder function as well as pain relief, then a muscle transfer will have to be performed to compensate for the rotator cuff deficiency, or a constrained implant can be used. Our experience with the latissimus dorsi transfer for external rotator deficiency has shown better results in younger male patients who do not have superior escape of the humeral head out of the coracoacromial arch during active elevation and the patient should have at least active shoulder elevation to shoulder height. Therefore, in elderly patients, particularly frail women, we avoid muscle transfers in conjunction with revision arthroplasty cases. Third, a constrained reverse total shoulder prosthesis has the advantage of early recovery of above-shoulder motion, compared to muscle transfers. The disadvantages of the constrained prosthesis are the 10% to 20% risk of dislocation, and the inability to externally rotate the arm with elevation in patients without any infraspinatus or teres minor function.

COMMON PROBLEMS TO ANTICIPATE

When there is a possibility that the humeral stem needs to be removed, the surgeon must pay close attention to the design of the proximal portion of the stem. If there is porous coating, or similar surfacing designed to facilitate bone ingrowth, the surgeon should anticipate a difficult removal of the stem, particularly when it is cemented. Often the tuberosities are thin and prone to fracture with excessive torque or aggressive application of the osteotome. A longitudinal osteotomy of the shaft may be necessary, so the surgeon should have a cerclage cable system ready for repair as well as long-stem implants. Also, damage to the tuberosities could preclude reattachment of the rotator cuff, so bone graft and a constrained prosthesis should be available. It is also wise to have a full set of power and hand cement extraction tools. In addition, the surgeon should have a head light and fluoroscopy because they can be helpful to visualize the mid- and distal aspect of the humerus and medullary canal.

Most failed shoulder arthroplasty cases have some form of stiffness, and this may be secondary to bony impingement or soft tissue contracture. If a malpositioned tuberosity or glenoid bone block is identified on the CT scan or at the time of surgery, then an osteotomy will be required if there exists loss of passive range of motion after a complete soft tissue release and if the prosthetic cannot be adapted to compensate for the tuberosity malposition. In all cases the surgeon should expect to perform complete circumferential soft tissue releases and excision of all capsular, subacromial, and subdeltoid scar. The subscapularis should be taken off the lesser tuberosity, except when there is a malunion, which requires an osteotomy or a nonunion of the lesser tuberosity, in which case the subscapularis tendon should be left attached to the mobilized lesser tuberosity fragment. The maximum subscapularis tendon length should be maintained in all revision cases. We do not recommend subscapularis Z-lengthening to improve external rotation because the tendon quality is often insufficient to allow for a secure repair. The incidence of subscapularis insufficiency increases with this technique due to tearing of the tendon and alteration of the moment arm. Separation of the anterior capsule from the tendon should be done at the midsubstance of the tendon or at the glenoid side to avoid thinning of the tendon. Inferior capsular excision will also be necessary, so the surgeon should have the appropriate retractors to isolate and protect the axillary nerve during this portion of the case. Another cause of the severely stiff shoulder is a captured cuff, where the subacromial and subdeltoid scar captures the rotator cuff so that it cannot glide independently of the deltoid and acromion. This subdeltoid subacromial scar can also inhibit dislocation of the prosthesis if it is not excised first.

Careful assessment should be made preoperatively for superior migration and superior escape of the proximal humerus. This abnormality will often cause distortion of the anatomy during the exposure of the case because the insertion of the pectoralis, deltoid, and rotator cuff muscles are more proximal in relation to the coracoid. The constrained prosthesis should also be available if the rotator cuff is deficient.

Preoperative assessment with CT scans should allow the surgeon to determine whether a glenoid implant can be used at the time of the revision. If the bone loss is substantial secondary to longstanding glenoid implant loosening, then the surgeon should have a femoral head allograft available, as well as standard and cannulated screw fixation systems. Medialization of the humerus can often be found with glenoid bone erosion in a hemiarthroplasty. When this is severe, if the surgeon decides

to resurface the glenoid, he or she should avoid completely restoring the normal lateral offset of the humerus, as this will overstuff the joint and lead to certain postoperative stiffness.

Instability is a difficult complication to treat in isolation, and it is even more difficult to treat in the face of glenoid bone loss. The tendency for the inexperienced surgeon is to focus on the bone defects that are obvious on the radiographs. What is often not appreciated is the soft tissue deficiency or redundancy in the direction of the dislocation, which can persist even after the bone loss is restored. The surgeon should anticipate performing capsular plication if posterior instability persists, and this can be done with arthroscopic suture anchors inserted through a posterior portal or with three or four purse-string sutures placed into the posterior capsule from the anterior wound. It is useful to have a mechanical arm holder and a laminar spreader to hold the head away from the glenoid and to expose the posterior capsule. This exposure is often very good due to the redundancy of the posterior capsule.

REVISION EQUIPMENT

Most of the retractors used for primary shoulder arthroplasty are used during the revision cases, with a few exceptions. For removal of the humeral stem, we always order the equipment specific to the stem design. This equipment usually attaches to the stems better than the universal extraction equipment. Thin osteotomes are used to loosen the proximal portion of the stem, and we have saws and a cable system ready in case a longitudinal osteotomy is necessary. Long-stem implants should always be special ordered because they may not come in the standard total shoulder setup, depending on the manufacturer. An assortment of bone tamps may also be used to tap the stem out of the humerus, as well as a set of revision instruments, including ring curettes with long handles, long thin osteotomes, and end-cutting cement removal drills. Anspach or Midas Rex power tools, a headlight, and fluoroscopy equipment should also be available.

Visualization of the entire glenoid, including portions of the body of the scapula, is necessary for bone-grafting cases, and these cases require extensive capsular releases to achieve the proper visualization of the glenoid. We use Fukuda-type retractors with an assortment of designs. The large-diameter Fukuda-type retractors are useful during reaming of the glenoid because they fit well with the circumference of the reamer. Other Fukudas with a thin opening are better for retraction during the exposure of the inferior glenoid. We also use a Gerber retractor to visualize the posterior capsule if a suture plication is necessary (Fig. 25-3).

INCISIONS

Many revision cases have more than one scar from previous surgeries, and they often cross each other at various angles. The incision for the deltopectoral approach has usually been made previously, so the risk of skin necrosis is small when this is the only incision to be used. If the procedure requires better visualization of the posterior glenoid or external rotators, we make a longitudinal incision from the posterior axillary crease to the spine of the scapula. When dual incisions are considered, the patient is positioned in the superior beach-chair position, taking care to get the shoulder far lateral so that the medial border of the scapula is exposed within the surgical field. This is facilitated by using a bean bag, a large bump behind the thorax, or an arthroscopy positioner. When the posterior incision is made, the operating room table is elevated and laterally tilted to the opposite side, and the surgeon sits onto a stool. When incisions are made parallel to one another, we try to have at least 4 cm between the incisions, but when we have had closer or crossing incisions we have rarely had any problems with wound healing in the shoulder.

EXPOSURE

The key to a successful revision shoulder arthroplasty is obtaining generous exposure, with a wide exposure being a primary goal and necessary to avoid compromising the removal of scar and prior implants, repair of the soft tissues, and insertion of the new implant and bone grafts. A wide exposure will result in the least damage to the tissue, as excessive retraction is avoided. If the surgeon is struggling to visualize the posterior attachment of the rotator cuff on the humerus or the posterior glenoid bone defect, insertion of implants will undoubtedly be difficult if not impossible, and ultimately the surgery and result will be compromised. When the initial exposure is compromised, consider extending the incision, releasing and excising more scar tissue, or incising tendons in a way that facilitates repair at the end of the case.

The first step in the exposure for the deltopectoral approach is defining the interval between the deltoid and the pectoralis. The interval will often be scarred from previous surgery, which makes assessment of muscle fiber orientation impossible. The surgeon must find the coracoid proximally and use it to define the interval. We sometimes cut directly down to the conjoined tendon on the coracoid and begin splitting the muscle distal to that point. We also start distal to the prior incision and use virgin tissue planes to find the cephalic vein and the deltoid muscle. We keep the cephalic vein lateral with the deltoid because of the excessive lateral perforators that require ligation if the vein is taken medially. We then work our way to the center of the interval, paying close attention to perforating vessels

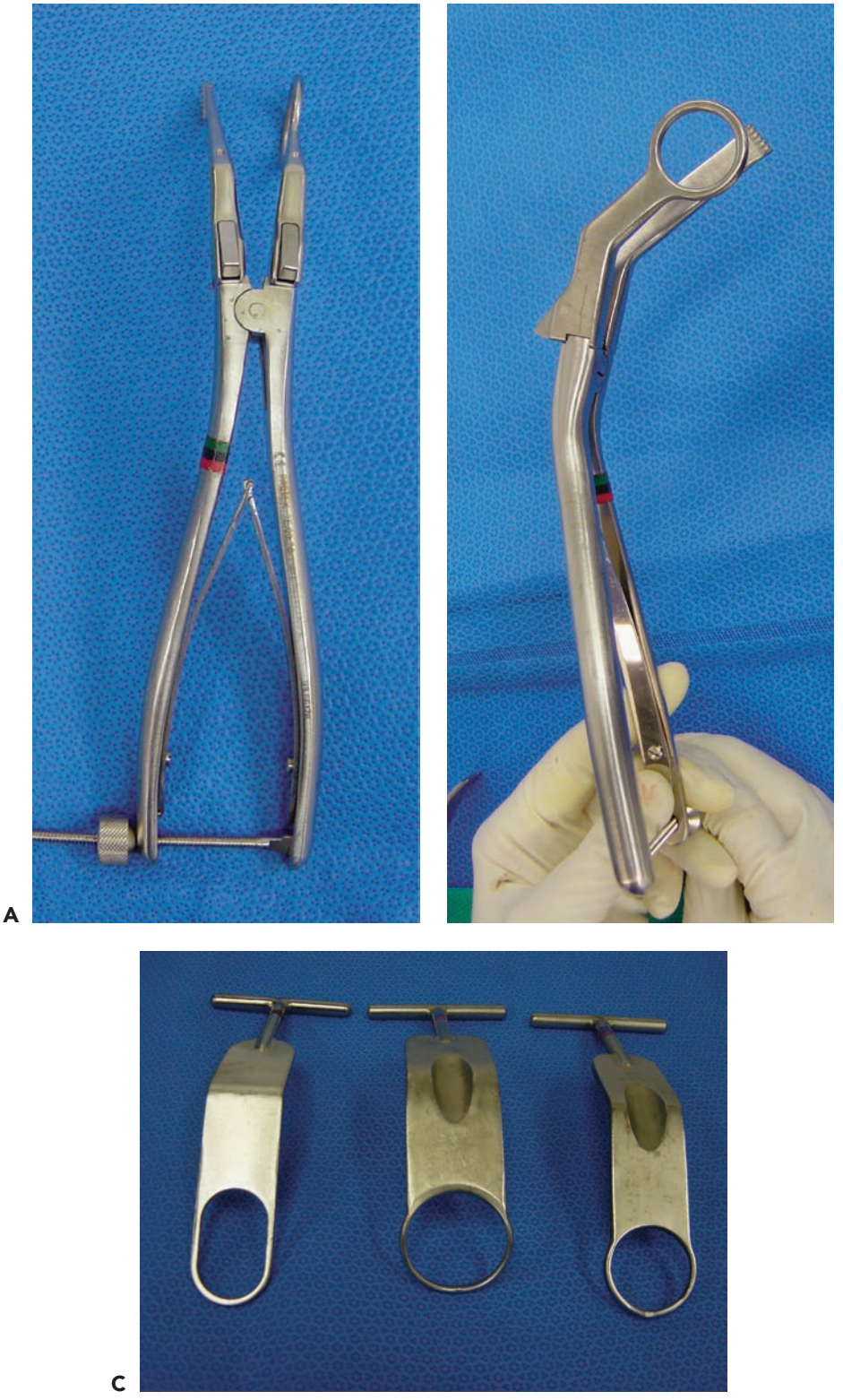


Figure 25-3 **(A–C)** The Gerber retractor is a modified laminar spreader that helps expose the posterior structures of the shoulder. A modified Fukuda retractor can help accommodate large glenoid reamers.

and the cephalic vein, which may be still patent. The most common error at this point of the procedure is not extending the interval proximal enough. The surgeon should be able to easily visualize 1 cm proximal to the coracoid process and should not hesitate to ligate the cephalic vein if it is at risk to rupture. The surgeon should incise the pectoralis tendon 3 to 4 cm distally, leaving a 5-mm cuff attached to the bone for tenodesis of the biceps tendon. Two or three figure-of-eight sutures through the biceps tendon can be placed at this point in the operation, and the tendon can be tenolysed proximally. The rest of the proximal tendon can serve as a landmark for the bicipital groove later in the procedure.

The next step in the exposure is the development of the tissue plane underneath the deltoid laterally. There is usually a perforating vein 2 cm proximal to the deltoid insertion, which the surgeon will tear during careless blunt dissection. Usually the interval is too scarred to allow blunt finger dissection, and sharp dissection is required to elevate the deltoid from the humerus. Do not injure the axillary nerve and accompanying vessels, which lie just distal to the flare of the tuberosities. Once proximal to the shaft, sharp dissection can be done without the fear of damaging vital structures. Scarred bursa should be excised from the rotator cuff, and if the bursa cannot be distinguished from the rotator cuff, the surgeon should incise at the tissue layer just deep to the deltoid muscle fibers up to the acromion. Once this plane is developed, and bent Hohman retractor can be placed over the rotator cuff to hold the deltoid away from the field. In cases where severe scarring limits visualization, a second incision should be made over the interval between the posterior and middle deltoid. This will allow for complete release of scar tissue around the posterior cuff and make possible rotator cuff repair much easier.

Medial exposure should be completed next. The surgeon should sharply incise lateral to the conjoined tendon layer and develop the plane superficial to the subscapularis muscle tendon. This usually requires both blunt and sharp dissection. The brachial plexus may need to be exposed to ensure safe dissection and retraction of the nerves. In some cases safe medial exposure may require osteotomy of the coracoid and distal dissection of the conjoined tendons. Regenerated vessels from the previously ligated anterior circumflex vessels will often course through the scar that needs to be cauterized and incised. Blunt dissection with a Cobb elevator wrapped in a gauze sponge sometimes works well to develop the correct plane and absorb any blood to facilitate visualization. The Kolbel retractor can then be placed under the conjoined tendons for easy visualization of the subscapularis tendon. The dissection should be completed distally to the humeral shaft until the latissimus dorsi tendon is seen. The axillary nerve should be palpated and visualized at this point. It can be visualized if a long right angle retractor is placed inferior and medially.

The subscapularis should be taken off the lesser tuberosity so that the maximum length of the tendon can be preserved. In revision cases, the tendon may be extremely thick because of the excessive capsule scar, or it can be thinned secondary to multiple surgeries and poor healing. We sharply elevate the tendon starting at the biceps groove and keep the capsule and tendon together until we can assess what we have to work with. If the interval cannot be palpated because of scar tissue, we extend the subscapularis takedown proximally until the biceps tendon is seen, and then we incise through the interval scar toward the base of the coracoid. Inferiorly, we incise the tendon approximately three-fourths of its width. At this point, we use a Cobb elevator to sweep away the subscapularis muscle from the capsule beneath. If this cannot be done by blunt dissection, then this interval is created by sharp dissection with a scalpel. The inferior capsule is bluntly cleared with a Cobb and sponge so that it is easily visualized and separated from the axillary nerve. After a blunt retractor is placed between the capsule and axillary nerve, the capsule is incised from its humeral attachment starting proximally and continuing distally to the latissimus dorsi tendon. The capsule is then incised deep around the medial side of the humerus as far as the surgeon can see directly, so that the nerve is not cut by a blind pass of the knife. At this point, the capsule and tendon may still be intimately attached medially, and these two structures are separated sharply with a scalpel. The capsule should be separated from the tendon and released from the glenoid to restore tendon excursion. By this method the entire anterior–inferior capsule is excised. Final release of the subscapularis tendon from the undersurface of the coracoid should completely free the tendon and muscle circumferentially.

At this point, the humeral head can be dislocated. A Hohman retractor is placed between the humeral head and glenoid and is levered inferior to the coracoid to avoid fracture of the process. The Hohman acts as a "shoehorn" to bring the humeral head out of the wound. External rotation and posterior force applied to the head should dislocate the head. If the dislocation cannot be accomplished because the tissue is too tight, additional releases should be performed in the following order: (1) release more inferior and posterior capsule and scar, and (2) then release a small portion of the supraspinatus tendon, leaving room for suture repair at the end of the case. In cases where there is a malunion of the greater tuberosity, adequate exposure of the proximal humerus may require osteotomy of the greater tuberosity. If these releases still do not allow for an atraumatic dislocation of the head, then separation of the modular head can be done in situ. The surgeon holds the proximal humerus laterally with a bone hook and separates the head from the stem with an osteotome or head removal instrument provided by the manufacturer. After the head is removed he or she sharply excises all of the scar

tissue from the posterior and superior aspects of the joint including the glenoid fossa circumferentially, leaving only the posterior and superior cuff intact.

HUMERUS REVISION TECHNIQUES

Maintaining the rotator cuff attachments to the proximal humerus and keeping the tuberosities in continuity with the shaft are the most important goals of revision humeral arthroplasty. If that anatomic relationship cannot be maintained, then an unconstrained arthroplasty will likely result in poor function if the tuberosities cannot be securely fixed in a near anatomic position or do not heal. Care should be taken during the extraction of well-fixed humeral components to avoid levering with osteotomes, as the proximal bone can be quite thin and fragile in the revision setting. Bone overgrowth and scar should be carefully removed from the lateral fin of the prosthesis, as this often impedes easy extraction. Proximal porous coating of the stem stimulates an excellent metal–bone bond, and in our experience, precludes extraction without an osteotomy of the shaft in at least 50% of the cases.

Once the decision is made to perform an osteotomy of the shaft, the incision should be extended distally, and should extend distal to the deltoid insertion and be in the interval between the deltoid and biceps muscles (Fig. 25-4). The shaft should be exposed in line with the biceps groove and the shaft cut longitudinally lateral to the biceps groove to the tip of the prosthetic. We use a power saw to cut through the bone and cement mantle if there is one. A half-inch osteotome is then placed in several locations within the osteotomy site and the sharp edge of the osteotome is placed at the prosthetic cement interval and gently tapped with a mallet to break this bond. When this is completed, the osteotome is placed in the osteotomy and given a quarter turn to wedge open the osteotomy site and break any further bond between the stem and cement. Once the stem is loosened, it can be pulled out with the appropriate extraction device. This technique does not work well for porous-coated stems with bone ingrowth or with cement interdigitation into the porous coating. In these cases thin osteotomes must be used to either cut an interval between the stem and cement through a proximal intramedullary access or through a formal bicortical window that is removed to allow access to the entire anterior face of the stem.

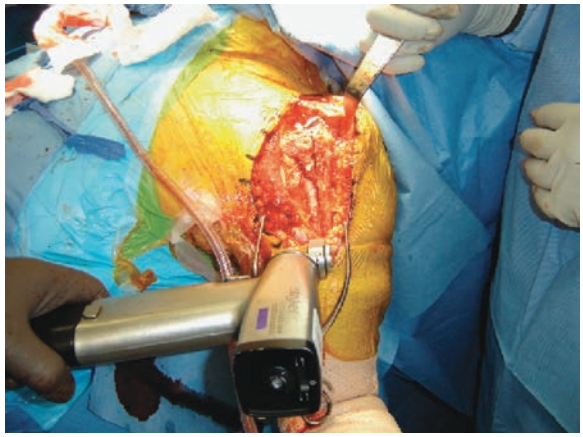
Cement removal can be taken out with power burrs for proximal cement seen within the medullar canal and long thin osteotomes and long power end-cutting drills bits designed for cement removal. In cases where there is no indication of infection, all of the cement does not need to be removed and another stem can be inserted into the previous cement mantel. Leaving the cement in place in noninfected cases may be the best option to avoid multiple fractures or perforations of the thin humeral cortex. In these cases, all loose cement needs to be removed and the inner portions of the cement mantel need to be removed to allow for a stem of sufficient diameter. Distal cement needs to be removed to allow for full seating of the humeral component. Use of fluoroscopy is often helpful to avoid penetration of the cortex, and having the proper drills and equipment is essential. When the humeral shaft is cut in only one cortical surface and then cerclage wired, a long-stem component is not required. If the shaft is unstable and there is a bicortical window or any transverse component to the shaft fracture, then the stem should extend distally two canal diameters beyond the extent of the distalmost fracture line; in most cases, this will mean using a long-stem cemented prosthetic. In patients with short stature, the standard long stems may be too long and the surgeon will need to be prepared to cut the stem in the operating room. This can be done with a diamond high-speed cutting tool (Fig. 25-5).

Cerclage cables can be passed around the shaft to reconstitute the canal, and then the canal can be prepared for the appropriate stem. We prefer to use the 1.6-mm Dall Miles cables. We do not recommend press fitting a stem once the metaphysis has been disrupted. When cementing the stem, care should be taken to avoid the cement extruding from the osteotomy or fracture sites. Wide exposure helps achieve this objective.

GLENOID BONE LOSS

There are three different treatments for glenoid bone loss, including impaction grafting, glenoid sculpting, and bulk allograft. If the defect is contained by a rim of bone, then morselized bone graft can be impacted to fill the defect. There are no scientifically determined guidelines as to when a new glenoid component can be placed into a glenoid with significant bone loss. We use as a general guideline that a glenoid implant can usually be implanted over the graft so long as there is at least 50% of the back surface of the glenoid component in contact with native subchondral bone and 100% of the peripheral rim is intact. If the bone loss in the central part of the glenoid vault is too large, then the current pegs or keel component designs will not allow for stability and a glenoid component of this design should be avoided. In this case, the glenoid is bone grafted and left without an implant (Fig. 25-6). If the patient has pain after the bone graft incorporates (1 year or longer after the surgery), the patient could undergo another procedure for placement of a glenoid or conversion to a reverse shoulder arthroplasty if indicated.

Hill and Norris reported their results of treating 17 glenoid defects with bone grafting in primary total shoulder arthroplasty. All of the patients in the study had a history



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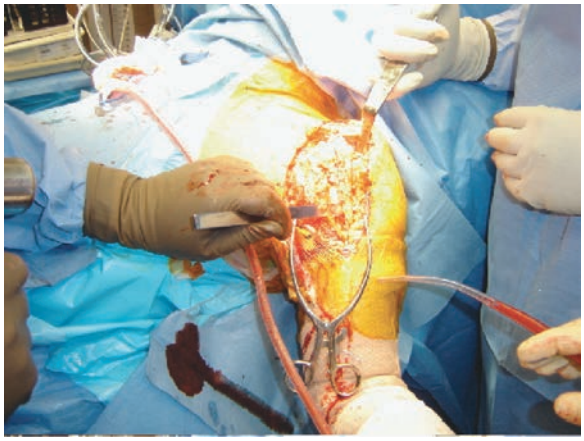

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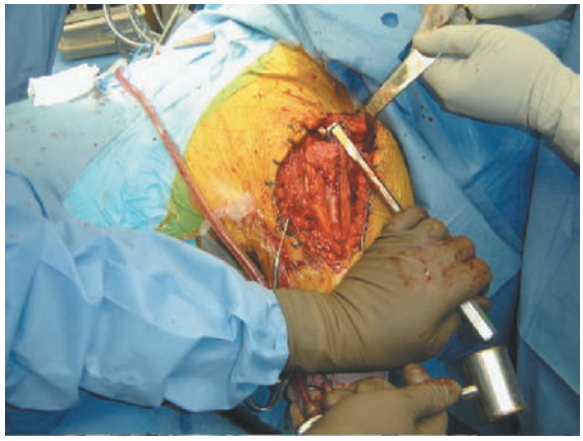

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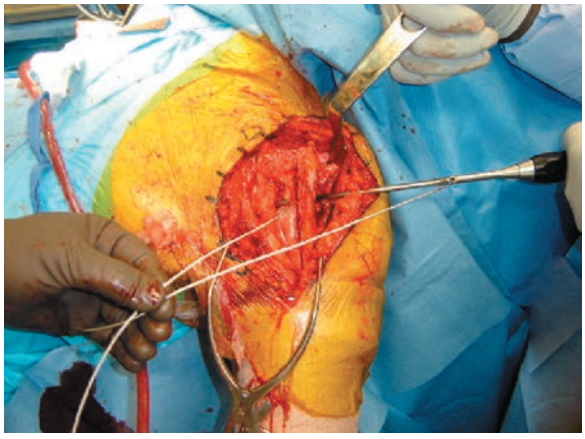

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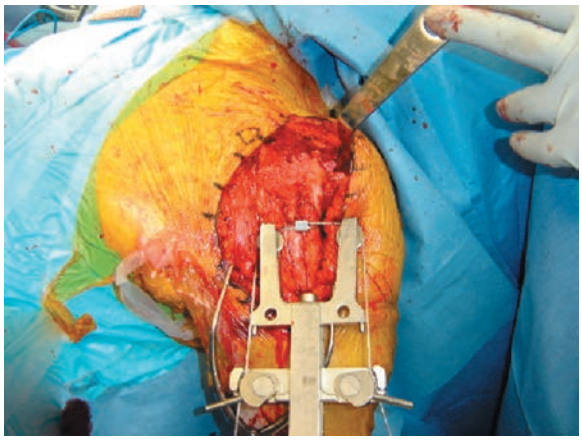

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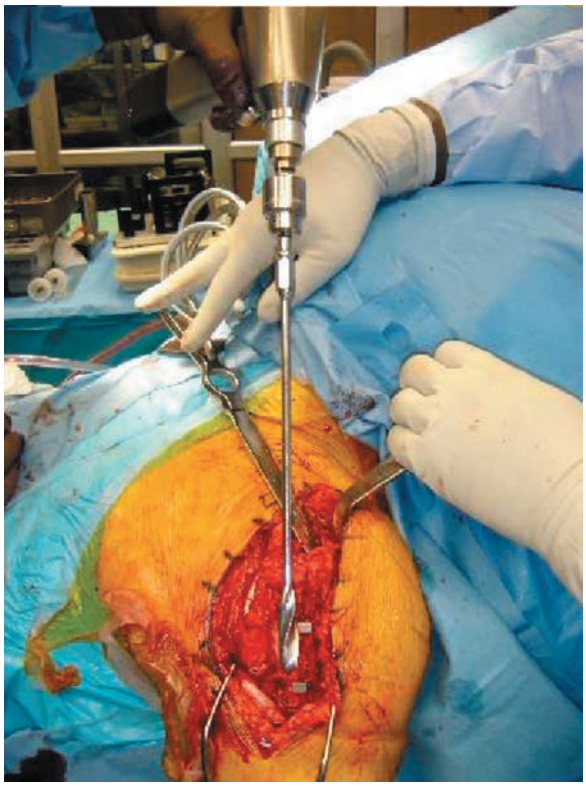

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Figure 25-4 **(A–C)** A unicortical osteotomy is made with a saw, starting in the biceps groove and extending distally. An osteotome is used to loosen the bond between the prosthesis and the cement before the stem is removed with a bone tamp or slap hammer. **(D–F)** Cables are placed around the shaft and then the canal is prepared for the revision stem using a reamer.

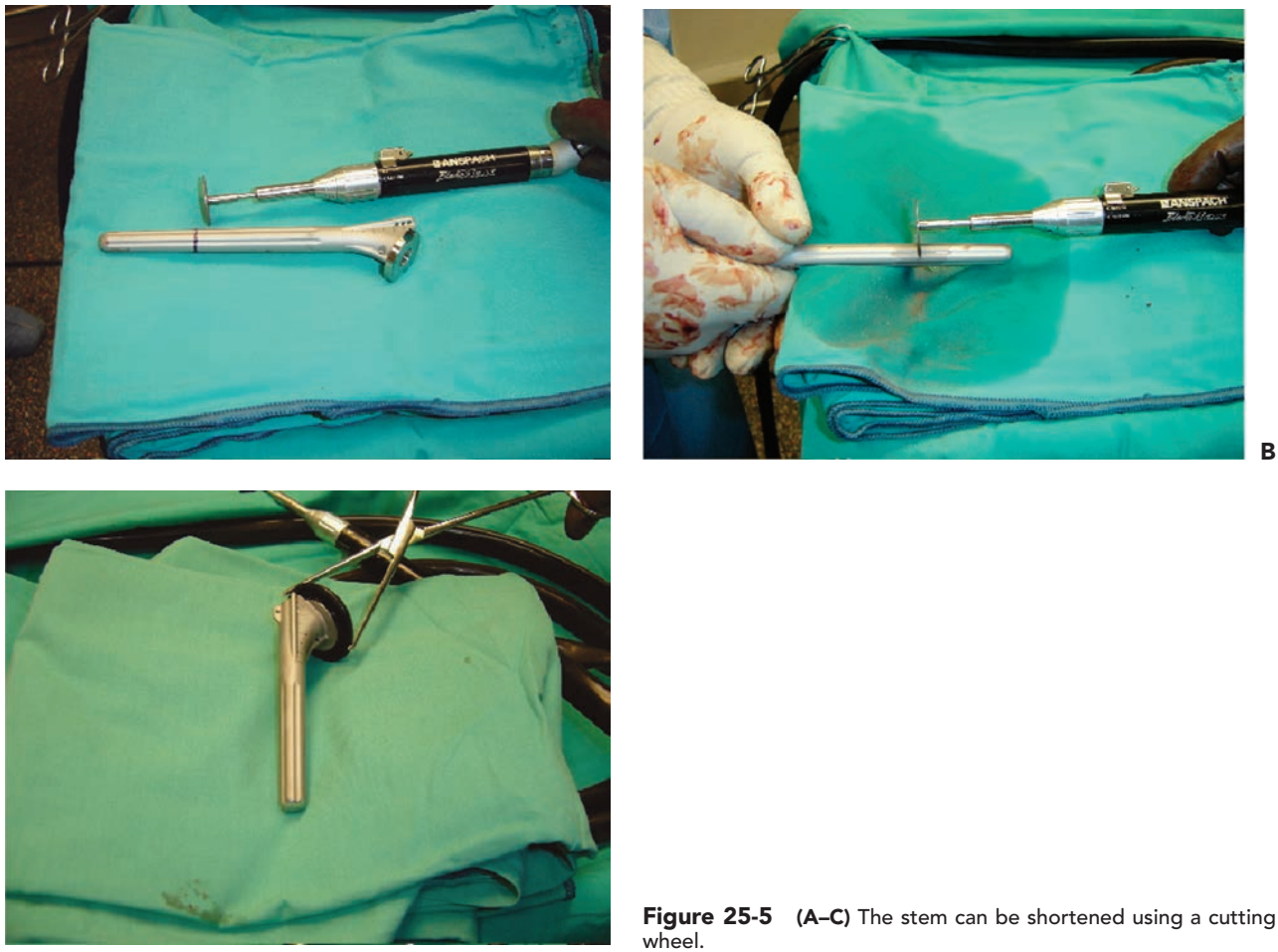


Figure 25-5 **(A–C)** The stem can be shortened using a cutting wheel.

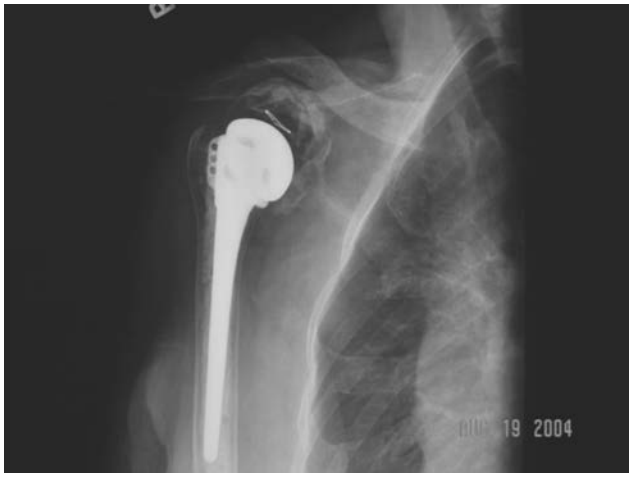
of instability. Twelve of the defects were in the posterior, and five were anterior. The bone graft was taken from the humeral head or from the iliac crest. The average depth of the glenoid defects was 12 mm (range 4 to 15 mm). The average correction of version was 33 degrees. While five total shoulders in the series failed because of persistent instability and rotator cuff tears, only one of the shoulders failed secondary to loosening of the bone graft.[3]

The editor's experience with glenoid bone loss during revision surgery was reviewed. We retrospectively reviewed 32 patients who underwent glenoid revision surgery after total shoulder arthroplasty (TSA). The etiologic factor for glenoid loosening was component malposition (20 patients) or glenohumeral instability (12 patients).

Results were reviewed at a mean follow-up of 4 years (range, 2 to 8 years). Glenoid reimplantation resulted in significant pain relief ($p < 0.0001$), improvement in American Shoulder and Elbow Surgeons (ASES) score ($p < 0.02$), and external rotation (24 to 44 degrees, $p < 0.004$). Revision to a hemiarthroplasty also resulted in significant pain relief ($p < 0.01$) and improvement in ASES score ($p < 0.05$).

For the treatment of glenoid loosening without glenohumeral instability, both reimplantation of a glenoid component and revision to a hemiarthroplasty improved function, satisfaction, and level of pain. Reimplantation of a new glenoid component offered greater improvements in pain ($p < 0.008$) and external rotation (increase of 20 degrees vs. 3 degrees, $p < 0.03$) compared to hemiarthroplasty. For patients with preoperative glenohumeral instability, revision surgery did not improve motion, function, or pain significantly. Risk factors associated with a poor outcome after revision arthroplasty included persistent glenohumeral instability, rotator cuff tears, and malunion of the greater tuberosity.

If a peripheral rim defect is less than 10 mm without significant medial bone loss and there is an intact subchondral bone, then you can normalize version by reaming the high side of the glenoid using a burr and reamer. For peripheral defects that are greater than 1 cm or when reaming the "high side" would result in too much bone loss to allow for placement of a glenoid or would result in humeral instability, then a bulk graft needs to be used. In revision cases this would require allograft materials (femoral head)



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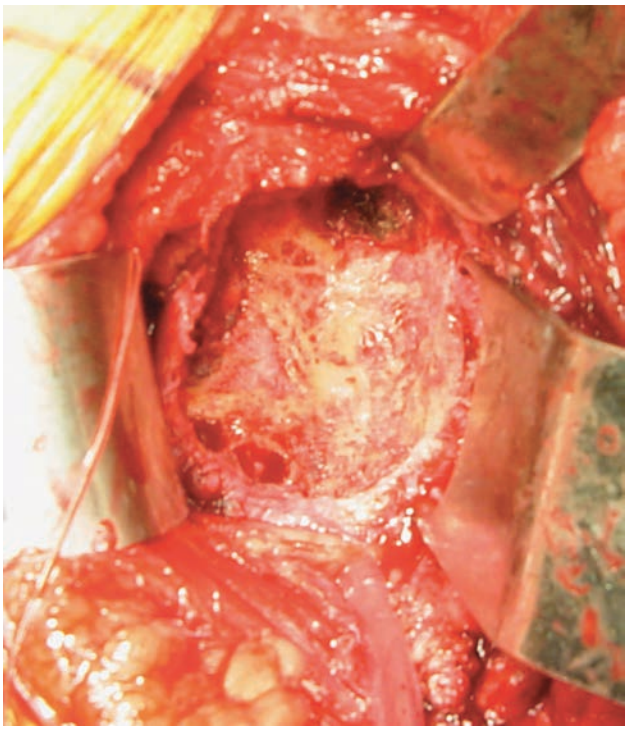

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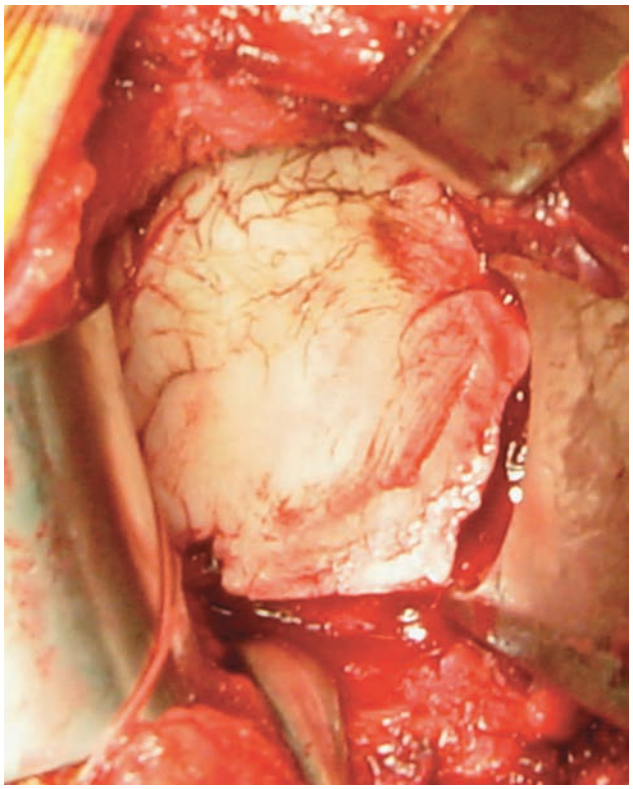

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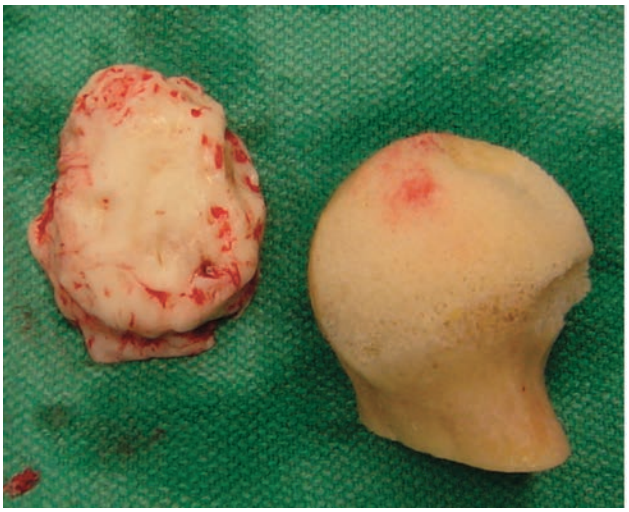

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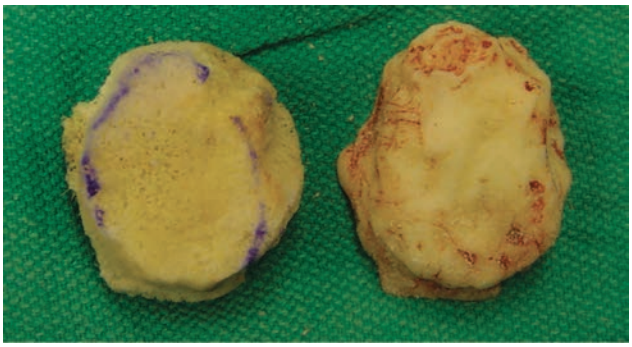
Figure 25-6 **(A)** X-ray demonstrates a loose keeled glenoid component surrounded by a large radiolucency, which is usually associated with a large cavitary glenoid defect. **(B)** The glenoid defect is large after the soft tissue is removed. **(C)** Cement is used to make a mold of the defect.

or in primary cases the patient's excised humeral head (if of sufficient size and quality) can be used (Fig. 25-7). In these cases the glenoid defect is made to resemble a step cut with the right angle cut being at the base of the defect. The graft is cut to fit the defect and secured in place with two cannulated small fragment screws inserted percutaneously through a posterior portal. The screws should be inserted below the rim of the graft and at approximately a 45-degree angle to the surface of the graft. The screws should engage the opposite glenoid cortex. The graft should be made larger than needed as it relates to the height of the glenoid surface. The graft and native glenoid is then reamed to be a smooth surface. The newly reamed surface can then be instrumented for either a pegged or keeled component. We make an attempt to use an Anchor peg all polyethylene component to minimize the cement needed for fixation. All



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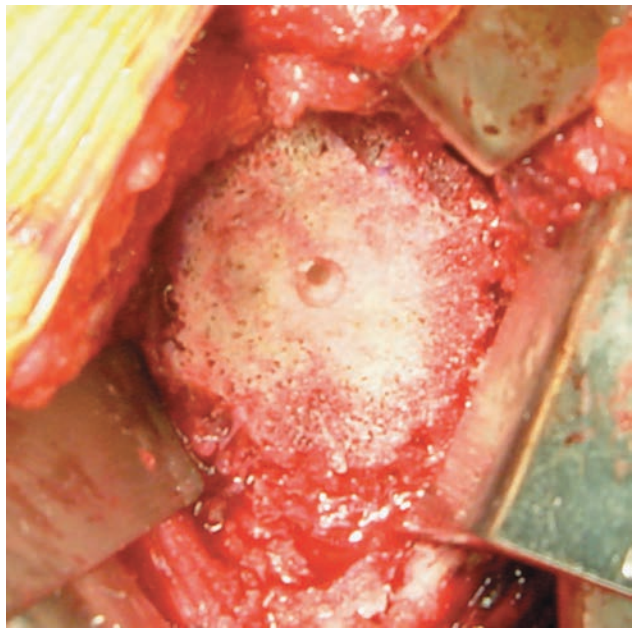

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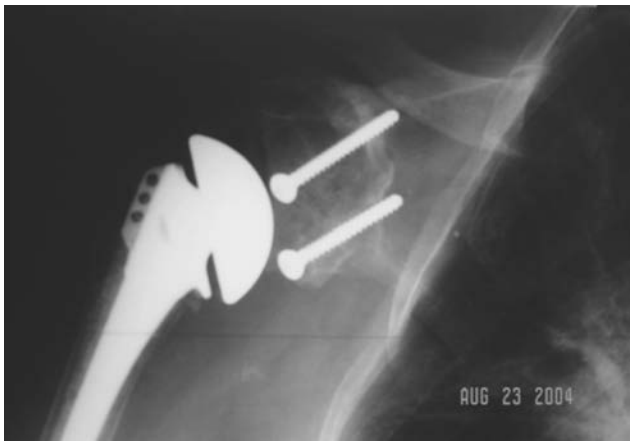

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Figure 25-6 (*continued*) **(D–E)** The cement mold is used as a guide to carve a femoral head bone graft that will match the glenoid defect. **(F)** After the femoral head is fixed into position with two screws, the graft is reamed to conform to the shape of the humeral head. **(G)** Postoperative x-ray shows position of the screws. The screw heads are anterior to the articular surface.

attempts should be made to avoid cement between the graft and the glenoid vault.

Most revision cases, however, have much larger bone defects secondary to persistent instability or long-standing glenoid implant loosening. These large defects often do not resemble the normal anatomy. The glenoid walls are expanded due to bone remodeling, and the defects are irregular in shape. There is variable loss of subchondral bone, the glenoid rim, and the glenoid walls. These three structures define the glenoid vault. We describe a classification of contained and uncontained glenoid bone loss by loss of these three structures. When the subchondral bone is missing, the defect is contained (intact rim and walls). When the rim and/or walls are deficient, then the defect is considered uncontained. In cases of uncontained glenoid bone loss, bulk allograft should be used. If fixed appropriately, the humeral component can articulate with the graft and the patient will have significant pain relief along with shoulder-level function or better if the cuff and tuberosities are intact. If the pain persists, a glenoid component may be implanted at a second procedure, once the bone has consolidated at least 6 months later.

Before implanting a bulk allograft, the extent of the defect should be determined by removal of all soft tissue and loose bone and cement from around the glenoid vault. Retractors should be monitored carefully, because they may lever on the thin walls and fracture the fragile glenoid. Once the defect is completely visualized, we fill the entire defect with bone cement and let it harden. The cement should be inserted just before it hardens to avoid cement extravasation outside the defect. The cement is molded in situ to be the size and shape of the intended bone graft. A screw is put in the middle of the cement to facilitate its removal after it hardens. The hardened cement can now be used as a model to shape the femoral head bone graft so that it will maximize the contact area between the graft and the defect. We fix the bulk allograft with two or three cannulated screws, making sure the screw heads do not protrude on the articular surface of the graft. Finally, a glenoid reamer is used to create a concave surface on the graft with the appropriate version.

TUBEROSITY MALUNION

Just as the integrity and function of the rotator cuff determines the outcome of shoulder arthroplasty, so does the position of the tuberosities in cases with a previous frac-

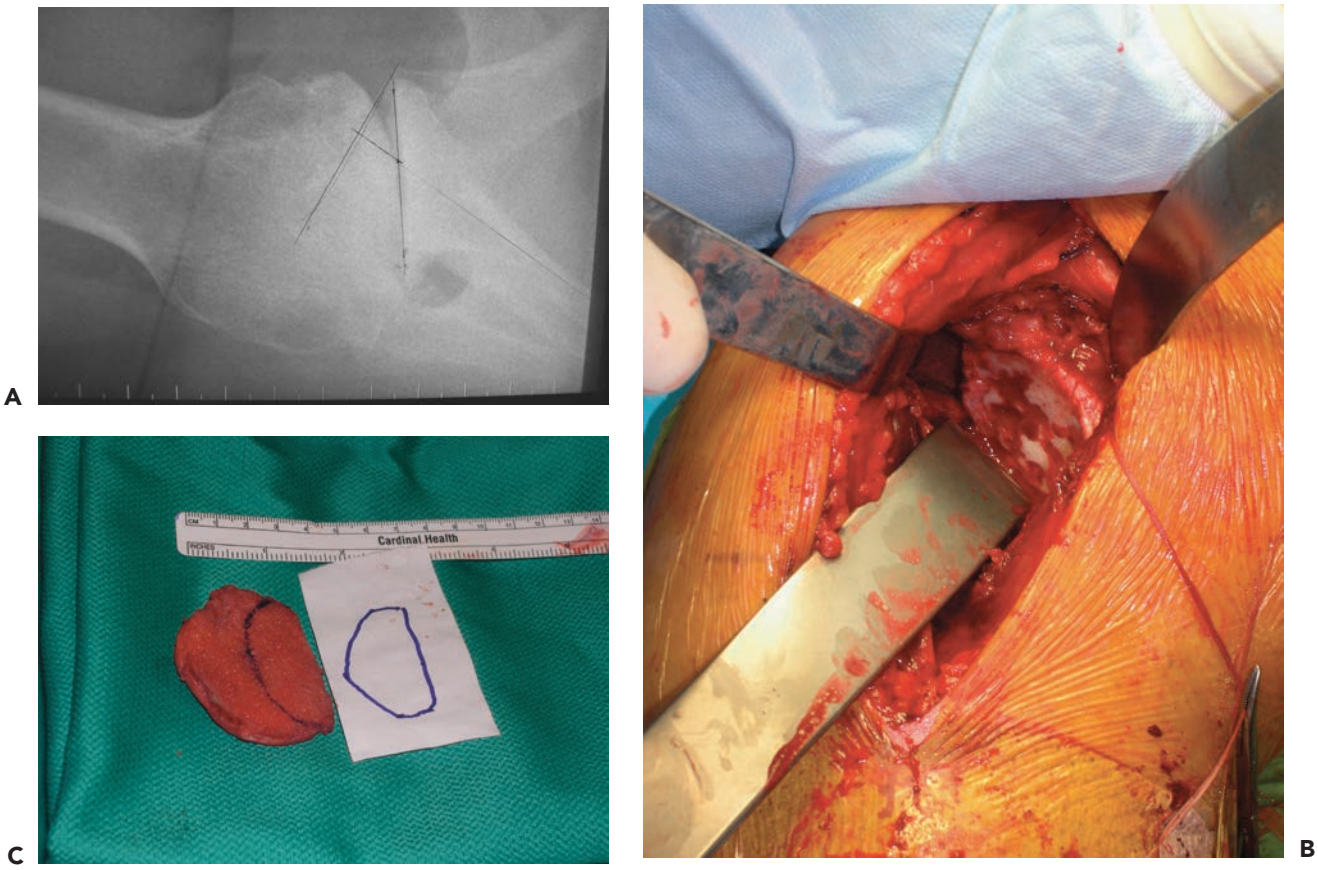
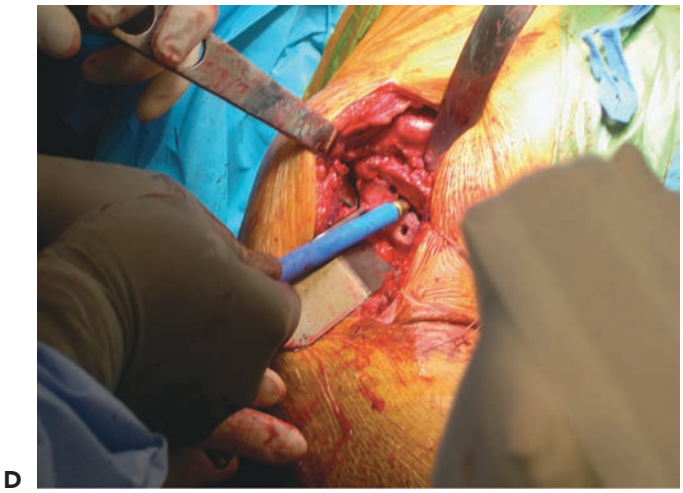


Figure 25-7 **(A)** Lateral x-ray shows significant posterior glenoid wear that requires bone grafting. **(B)** Appearance of the glenoid before preparation.

ture. A study by Boileau et al. evaluated the outcomes of their hemiarthroplasty in patients who had previous proximal humerus fractures.[1] Forty-nine cases had collapse of the humeral head or dislocation of the humeral head without displacement of the tuberosities that required an osteotomy, and 22 cases had malunions of the tuberosities or nonunion of the surgical neck, which required reattachment of the tuberosities to the proximal prosthesis. The adjusted Constant scores were 73% for the nonosteotomy group and 58.5% for the osteotomy group. The active elevation for the nonosteotomy group was 123 degrees, while the osteotomy group did not recover elevation beyond the horizontal (77 degrees). All 11 patients who were dissatisfied with the results of their hemiarthroplasty underwent osteotomy of their tuberosity malunions. The authors recommend avoiding an osteotomy unless it is absolutely necessary. Altering the version, offset, or size of the prosthesis may be enough to overcome the malposition of the tuberosities. Moving the humeral head to accommodate tuberosity malposition may require altering the stem diameter or length and thereby allowing greater flexibility in positioning the head within the confines of the tuberosities. A smaller stem will need to be cemented.

If the tuberosity malunion is too severe and external and internal rotation are limited or if there is abnormal contact of the tuberosities with the glenoid or coracoid or acromion despite altering the position of the humeral prosthesis, then an osteotomy of the tuberosity is indicated (Fig. 25-8). The osteotomy should be performed with a saw to avoid uncontrolled fractures through the proximal humerus. Correct placement of the osteotomy requires complete exposure of both the outer and inner surfaces of the cuff and tuberosity. The osteotomy should be oblique to the humeral shaft to maximize bone contact when it is repositioned onto the new stem and humeral shaft. After the osteotomy is completed, non-absorbable sutures should be placed through the tendon as well as through the bone. A complete posterior and superior capsular release will be required to mobilize the fragment to its new anterior and inferior position. It may be necessary to place the tuberosity in a better than before the osteotomy but not in an anatomic position. In some cases with longstanding posterior nonunion or malunion with



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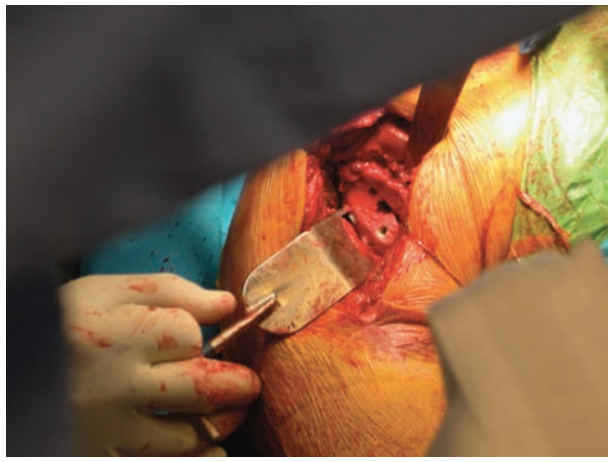

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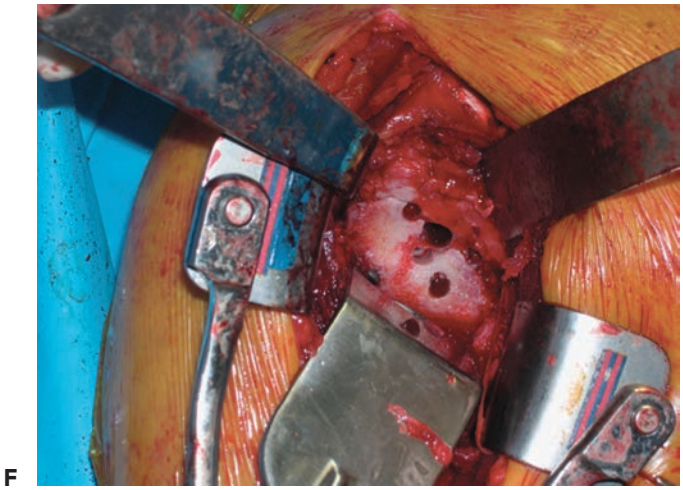

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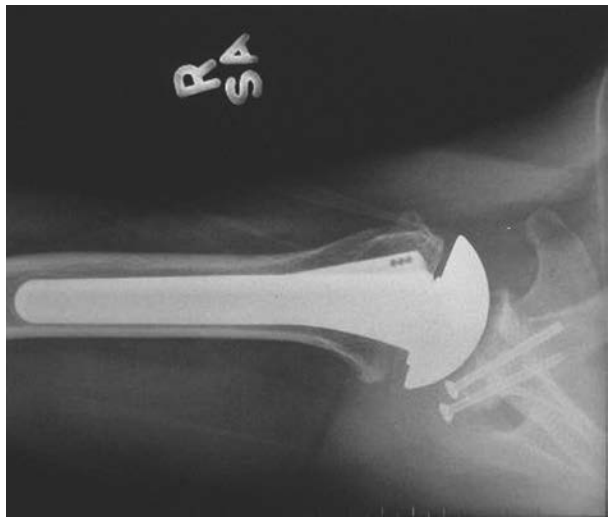

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Figure 25-7 (*continued*) **(C–F)** An outline of the glenoid defect is made on paper and then traced over the humeral head that was resected. After the bone graft is cut to the correct size, it is fixed to the glenoid with screws. Then the glenoid and bone graft are reamed and prepared in the usual manner for fixation of the glenoid component. **(G)** Postoperative lateral x-ray shows restoration of the normal glenoid version.

complete capsular excision, there is still contracture of the muscle such that placement of the tuberosity in its normal anatomic positions will lead to severe external rotation contracture. In these cases, we will place the tuberosity as close to its normal position while still allowing the wrist to come within 2 to 3 in. of the abdomen when the elbow is by the patient's trunk. The patients are always placed in a brace for 6 weeks after surgery. Most patients will regain sufficient internal rotation to reach their abdomen within pull off of the tuberosity. Fixation of the tuberosities also includes a circumferential suture that passed through the medial hole of the prosthesis for compression of the tuberosity. The sutures can be passed with a large sharp needle, or if the bone is thin after humeral canal preparation, drill holes can be placed for passage of the sutures. At least one suture should be passed distally to resist superior migration of the greater tuberosity, and at least two sutures should be placed to resist external rotation of the greater tuberosity. If the fins of the prosthesis are exposed, then the sutures should be passed through the fins. The sutures through the tendon are best for resisting tension, and the sutures through the tuberosity bone are best for compression. If after the osteotomy and repositioning of the tuberosity there is minimal bone contact, morselized bone graft should be used to bridge the gap. When the tuberosities and/or cuff are deficient and when after osteotomy there is a high likelihood for nonunion or poor cuff function, then use of the reverse shoulder arthroplasty would be the best option, particularly in patients over the age of 65 years.

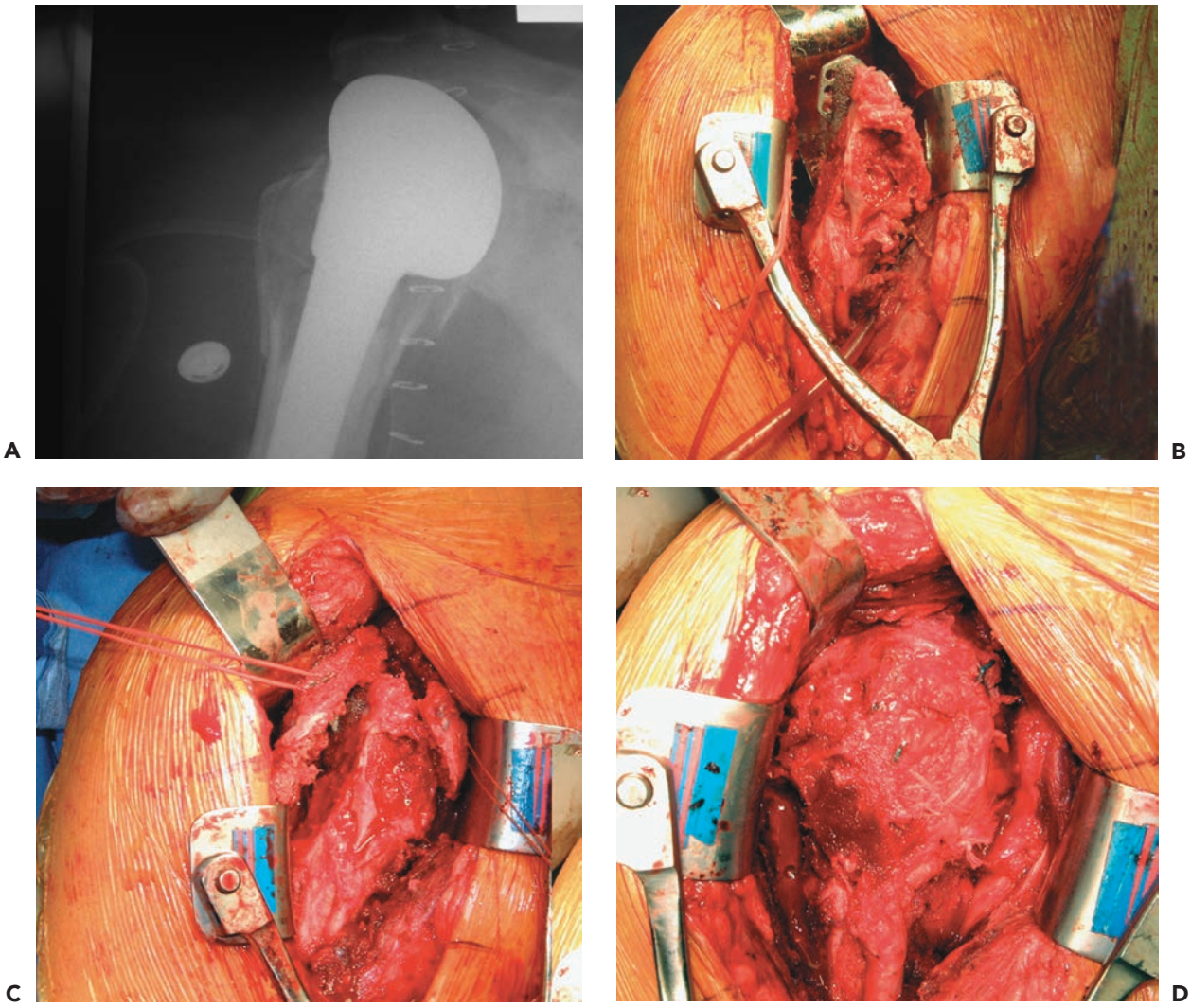


Figure 25-8 **(A)** The greater tuberosity healed 1 cm inferior to its anatomic position. **(B–D)** Intraoperatively, the tuberosity was inferior and posterior to its anatomic position. Release of adhesions was done along the superior and inferior borders of the tendon to allow full excursion with minimum tension. Nonabsorbable sutures were used to attach both tuberosities around the prosthesis.

SEVERE SOFT TISSUE DEFICIENCY AND INSTABILITY

In some patients with severe anterior soft tissue deficiency and anterior instability, a soft tissue reconstruction of the shoulder can be a good option when there is otherwise good bone, deltoid, and superior and posterior cuff function. This circumstance most often occurs with late recognition of a postoperative dehiscence of a subscapularis repair after prosthetic arthroplasty. In these cases, when pain and poor function or prosthetic instability indicate the need for surgery, a soft tissue reconstruction using a long strip of iliotibial band can be used to reconstruct the anterior and inferior capsular tissues. The sternocostal portion of the pectoralis muscle can be transferred either below or above the conjoined tendon to provide some dynamic stabilizer function[4] (Fig. 25-9). As with all revision surgery, balancing the soft tissues, by excision of all contracted tissues both anteriorly and posteriorly, is required to achieve a satisfactory clinical result.

SEVERE SOFT TISSUE AND BONE DEFICIENCY

In some cases after failed arthroplasty there is severe soft tissue loss of both the rotator cuff and deltoid as well as severe glenoid and/or humeral bone loss such that any

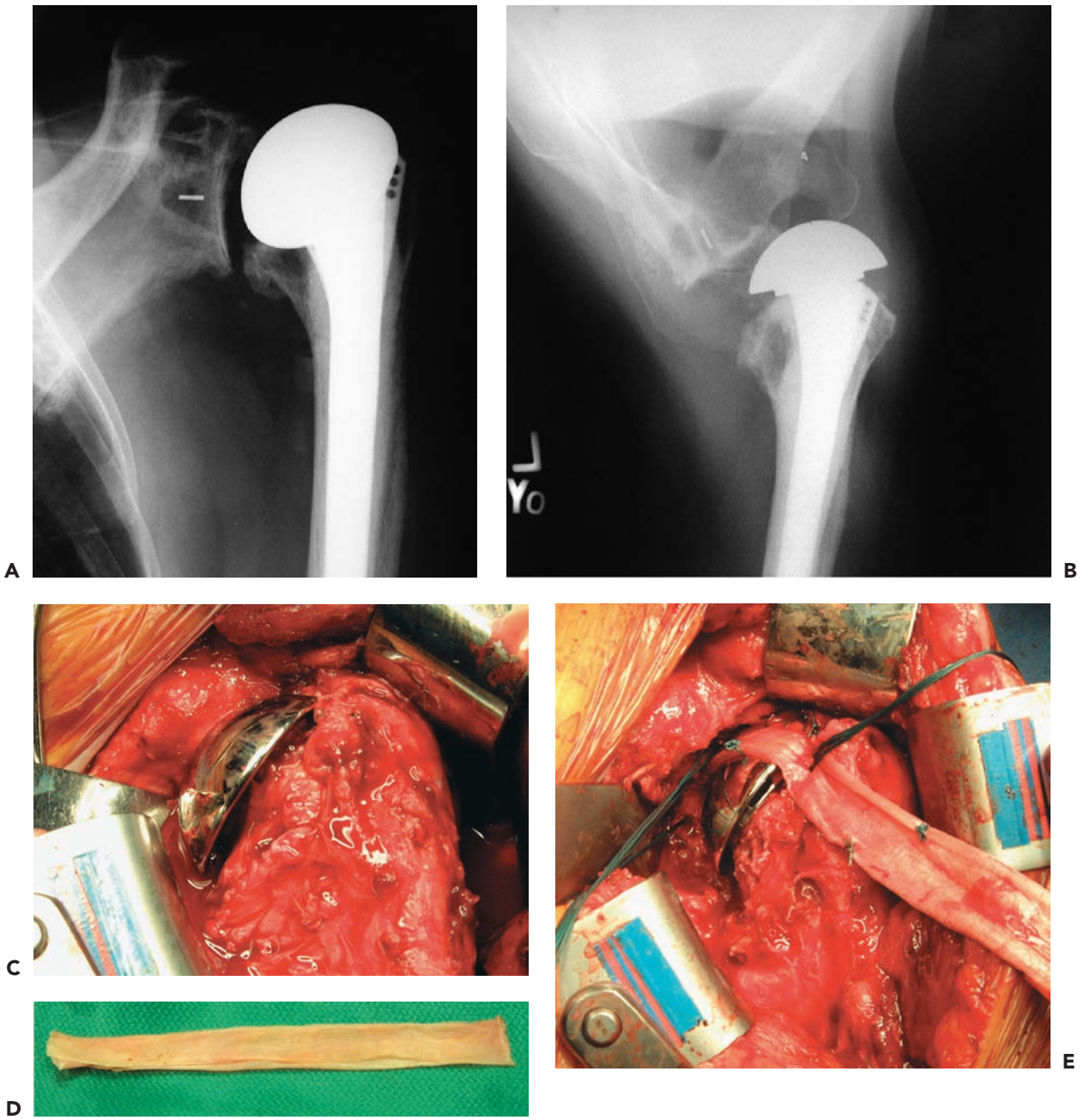
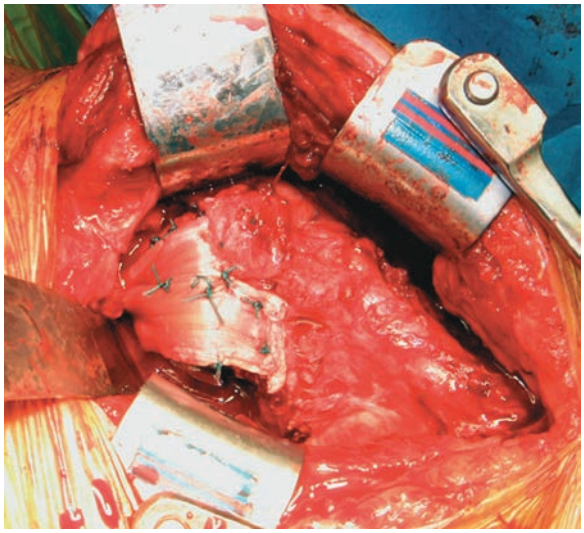


Figure 25-9 **(A,B)** X-rays demonstrate a total shoulder arthroplasty that is unstable anteriorly. **(C)** The subscapularis, a significant active restraint to anterior dislocation, was found detached from the humerus.

prosthetic arthroplasty will be of little value. In these cases either resection arthroplasty or glenohumeral fusion is required. Glenohumeral fusion can be a significant challenge due to both poor soft tissue coverage as well as bone loss. The senior author has converted 11 cases to arthrodesis for failed arthroplasty with severe soft tissue (cuff and deltoid) and bone loss. When there are intact tuberosities, near-normal glenoid bone, and absence of infection (failure of a hemiarthroplasty for arthritis or fracture), then a fusion with structural allograft, plating, and at least 3 months in a spica cast has been successful in relieving pain and getting shoulder-level function in all five cases treated to date. In patients with absence of the tuberosities and/or deficient glenoid bone, a vascularized free fibula autograft is required. In most cases a second or third bone-grafting procedure may be required to achieve



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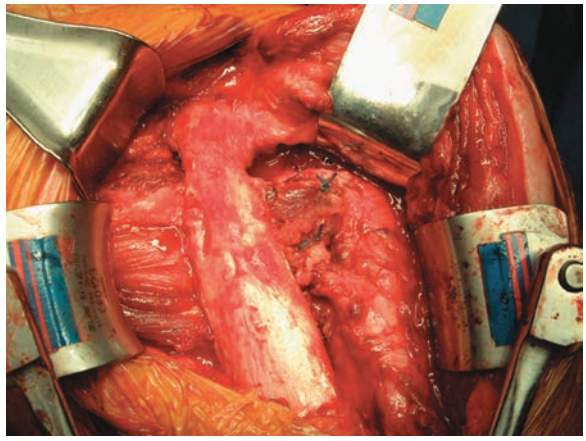

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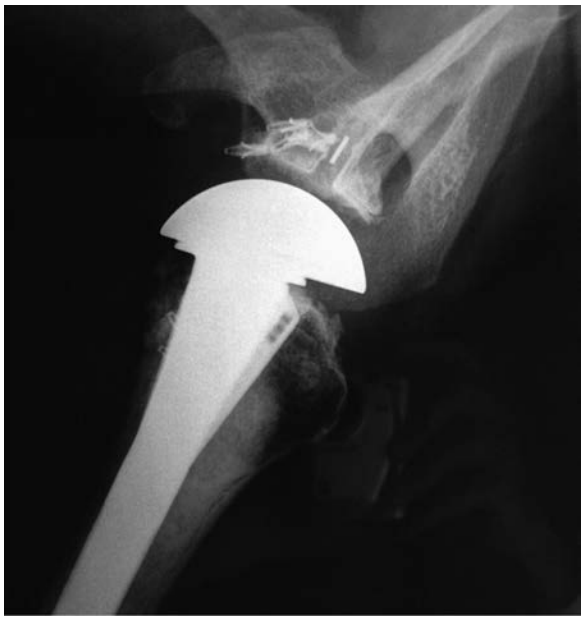

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Figure 25-9 (*continued*) **(D–G)** An Achilles tendon allograft was used to reconstruct the anterior capsule. It was sewn into the glenoid and humerus in a Z pattern from superior to inferior. Then the sternal head of the pectoralis major muscle was transferred to the humerus to substitute for the deficient subscapularis. **(H)** Postoperative x-ray demonstrates a stable construct.

union. Of the six cases treated to date, the senior author has had one persistent nonunion. All the other cases have healed with at least chest-to-shoulder-level function.

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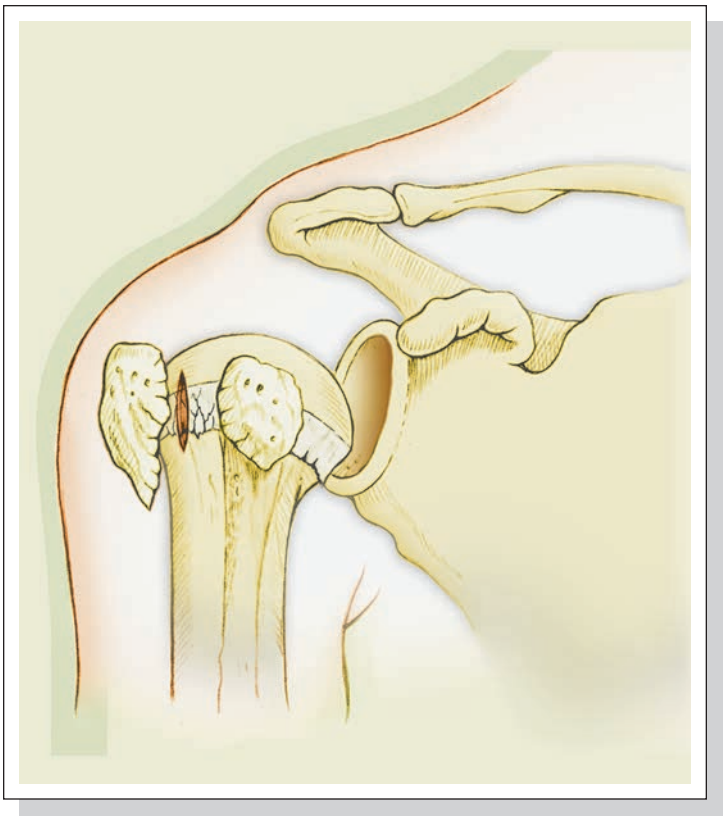
Fractures

V



26 Fractures of the Scapula: Diagnosis and Treatment

Thomas P. Goss *Brett D. Owens*

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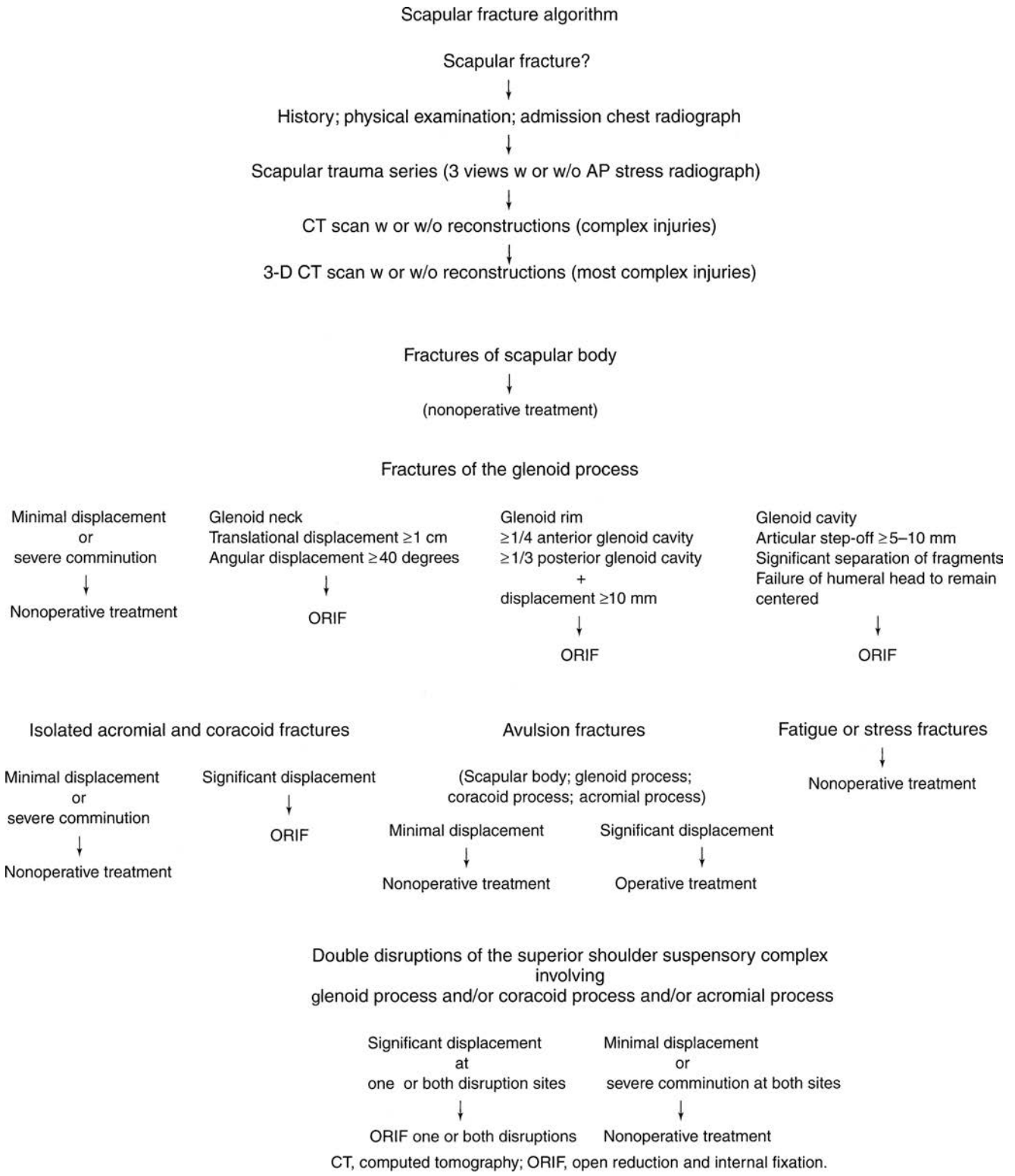
CLASSIFICATION AND INCIDENCE

General Considerations

Fractures of the scapula account for 1% of all fractures, 5% of shoulder fractures, and 3% of injuries to the shoulder girdle. There are two reasons for this relative infrequency: (a) the scapula lies over the posterior chest wall protected by the rib cage and thoracic cavity anteriorly and a thick layer of soft tissues posteriorly, and (b) the relative mobility of the scapula allows for considerable dissipation of traumatic forces. The vast majority of scapular fractures



(90+%) are insignificantly displaced, primarily because of the strong support provided by the surrounding soft tissues, making nonoperative management the treatment of choice for most scapular fractures. As a result, these injuries have received little attention in the literature. Scapular fractures involve a major articulation, however, and when significantly displaced are capable of causing considerable morbidity. Consequently, these injuries deserve more respect and recently have received more consideration in major texts,[11,23,28,32,129,142,143,149,158,169,175,183] general and review articles,[42,51,62,63,68,88,107,125,127,128,145,146,165,168,187,199,202,210] and papers dealing with specific issues.

Scapular fractures are usually the result of high-energy (usually direct but occasionally indirect) trauma. Consequently, they have an 80% to 95% incidence of associated osseous and soft tissue injuries (local and distant) that may be major, multiple, and even life-threatening. These individuals need to be carefully evaluated when they present in the emergency room, and appropriate supportive care must be rendered (see Scapular Fracture Algorithm). As a result, scapular fractures are often diagnosed late or definitive treatment is delayed. This may compromise the patient's final functional result. In addition, if associated injuries involve the shoulder complex, the individual's "scapular fracture" recovery may be compromised still further.

Most scapular fractures involve the body and spine (approximately 50%)[119] (Fig. 26-1). These injuries are often rather alarming radiographically (extensive comminution and displacement are frequently present; Fig. 26-2); however, there is very little enthusiasm in the literature for operative treatment,[72] since bone stock for fixation is at a premium and these injuries seem to heal quite nicely with nonoperative/symptomatic treatment[76]—a good to excellent functional result can be expected. The reason for this positive prognosis probably relates to the fact that (a) the scapulothoracic interval is cushioned by a thick layer of soft tissues, and (b) the mobility of the scapulothoracic articulation compensates for most residual deformities of the scapular body. The literature does mention a fracture of the scapular body with a lateral spike entering the glenohumeral joint as an indication (albeit extremely uncommon) for surgical management.[71] A similar recommendation was made in the case of an individual with a fracture of the scapular body and intrathoracic penetration of the fragments.[18] On rare occasions, a scapular body malunion may result in scapulothoracic pain and crepitus requiring surgical exposure of its ventral surface and removal of the responsible bony prominence(s).[121] Finally, nonunion of a scapular body fracture requiring surgical management has been described.[40,67,93,132]

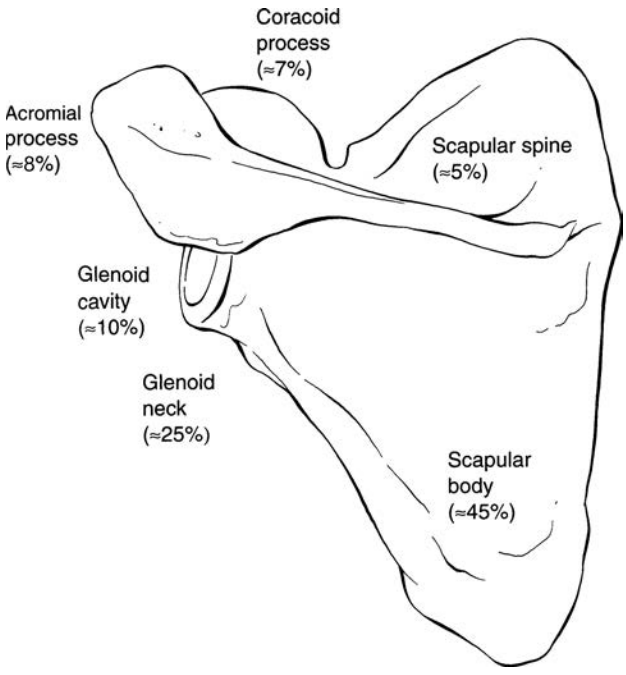


Figure 26-1 Incidence of scapular fractures according to region: the scapular body (≈45%), the scapular spine (≈5%), the glenoid neck (≈25%), the glenoid cavity (≈10%), the acromial process (≈8%), and the coracoid process (≈7%). (From McGahan JP, Rab GT, Dublin A. Fractures of the scapula. *J Trauma* 1980;20:880, with permission.)

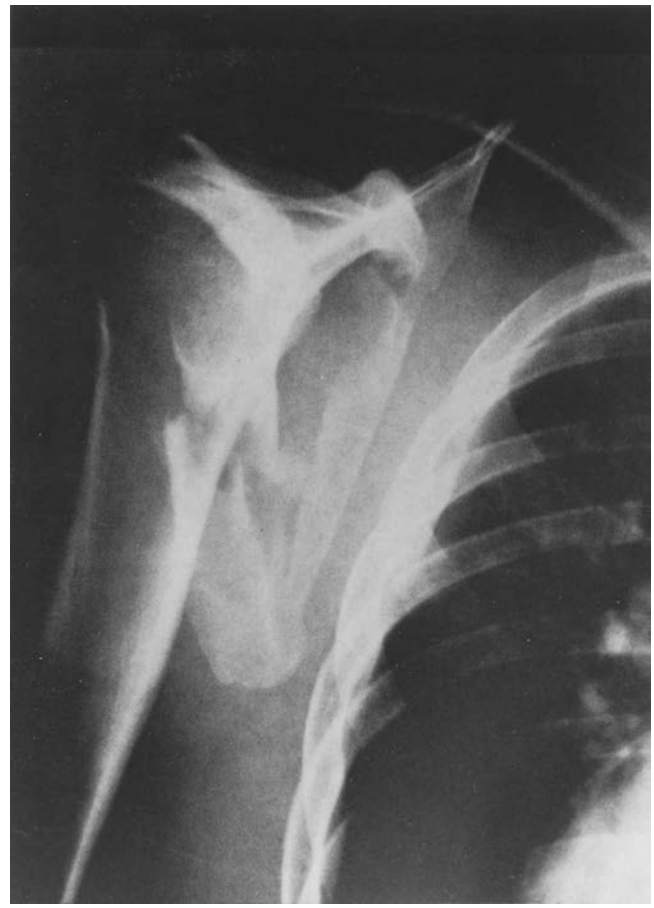


Figure 26-2 Anteroposterior radiograph showing a severely comminuted fracture of the scapular body. (From Neer CS II. Fractures. In: Neer CS II, ed. *Shoulder reconstruction*. Philadelphia; WB Saunders, 1990;412, with permission.)

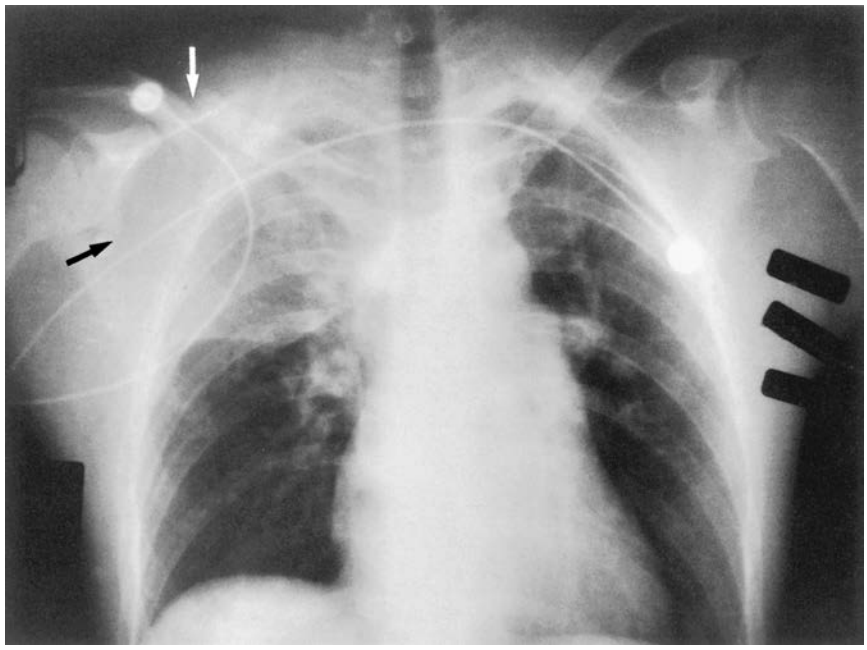


Figure 26-3 An admission anteroposterior chest radiograph of a patient who sustained multiple trauma. The fractured clavicle and scapula (*arrows*) were incidental findings.

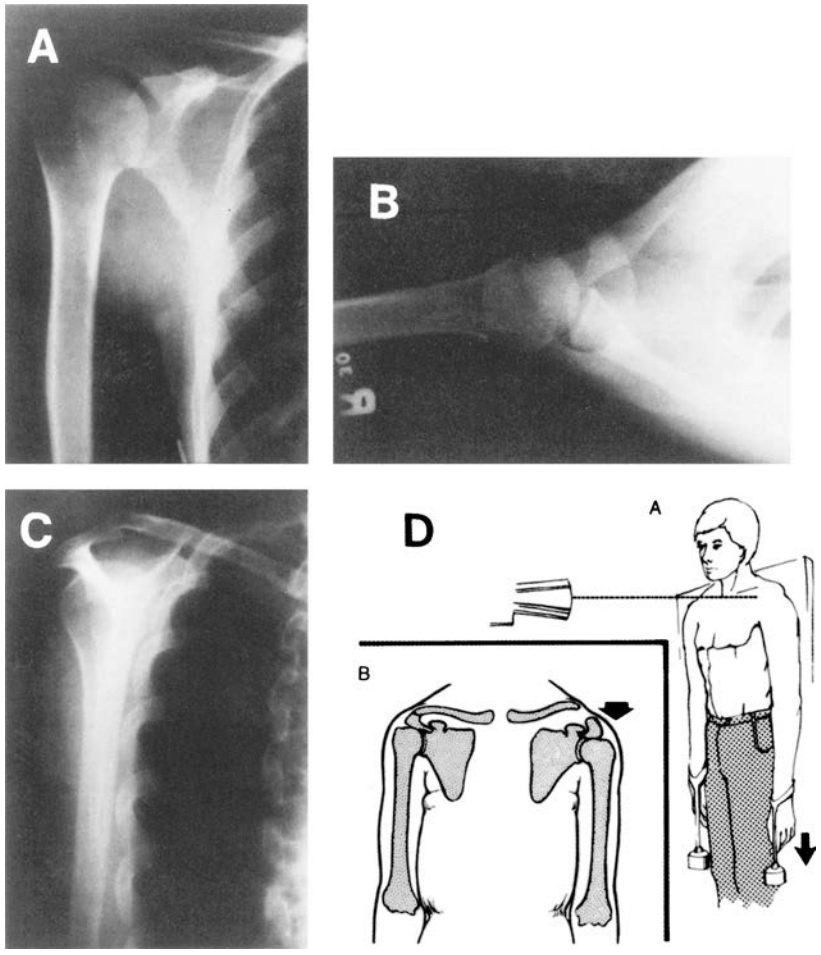


Figure 26-4 The "scapular trauma series": **(A)** a true anteroposterior (AP) projection of the scapula; **(B)** a true axillary projection of the glenohumeral joint and scapula; **(C)** a true lateral projection of the scapula; and **(D)** a weight-bearing AP projection of the shoulder complex designed to evaluate the integrity of the clavicular–scapular linkage (optional and dependent on the clinical situation). (D: From Rockwood CA. *Fractures.* Philadelphia: JB Lippincott, 1975:733, with permission.)

The remaining 50% of scapular fractures involve its three processes (see Fig. 26-1). Fractures of the glenoid neck constitute approximately 25% of the total, whereas fractures of the glenoid cavity (the glenoid rim and glenoid fossa) make up approximately 10%. (Fractures of the glenoid process, therefore, account for approximately 35% of all scapular fractures.) Approximately 8% of these injuries involve the acromial process, and approximately 7% involve the coracoid process.[127] (An *acromial fracture* is defined as any fracture that runs from the posterior margin of the scapular spine or acromion to the undersurface of the acromial process all the way to the deepest point of the spinoglenoid interval.)

Diagnosis

The physician's attention is initially drawn to the scapular region by the patient's complaints of pain and abnormal physical findings in the area (swelling, crepitus, ecchymosis, and such). The specific diagnosis of a scapular fracture, however, is ultimately radiographic. These injuries are often initially missed[73] or detected incidentally on the patient's admission chest radiograph (Fig. 26-3). A "scapula trauma series" is then indicated, including true anteroposterior (AP) and lateral views of the scapula as well as a true axillary projection of the glenohumeral joint (Fig. 26-4). The scapula is a complex bony structure. One must be able to visualize and evaluate the scapular body and spine as well as its three processes: the glenoid process, the acromial process, and the coracoid process. The glenoid process is composed of the glenoid neck and the glenoid cavity, which, in turn, is made up of the glenoid fossa and the glenoid rim. Finally, the scapula takes part in three articulations (the acromioclavicular [AC] joint, the glenohumeral joint, and the scapulothoracic articulation), each of which must be carefully evaluated. One should look for associated shoulder girdle injuries, including those involving the clavicle, the proximal humerus, and the sternoclavicular joint.

If an injury to the linkage between the clavicle and the scapula (i.e., a disruption of the coracoclavicular and/or acromioclavicular ligaments) is suspected, a weight-bearing AP film of the shoulder should be obtained (see Fig. 26-4). In some situations, transthoracic lateral and oblique projections of the region may be of value. The scapula trauma series should suffice for most injuries, but if the fracture pattern appears to be complex (multiple fracture lines and significant displacement), computed tomography (CT) scanning is necessary. The superior images allow evaluation of the acromioclavicular joint and the acromial process, while the inferior images show the scapular body and spine and scapulothoracic articulation (Fig. 26-5).

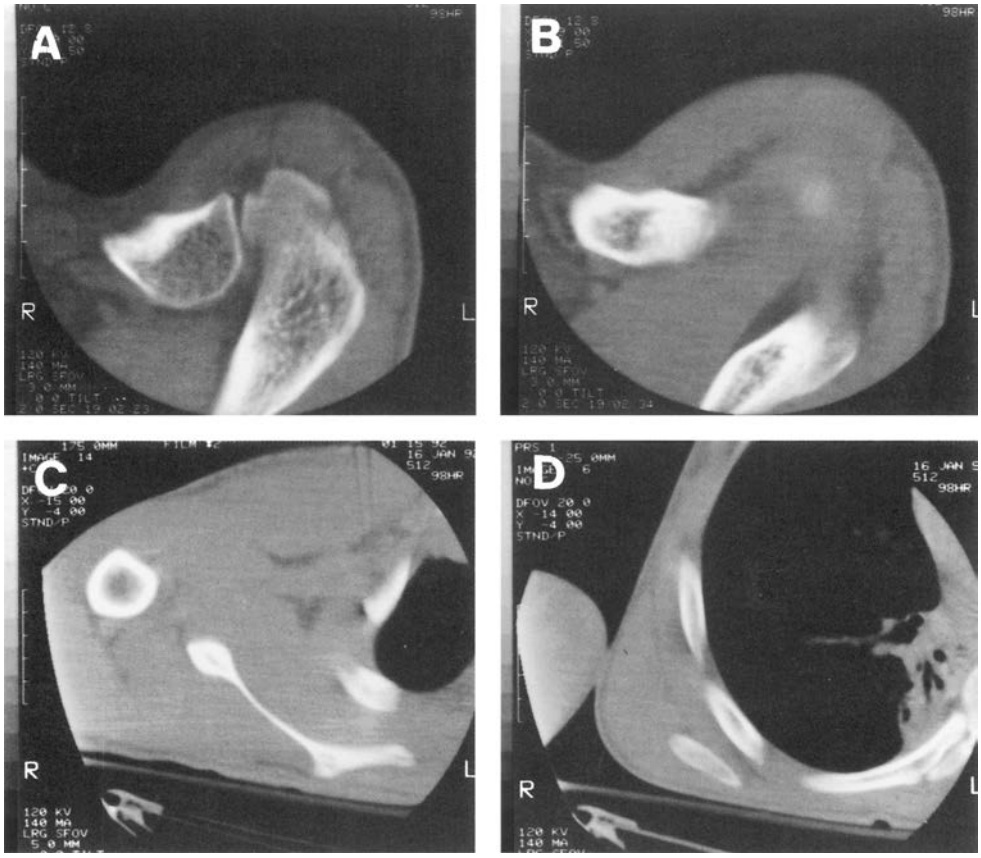


Figure 26-5 Multiple axial computed tomographic images of the scapula: **(A,B)** superior images showing the acromioclavicular joint and the acromion; and **(C,D)** Inferior images showing the scapular body and scapulothoracic articulation.

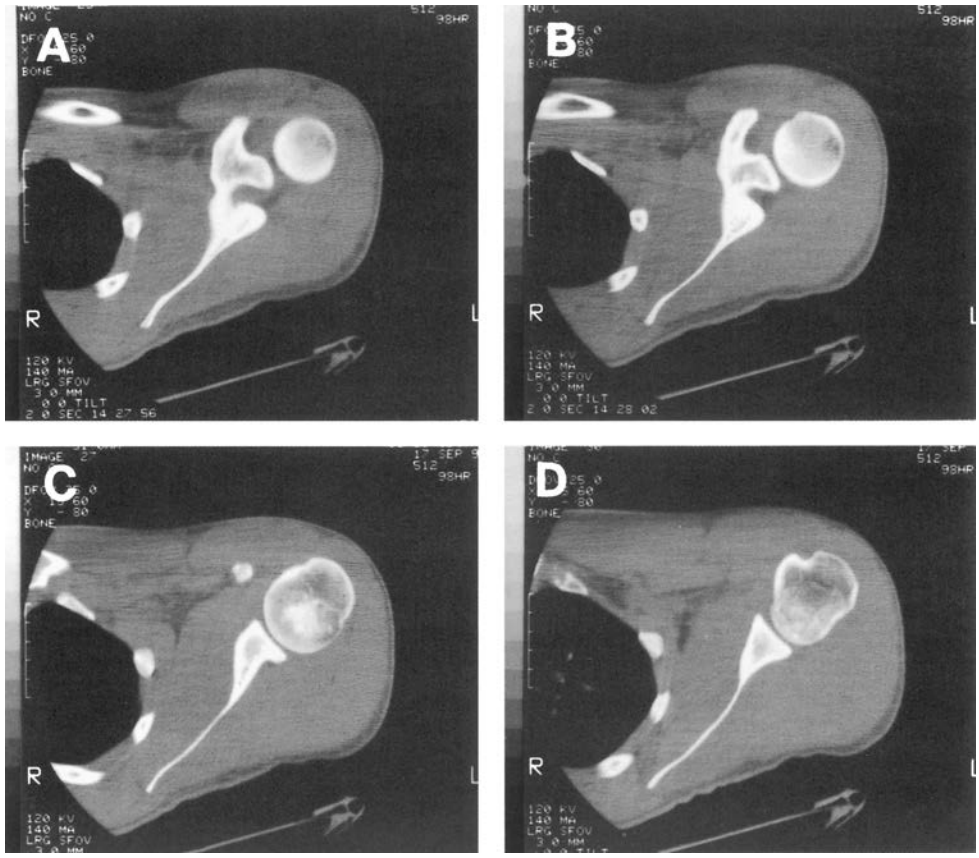


Figure 26-6 Multiple axial computed tomographic images: **(A,B)** most superior; and **(C,D)** most inferior, through the glenohumeral region of the scapula.

The middle images allow one to see the glenoid neck, the glenoid cavity (glenoid rim and glenoid fossa), the coracoid process, and the glenohumeral articulation (Fig. 26-6). In certain clinical situations, reconstructed views can be of great value (Fig. 26-7). Finally, three-dimensional (3-D) scanning can be extremely helpful to the orthopedist trying to evaluate the most complex fracture patterns (Fig. 26-8). Images rotated at 15-degree increments in the horizontal, vertical, and oblique planes can be examined and the most useful projections printed for later reference.

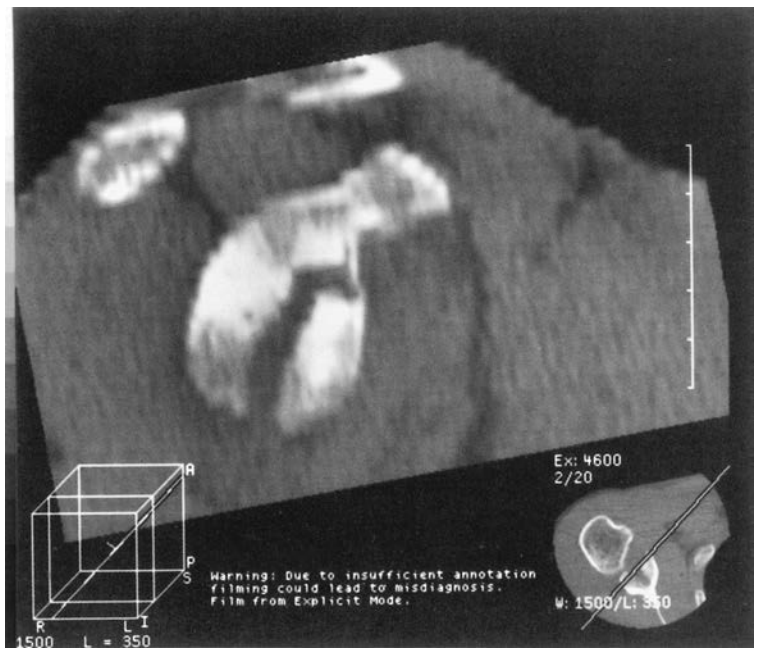


Figure 26-7 A reconstructed computed tomographic image showing the glenoid cavity en face (note the large anteroinferior glenoid rim fragment with severe separation of the articular surface).

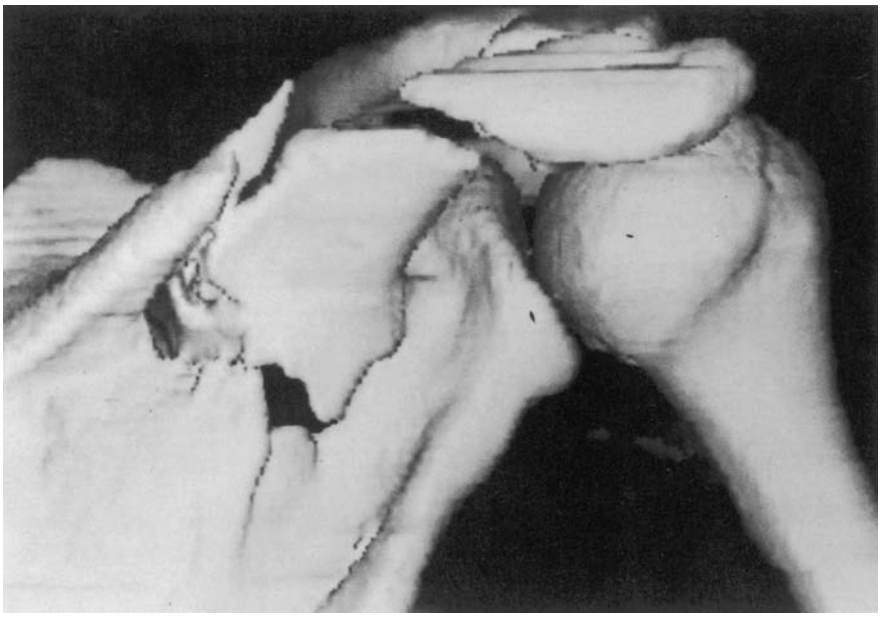


Figure 26-8 A three-dimensional computed tomographic image of an individual who sustained a segmental fracture of the acromion (note the severely displaced intermediate segment).

The proximal humerus can be eliminated for optimal visualization of the glenoid cavity, and other reconstructed images are possible. The exact role, if any, for arthroscopy (both diagnostic and therapeutic) is yet to be defined.

Nonoperative Treatment

Fractures of the scapular body and spine and insignificantly displaced fractures of the glenoid, acromial, and coracoid processes (see sections dealing specifically with injuries to these structures) are managed nonoperatively (Fig. 26-9). The patient's arm is initially placed in a sling and swathe immobilizer for comfort. Local ice packs to the affected area are helpful during the first 48 hours, followed by moist heat thereafter. Analgesic medications are prescribed as needed. Absolute immobilization is generally short term (48 hours), but may continue for up to 14 days, depending on the clinical situation. The patient is then permitted to gradually increase the functional use of his or her upper extremity as symptoms allow and sling and swathe protection is gradually decreased until the 6-week point. Physiotherapy is prescribed during this period and focuses on maintaining and regaining shoulder range of motion. The program begins

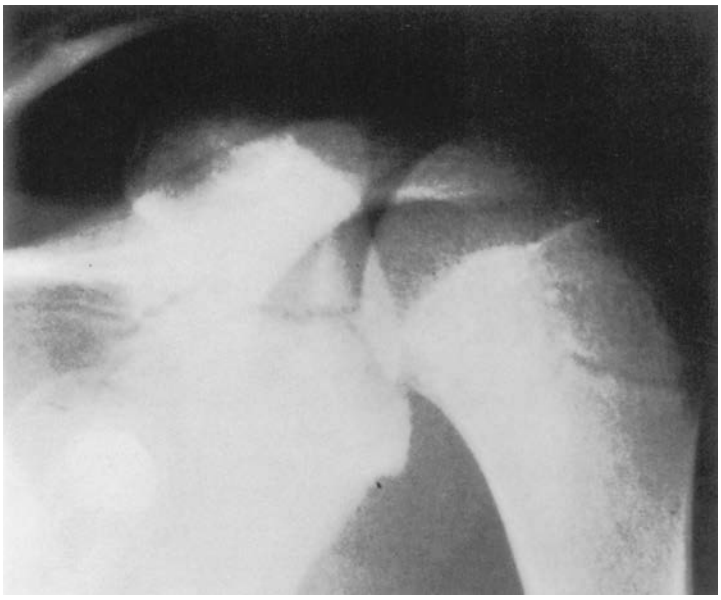


Figure 26-9 An anteroposterior radiograph showing an undisplaced fracture of the scapula involving the glenoid process.

with dependent circular and pendulum movements as well as external rotation to, but not past, neutral, and gradually moves on to progressive stretching techniques in all ranges. Close outpatient follow-up is necessary to monitor and guide the patient's recovery, and x-ray films are obtained at 2-week intervals to ensure that unacceptable displacement does not occur at the fracture site(s). At 6 weeks, osseous union is usually sufficient to discontinue all external protection and encourage functional use of the upper extremity. The rehabilitation program continues until range of motion, strength, and overall function are maximized. Six months to 1 year may be required for full recovery, but a good to excellent result should be readily obtainable.

FRACTURES OF THE GLENOID PROCESS

The glenoid process includes the glenoid neck and the glenoid cavity, which, in turn, is composed of the glenoid rim and the glenoid fossa. Each of these areas may be fractured in a variety of ways. Most investigators agree that if significantly displaced, surgical management is indicated or should at least be considered. Several surgical principles apply.[57,59,61,154]

General Considerations

Surgical Approaches

Depending on the clinical situation, the glenoid process may be approached from three directions or combinations thereof. [65,154] The anterior approach is used for fractures of the anterior glenoid rim and some fractures of the superior aspect of the glenoid fossa. The posterior approach is used for fractures of the posterior rim, most fractures of the glenoid fossa, and fractures of the glenoid neck. The superior approach may be used for (a) fractures of the glenoid fossa with a difficult-to-control superior fragment (in conjunction with either a posterior or an anterior exposure), and (b) fractures of the glenoid neck with a difficult-to-control glenoid fragment (in conjunction with a posterior exposure). Klingman and Roffman described a variation of the posterior approach that they believed allowed open reduction and internal fixation (ORIF) of anterior as well as posterior fragments.[98]

Anterior Approach

For the anterior approach (Fig. 26-10), the patient is placed on the operating room table in the "beach-chair" position. An anterior incision is made in Langer's lines, centered over the glenohumeral joint and running from the superior to the inferior margin of the humeral head. The deltoid muscle is exposed and the deltopectoral interval is dissected over the coracoid process. The conjoined tendon and pectoralis major muscle are retracted medially, whereas the deltoid muscle is retracted laterally with the cephalic vein.

The arm is externally rotated onto a sterile arm support (or a sterile arm positioner can be used) and the subacromial bursa is removed, exposing the subscapularis tendon. (In type III glenoid cavity fractures, opening the rotator interval may allow sufficient exposure.) The tendon is incised 2.5 cm medial to the medial border of the biceps groove and along its superior and inferior borders. It is then dissected off the underlying anterior glenohumeral capsule–glenoid neck periosteum and turned back medially. The anterior gleno-

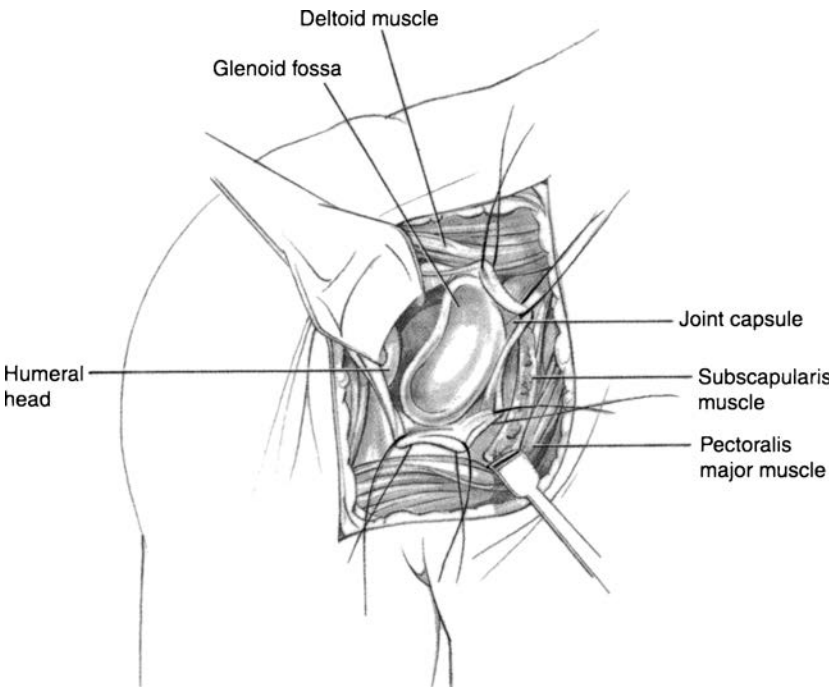


Figure 26-10 Anterior surgical approach to the glenoid cavity.

humeral capsule is incised in the same fashion (5 mm medial to the anatomic neck) and also turned back medially. With a humeral head retractor inserted into the glenohumeral joint (the Fukuda ring retractor is especially useful[46]) and holding the humeral head out of the way, the entire glenoid cavity can be inspected and the surgeon has ready access to its anterior rim. One must take care to avoid injury to the nearby axillary nerve.

Posterior Approach

The patient is placed on the operating room table in the lateral decubitus position, supported by a bean bag. An incision is made over the lateral one-third of the scapular spine, along the posterior aspect of the acromion to its lateral tip, and then distally in the midlateral line for a distance of 2.5 cm. Skin flaps are developed. The deltoid is dissected sharply off the scapular spine and the acromion and then split in the line of its fibers for a distance of not more than 5 cm starting at the lateral tip of the acromion. The deltoid is separated off the underlying infraspinatus and teres minor musculotendinous units and retracted down to, but not below, the inferior margin of the teres minor. (This approach allows maximal exposure; however, a more cosmetic vertical incision from the posterior axillary crease to the scapular spine with superolateral retraction of the undetached posterior deltoid muscle may suffice, especially if access to only the posteroinferior aspect of the glenoid cavity is needed, that is, type Ib and II glenoid cavity fractures.) The infraspinatus tendon is incised 2.5 cm medial to the greater tuberosity and along its superior and inferior borders. It is then dissected off the underlying posterior glenohumeral capsule and turned back medially. The posterior glenohumeral capsule is incised in the same fashion and also turned back medially. (Depending on the particular clinical situation, one may have sufficient exposure by detaching only the lower one-half of the insertion of the infraspinatus tendon and posterior glenohumeral capsule upon the humeral head, making an incision along their inferior border, and retracting the flap thereby created superiorly and medially.[61]) With injuries that involve only the posteroinferior and inferior aspects of the glenoid cavity, one may be able to avoid detaching these structures at all—developing the infraspinatus–teres minor interval and making a linear incision in the capsule may allow adequate access. With a Fukuda retractor inserted into the joint and holding the humeral head out of the way, the entire glenoid cavity can be inspected and the surgeon has ready access to its posterior rim as well as the glenoid neck (Fig. 26-11). The interval between the infraspinatus and teres minor muscles can be developed further and the long head of the triceps detached to gain access to the inferior aspect of the glenoid process and the lateral border of the scapular body (Fig. 26-12). One must take particular care to protect and avoid injury to the nearby suprascapular and axillary nerves.

Superior Approach

The superior approach can be added to either the anterior or the posterior exposure (Figs. 26-13 and 26-14) if a displaced, difficult-to-control or -stabilize superior glenoid fragment or glenoid process fragment is present. Either incision is

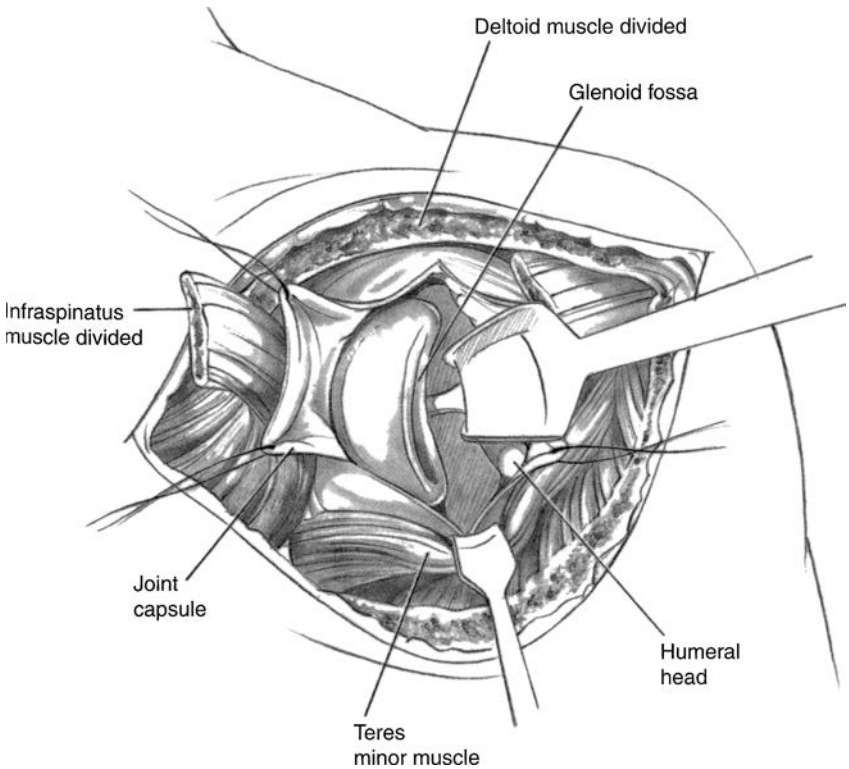


Figure 26-11 Posterior surgical approach to the glenoid cavity: standard exposure.

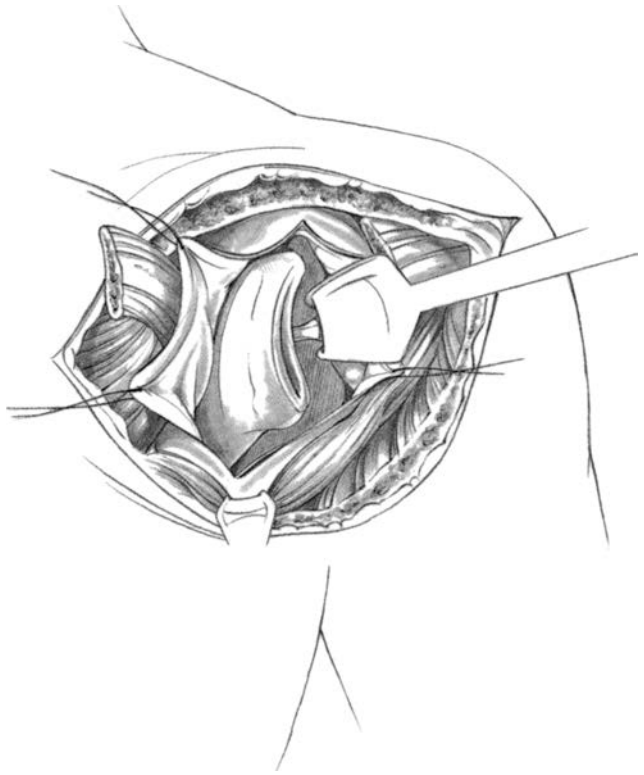


Figure 26-12 Posterior surgical approach to the glenoid cavity: development of the infraspinatus–teres minor interval to expose the posteroinferior glenoid cavity and the lateral scapular border.

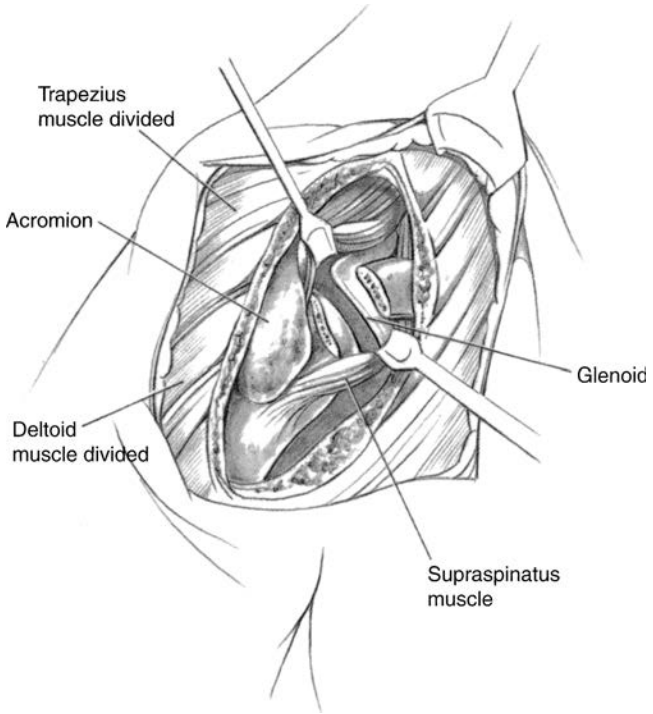


Figure 26-13 Superior surgical approach to the glenoid cavity: soft tissue and bony anatomy.

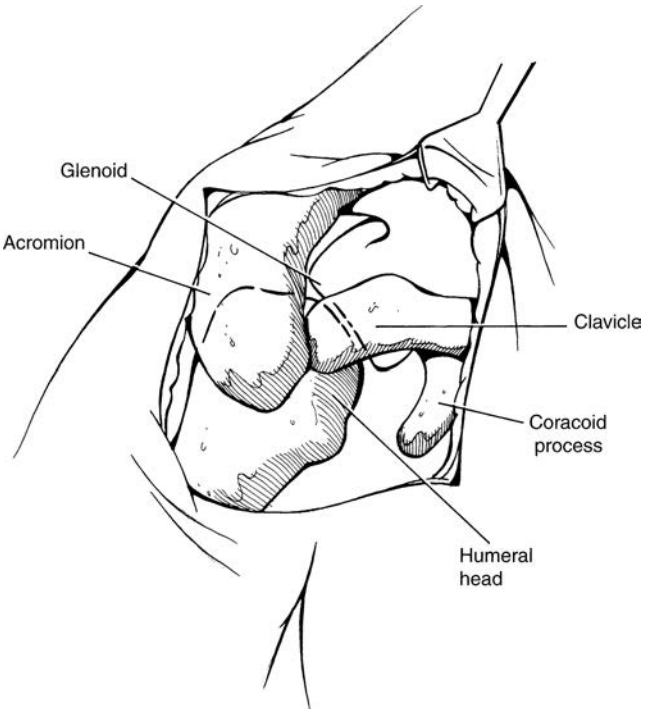


Figure 26-14 Superior surgical approach to the glenoid cavity: bony anatomy.

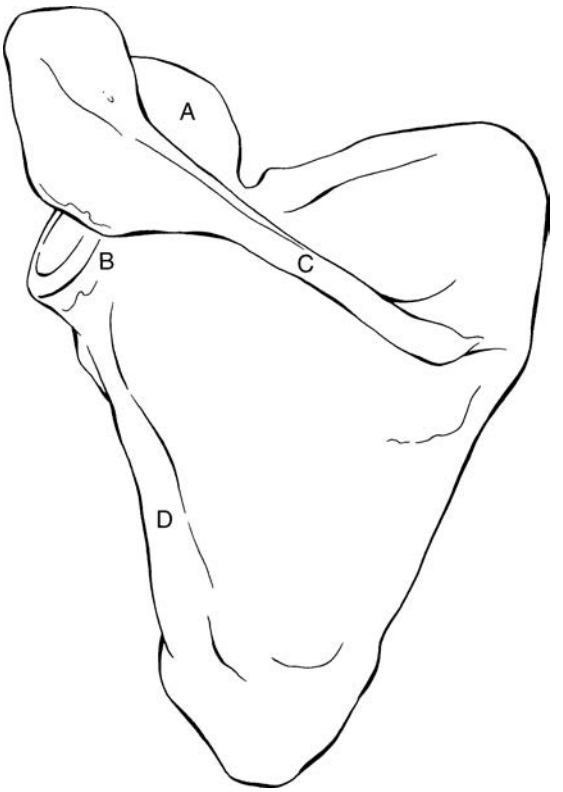


Figure 26-15 Illustration depicting the scapula and areas of sufficient bone stock for internal fixation: **(A)** the coracoid process; **(B)** the glenoid process; **(C)** the scapular spine; and **(D)** the lateral scapular border. (From Goss TP. Fractures of the glenoid cavity [operative principles and techniques]. *Tech Orthop* 1994;8:199, with permission.)

extended over the superior aspect of the shoulder. Soft tissue flaps are developed and retracted, exposing the superior aspect of the distal clavicle, the AC joint, the acromion, and the trapezius muscle. In the interval between the clavicle and the acromion (posteromedial to the acromioclavicular joint), the trapezius muscle and the underlying supraspinatus tendon are split in the line of their fibers, bringing one down upon the superior aspect of the glenoid process (the superior glenoid rim is located posterolaterally) and the base of the coracoid process (located anteromedially). One must take care to protect and avoid injury to the suprascapular nerve and vessels that lie medial to the coracoid process.

Bone Stock

Thick, solid bone for fixation is at a premium because much of the scapula is paper-thin. There are, however, four satisfactory areas: the glenoid neck, the lateral scapular border, the base of the scapular spine, and the coracoid process (Fig. 26-15).

Fixation Devices

A variety of fixation devices are available (Fig. 26-16). The most useful, however, are K-wires, malleable reconstruction plates (Fig. 26-17), and cannulated interfragmentary compression screws (Fig. 26-18). K-wires can be used for temporary as well as permanent fixation. The latter is the case when significantly displaced fracture fragments are too small to allow more substantial fixation, but one must be sure to bend the K-wire at its point of entry to prevent migration. Particularly helpful in the management of glenoid neck fractures are 3.5-mm malleable reconstruction plates, and 3.5- and 4.0-mm cannulated compression screws are especially useful in stabilizing fractures of the glenoid rim and glenoid fossa. These devices may be used

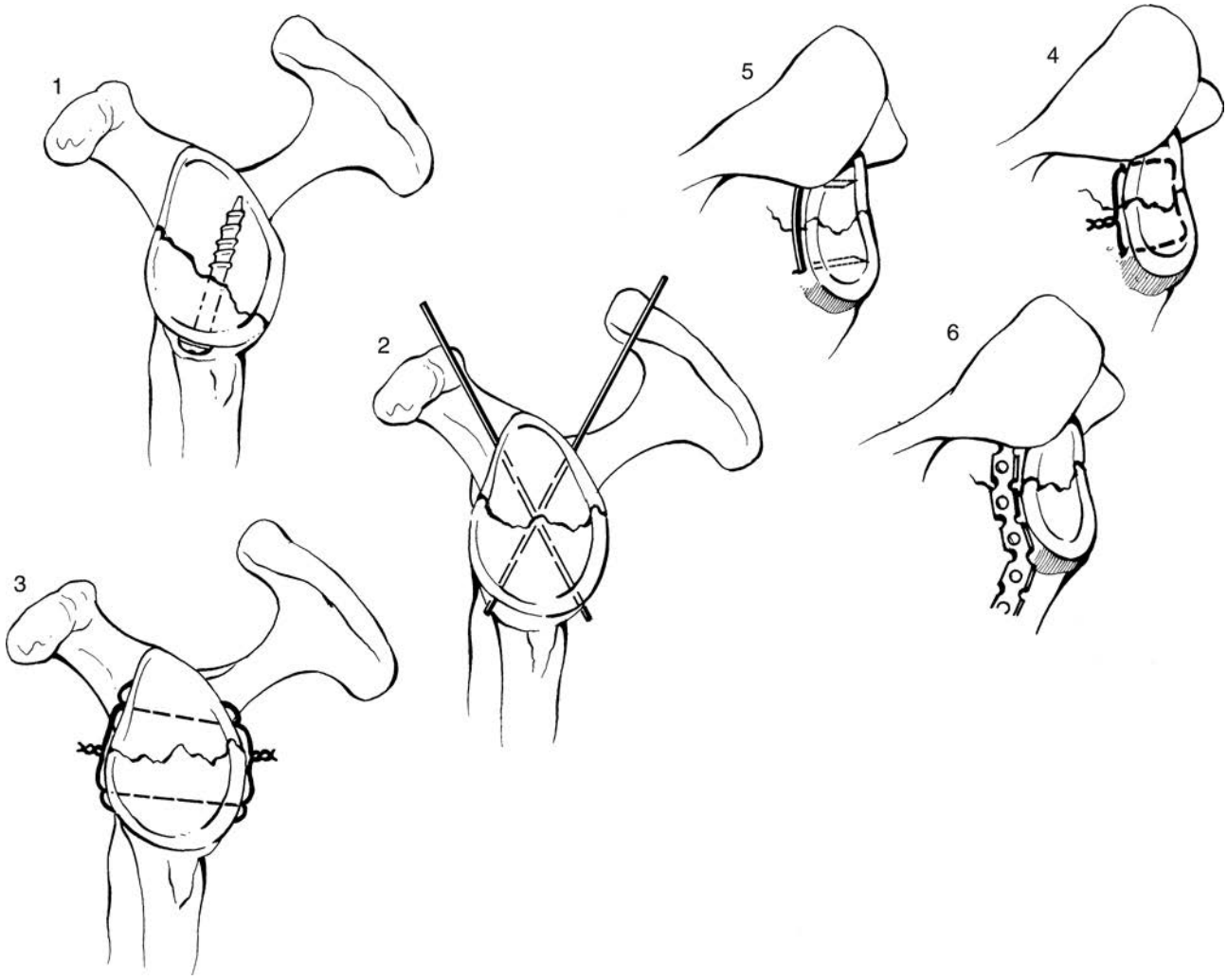


Figure 26-16 Illustrations depicting fixation techniques available for stabilization of fractures of the glenoid cavity: **(1)** an interfragmentary compression screw; **(2)** Kirschner wires; **(3)** a construct using Kirschner wires and cerclage wires or Kirschner wires and cerclage sutures; **(4)** a cerclage wire or suture; **(5)** a staple; and **(6)** a 3.5-mm malleable reconstruction plate.

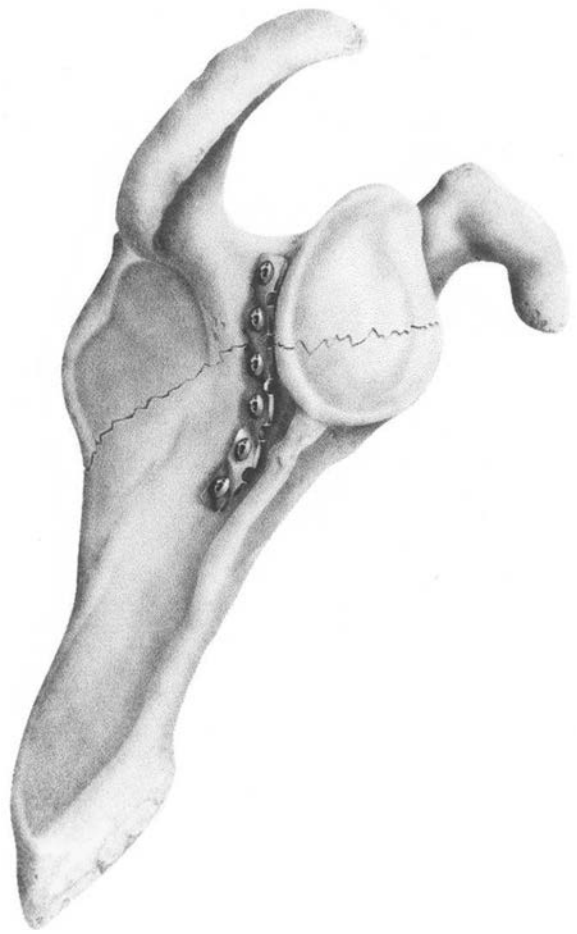


Figure 26-17 Illustration showing reduction and stabilization of a glenoid cavity fracture with a 3.5-mm malleable reconstruction plate. (From Goss TP. Glenoid fractures: open reduction and internal fixation. In: Wiss DA, ed. *Master techniques in orthopaedic surgery: fractures.* Philadelphia: Lippincott-Raven, 1998:10, with permission.)

alone or in combination, depending on the clinical situation and taking into account the available bone stock as well as the surgeon's preference and experience. Rigid fixation is desirable, but inability to achieve this goal does not preclude an excellent anatomic and functional result.

Fractures of the Glenoid Cavity

Classification and Incidence

Fractures of the glenoid cavity make up 10% of scapular fractures. The majority (90+%) are insignificantly displaced and managed nonoperatively. Those that are significantly displaced require surgical treatment or at least deserve surgical consideration. Ideberg reviewed over 300 such injuries and proposed the first detailed classification scheme.[85–87] This was subsequently expanded by Goss[58] (Fig. 26-19). Type I fractures involve the glenoid rim: type la, the anterior glenoid rim, and type Ib, the posterior rim. Fractures of the glenoid fossa make up types II to V. Type VI fractures include all comminuted (more than two glenoid cavity fragments) injuries.

Fractures of the Glenoid Rim

Fractures of the glenoid rim occur when the humeral head strikes the periphery of the glenoid cavity with considerable violence (Fig. 26-20). These are fractures distinct from the small avulsion injuries that occur when a dislocating humeral head applies a tensile force on the periarticular soft tissues.[32] Surgical management is indicated if the fracture results in persistent subluxation of the humeral head (failure of the humeral head to lie concentrically within the glenoid cavity) or if the reduction of the fracture or humeral head is unstable. DePalma[32] stated that instability could be expected if the fracture was displaced 10 mm or more and if one-fourth or more of the glenoid cavity anteriorly or one-third or more of the glenoid cavity posteriorly was involved. Hardegger et al.[71] concurred and stated that "operative reduction and fixation of the fragment is indicated to prevent recurrent or permanent dislocation of the shoulder." Guttentag and Rechtine[68] and Butters[23] agreed with these recommendations. Several papers describing operatively managed glenoid rim fractures have also appeared in the literature.[7,105,147,178,194] A true axillary view of the glenohumeral joint, CT images (routine and reconstructed), and, if necessary, 3-D scanning allow one to determine the size and displacement of the rim fragment, whether persistent subluxation of the humeral head is present, and, therefore, whether stability of the glenohumeral articulation is significantly compromised. Surgery, if necessary, is designed to restore articular stability and prevent posttraumatic degenerative joint disease (Fig. 26-21). Type Ia (anterior rim) fractures are approached anteriorly. The displaced fragment is mobilized, reduced anatomically, and fixed in position with cannulated interfragmentary compression screws (ideally two screws are used to provide rotational stability; Fig. 26-22). Type Ib (posterior rim) fractures are approached posteriorly, reduced, and stabilized in the same manner (Fig. 26-23). If the fracture is comminuted and cannot be internally fixed, the fragments are excised. A tricortical graft harvested from the iliac crest is then placed intra-articularly, filling the defect (Fig. 26-24). A simple repair of the periarticular soft tissues to the intact glenoid cavity is an option if the bone defect is less than 20% of the anterior-to-posterior dimension of the glenoid fossa, but restoration of rim contour is preferable.

Fractures of the Glenoid Fossa

Classification, Mechanism, and Surgical Indications

Fractures of the glenoid fossa occur when the humeral head is driven with sufficient force into the center of the glenoid

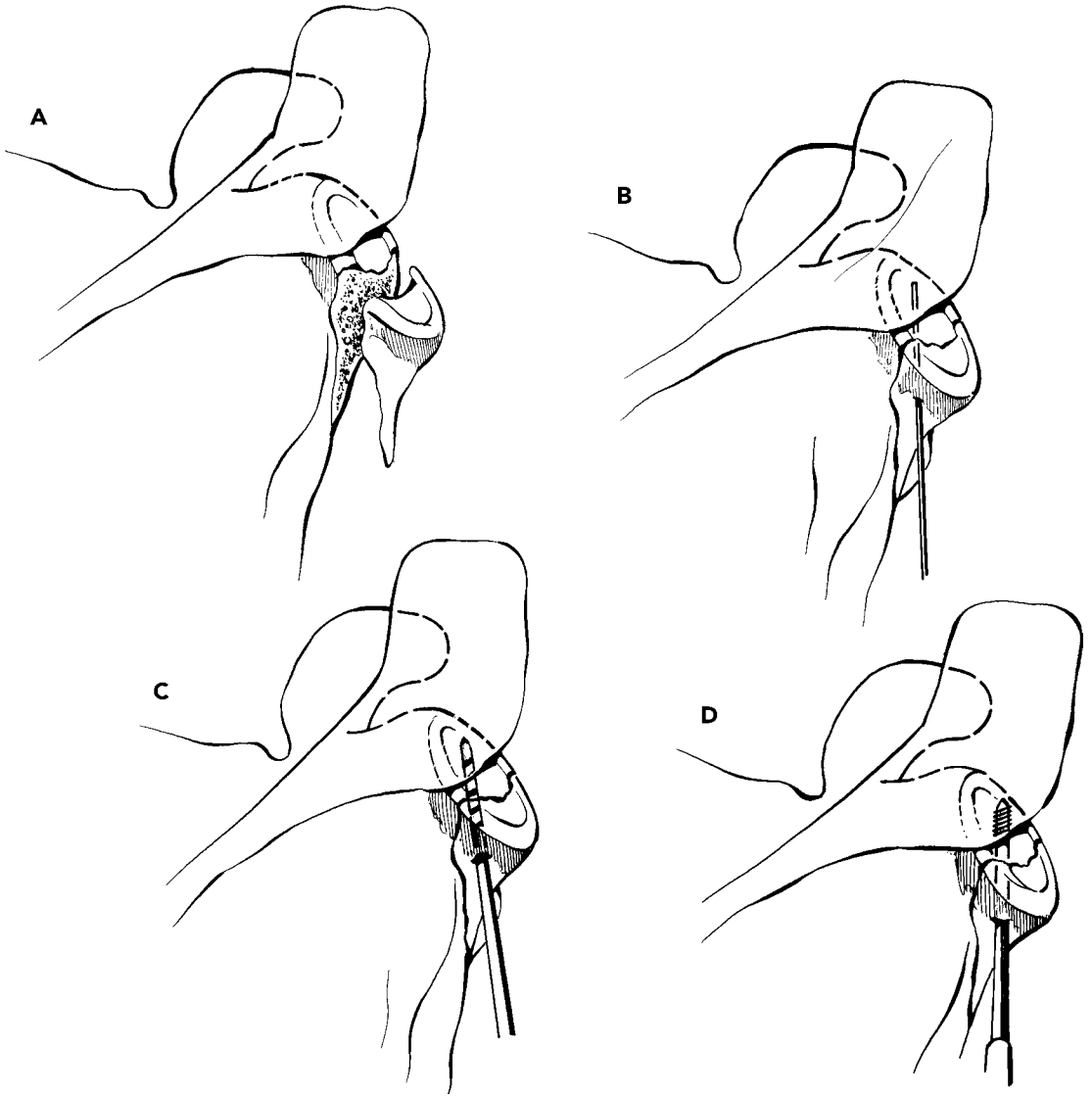


Figure 26-18 Illustrations showing reduction and stabilization of a type II fracture of the glenoid cavity using a cannulated interfragmentary compression screw: **(A)** a fracture of the glenoid cavity with a significantly displaced inferior glenoid fragment; **(B)** reduction of the glenoid fragment and stabilization with a guidewire; **(C)** use of the guidewire to pass a cannulated drill and eventually a cannulated tap; and **(D)** use of the guidewire to place a cannulated interfragmentary compression screw securely fixing the glenoid fragment in position.

concavity. The fracture generally begins as a transverse disruption (or slightly oblique) for several possible reasons: (a) the glenoid cavity is concave so forces tend to be concentrated over its central region; (b) the subchondral trabeculae are transversely oriented so fractures tend to occur in this plane; (c) the glenoid cavity is formed from two ossification centers so the central region may remain a persistently weak area; and (d) the glenoid cavity is narrow superiorly and wide inferiorly with an indentation along its anterior rim—this constitutes a stress riser where fractures are particularly prone to originate before coursing over to the posterior rim (Fig. 26-25). Once this transverse disruption occurs, the fracture may propagate in a variety of directions depending on the exact direction of the humeral head force.

With type II fractures, the humeral head is driven inferiorly, creating an inferior glenoid fragment. Surgery is indicated if there is an articular step-off of 5 mm or more and/or if the fragment displaces inferiorly carrying with it the humeral head, such that the humeral head fails to lie in the center of the glenoid cavity. These injuries can result in posttraumatic degenerative joint disease and/or glenohumeral instability.[56]

Type III fractures occur when the force of the humeral head is directed superiorly, causing the transverse disruption to propagate upward, generally exiting through the superior scapular border in the vicinity of the suprascapular notch. One might question whether this is a fracture of the glenoid cavity or a fracture of the coracoid process,

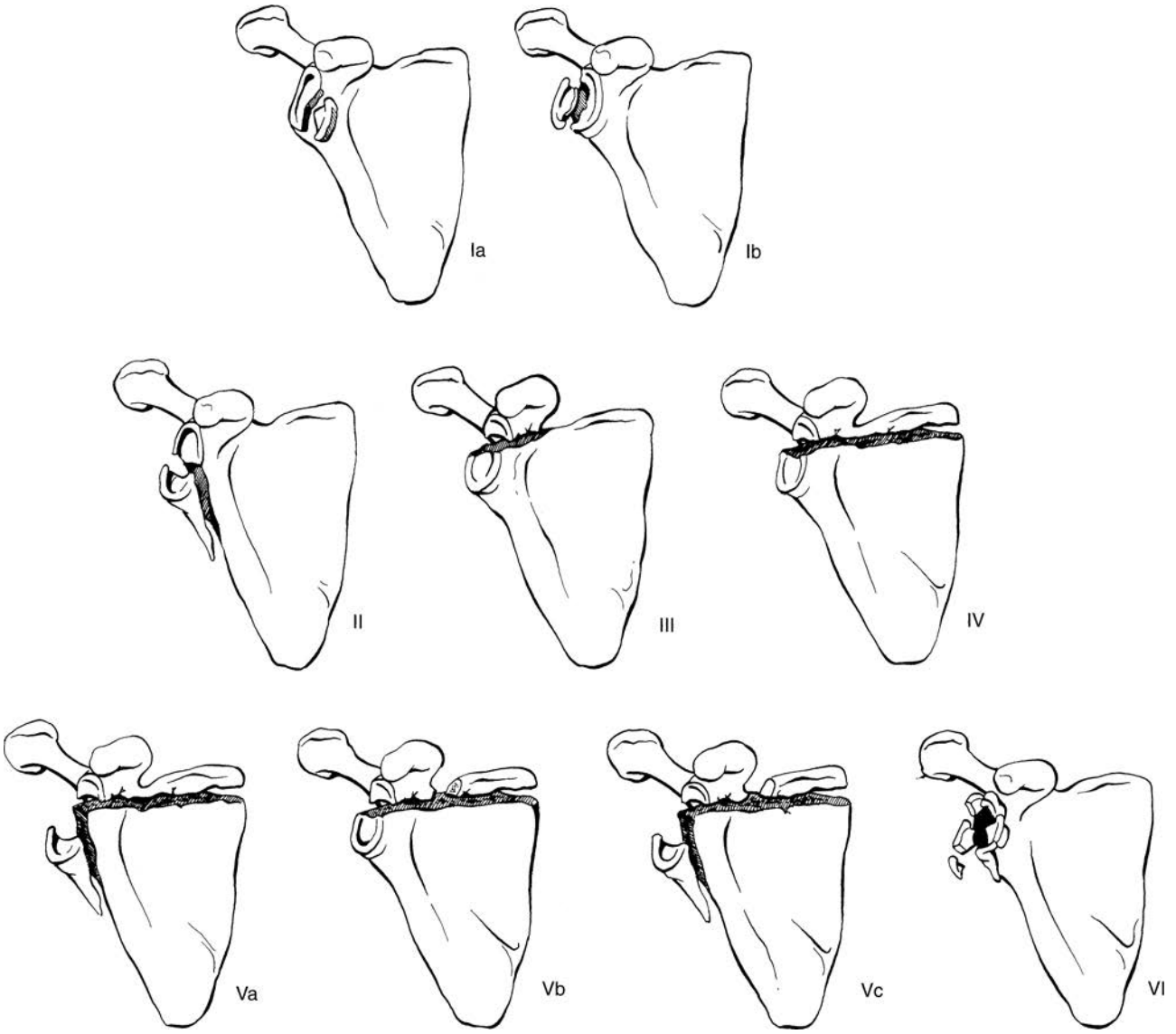


Figure 26-19 Goss-Ideberg classification scheme for fractures of the glenoid cavity.

since the superior third of the glenoid cavity and the base of the coracoid process are formed from the same ossification center (see Fig. 26-26B).[39,122] Regardless, displacement is usually minimal with the fragment lying medially. Consequently, as with fractures of the base of the coracoid process, these injuries are usually treated nonoperatively and heal uneventfully. Any glenoid cavity fracture may be associated with neurovascular damage owing to the proximity of the brachial plexus and axillary vessels as well as the considerable violence involved. Type III as well as type Vb, Vc, and VI injuries, however, are particularly prone, especially if there is an associated disruption of the clavicular–coracoclavicular (CC) ligamentous–coracoid (C-4) linkage or the clavicular–AC joint–acromial strut. Neer and Rockwood considered compression of the adjacent neurovascular structures by these and fractures of the coracoid process an indication for surgery.[143] They and others also described the occurrence of suprascapular nerve paralysis resulting from fractures involving the coracoid process and glenoid neck and extending into the suprascapular notch. (An electromyogram [EMG] was essential for diagnosis, and early exploration was recommended.[28,36,39,180])

Surgical management is considered if there is an articular step-off of 5 mm or more, with the superior fragment displaced laterally, or if there is a severe associated disruption of the superior shoulder suspensory complex[55] (the clavicular–CC ligamentous–coracoid [C-4] linkage or the clavicular–AC joint–acromial strut). These injuries can result in posttraumatic degenerative joint disease and severe functional impairment.

Type IV injuries occur when the humeral head is driven directly into the center of the glenoid fossa.[44] The

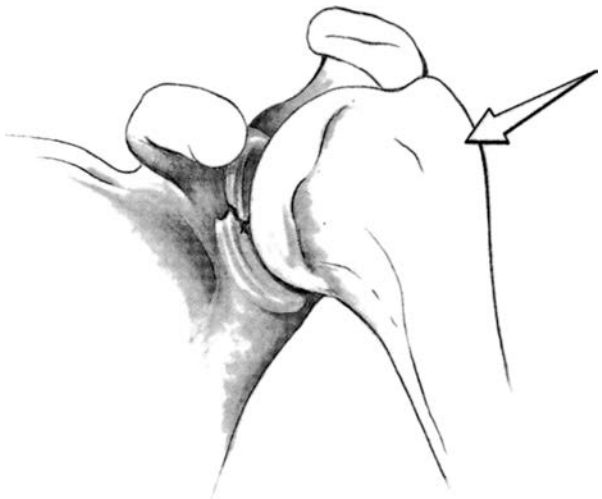


Figure 26-20 Illustration depicting one mechanism of injury responsible for fractures of the glenoid rim: a force applied over the lateral aspect of the proximal humerus (a fall on an outstretched arm driving the humeral head against the periphery of the glenoid cavity with considerable violence could also cause this injury).

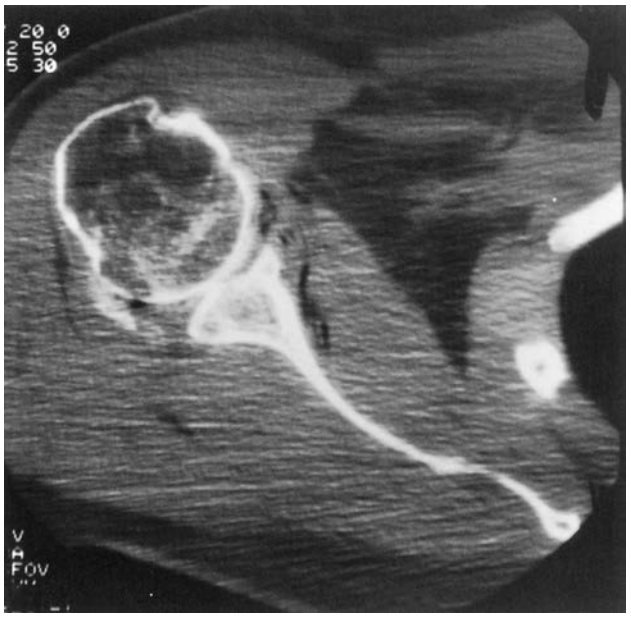


Figure 26-21 An axial computed tomographic image of an individual 9 months following a traumatic event. Note the previously undiagnosed displaced type Ia fracture of the glenoid cavity with anterior subluxation of the humeral head and "bone-on-bone" contact (intraoperatively the patient had significant posttraumatic degenerative disease of the glenohumeral joint).

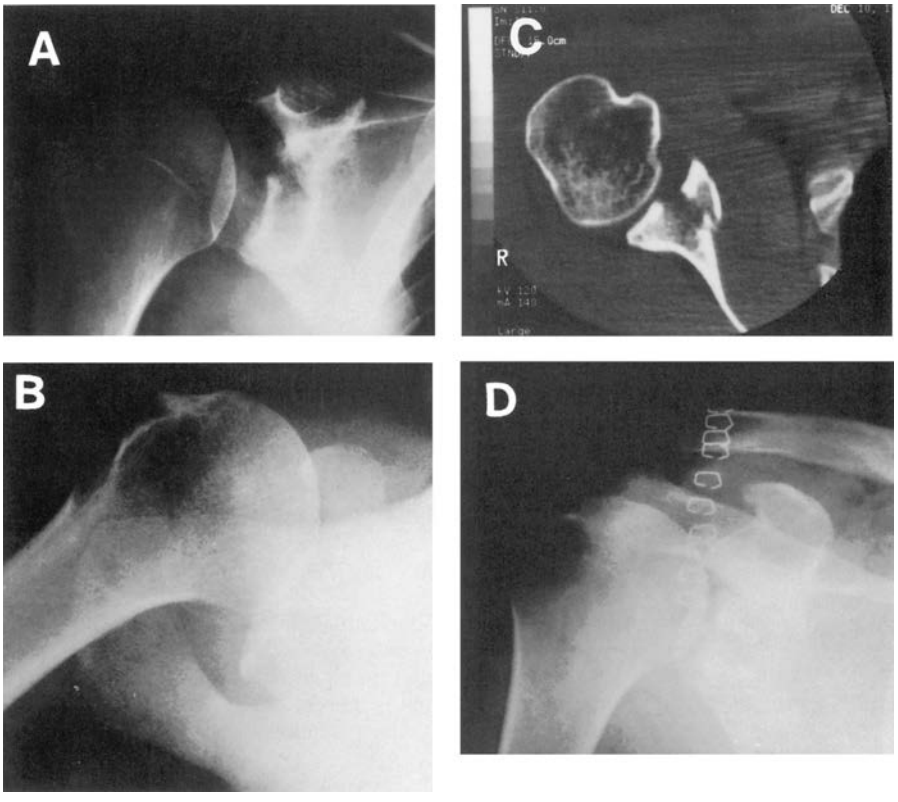


Figure 26-22 Radiographs of a person who sustained a type Ia fracture of the glenoid cavity: **(A)** a preoperative anteroposterior (AP) radiograph showing what appears to be a fracture of the anteroinferior glenoid rim; **(B)** a preoperative axillary radiograph showing what appears to be a fracture of the anterior glenoid rim with anterior subluxation of the humeral head; **(C)** an axial computed tomographic image showing a severely displaced fracture of the anterior glenoid rim; and **(D)** a postoperative AP radiograph showing reduction and stabilization of the anteroinferior glenoid rim fragment with two cannulated interfragmentary screws.

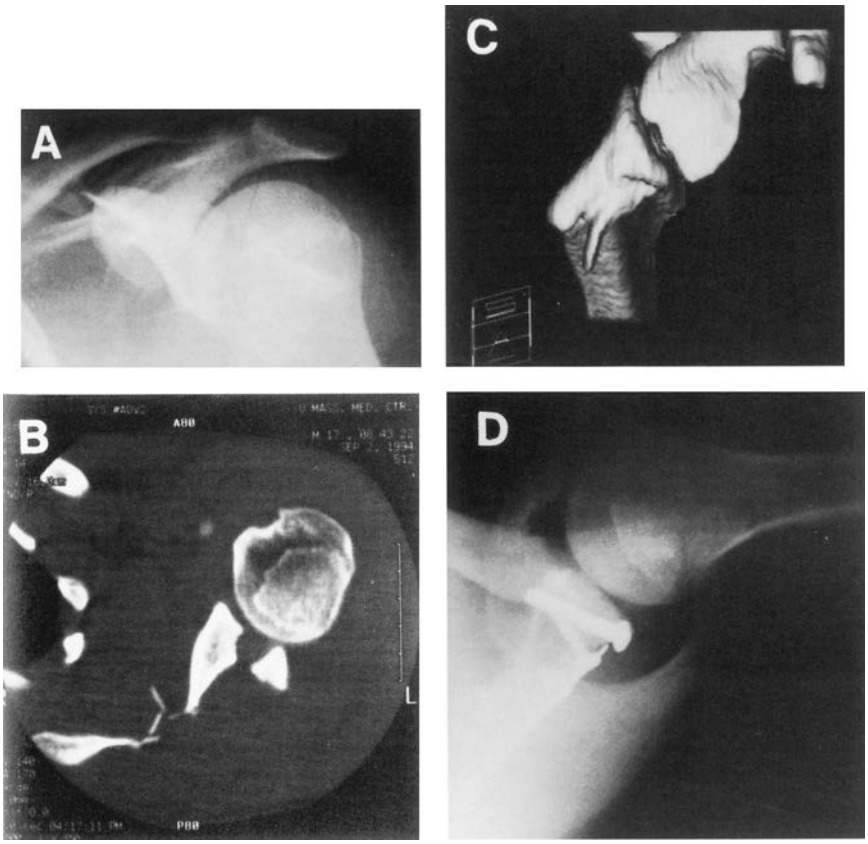


Figure 26-23 Radiographs of an individual who sustained a type Ib fracture of the glenoid cavity: **(A)** a preoperative lateral scapular radiograph showing what appears to be a fracture of the glenoid cavity with significant posterior involvement; **(B)** an axial computed tomographic **(A,B)** image showing a severely displaced fracture of the posterior glenoid rim with posterior subluxation of the humeral head; **(A)** a three-dimensional CT image of the glenoid cavity with the humeral head subtracted showing the severely displaced and rotated posteroinferior glenoid rim fragment; and **(D)** a postoperative axillary radiograph showing anatomic reduction and stabilization of the posterior glenoid rim fragment, with restoration of articular congruity.

fracture created courses transversely across the entire scapula, exiting along its vertebral border. If there is an articular step-off of 5 mm or more, with the superior fragment displaced laterally, or if the superior and inferior fragments are severely separated, open reduction and internal fixation is indicated to avoid symptomatic degenerative joint disease, nonunion at the fracture site (an extremely rare occurrence,[40] but a definite concern in the case shown in Fig. 26-27), and/or instability of the glenohumeral joint.

Type Va, Vb, and Vc fractures are combinations of the types II, III, and IV injuries and are caused by more violent and complex forces. The same clinical concerns and operative indications detailed for the type II, III, and IV fractures apply.

Type VI fractures include all disruptions of the glenoid cavity in which more than two articular fragments are present and are caused by the most violent forces. Operative treatment is contraindicated because exposing these injuries surgically will do little more than disrupt what little soft tissue support remains, rendering the fragments even more unstable and making a bad situation worse. An associated disruption of the superior shoulder suspensory complex may warrant operative correction, however, and this may indirectly improve glenoid articular congruity.

Diagnosis

A true AP projection of the glenohumeral joint, reconstructed CT images in the coronal plane, and even 3-D CT scanning may be necessary to accurately determine whether and to what degree articular incongruity and/or separation is present.[71]

Surgical Management

Type II Fractures

These fractures are approached posteriorly. The infraspinatus–teres minor interval is developed, exposing the displaced inferior glenoid fragment and the lateral scapular border. The fragment is reduced as anatomically as possible and stabilized, generally with two cannulated interfragmentary compression screws passed posteroinferiorly to anterosuperiorly or with a contoured reconstruction plate placed along the posterior aspect of the glenoid process and the lateral scapular border (Fig. 26-28). Excision of the fracture fragment and placement of a bone graft from the iliac crest is an option if severe comminution is present. Associated tears of the labral–capsular–ligamentous complex are repaired if possible, as they are with all fractures of the glenoid cavity. Detachments are corrected with nonabsorbable sutures passed through drill holes or use of suture

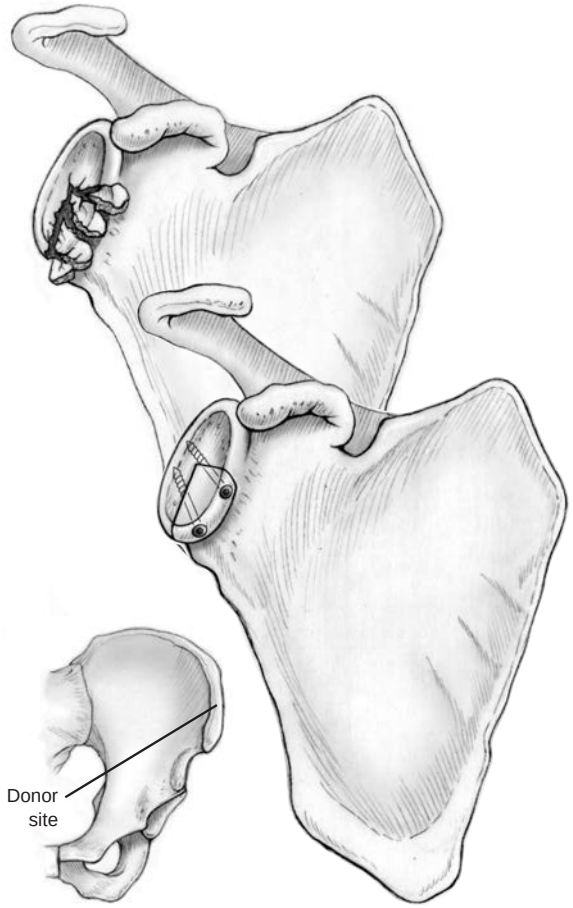


Figure 26-24 Illustration depicting the use of a tricortical graft harvested from the iliac crest to reestablish bony stability in a patient with a severely comminuted type Ia fracture of the glenoid cavity. (Adapted from Goss TP. Fractures of the scapula. In: Rockwood CA, Matsen FA, Wirth MA, Lippitt SB, eds. *The shoulder*, 3rd ed. Philadelphia: Saunders, 2004:413)

anchors, and intrasubstance tears are reapproximated with nonabsorbable sutures placed in a figure-eight fashion.

Type III Fractures

A posterosuperior surgical exposure (or an anterior approach through the rotator interval without taking down the subscapularis musculotendinous unit; see Fig. 26-29) is utilized. A K-wire can be placed into the superior glenoid fragment and then used to manipulate the fragment into satisfactory position relative to the remainder of the glenoid process, thereby restoring articular congruity. The K-wire is then driven across the fracture site and used to place a cannulated interfragmentary compression screw. When there is an associated disruption of the superior shoulder suspensory complex (SSSC), reduction and stabilization of the superior glenoid fragment may restore the integrity of this complex satisfactorily. If not, that injury may need to be addressed as well (Fig. 26-30). Conversely, if the superior glenoid fragment is severely comminuted and difficult to fix, operative restoration of the associated SSSC disruption may improve glenoid articular congruity indirectly and satisfactorily.

Type IV Fractures

These fractures are approached posterosuperiorly. A K-wire is placed into the superior glenoid–scapular segment and then used to manipulate that fragment into position relative to the inferior segment while directly visualizing the reduction via the posterior exposure. The K-wire is then driven across the fracture site and used to place a cannulated interfragmentary compression screw (see Fig. 26-27). A cerclage wire or cerclage sutures passed around the glenoid neck have also been used for fixation. As always, one must take care to avoid injury to adjacent neurovascular structures, in particular the suprascapular nerve and accompanying vessels that pass through the spinoglenoid notch.

Type V Fractures

Type Va fractures are approached, reduced, and stabilized according to the principles described for significantly displaced type II fractures, although a superior approach may need to be added to gain control of the superior glenoscapular fragment. The inferomedial portion of the scapula may be ignored. Type Vb injuries are approached, reduced, and stabilized according to the principles described for significantly displaced type III fractures. The superomedial portion of the scapula may be ignored. If an associated disruption of the superior shoulder suspensory complex is present, it may require operative reduction and stabilization.

Type Vc fractures are exposed by a posterosuperior approach. The superior and inferior glenoid fragments are reduced anatomically and rigidly fixed to each other. Ideally, the fixation is performed with a lag screw, which is generally easier to pass superior to inferior, or with a cerclage wire or suture that is passed through drill holes or suture anchors in the superior and inferior fragments. The glenoid fragment is then reduced and stabilized relative to the scapular body using the principles described for ORIF of glenoid neck fractures. The superomedial and inferomedial portions of the scapula may be ignored. Surgical management of an associated disruption of the superior shoulder suspensory complex is dependent on the exact nature of that injury (Fig. 26-31) (e.g., an associated clavicle fracture may be managed with plate and screw fixation or an intramedullary device).

Type VI Fractures: Management

The upper extremity is initially protected in a sling and swathe bandage, an abduction brace, or even overhead olecranon pin traction—whichever maximizes articular congruity as determined radiographically. Gentle passive circular and rotatory range-of-motion exercises performed by a

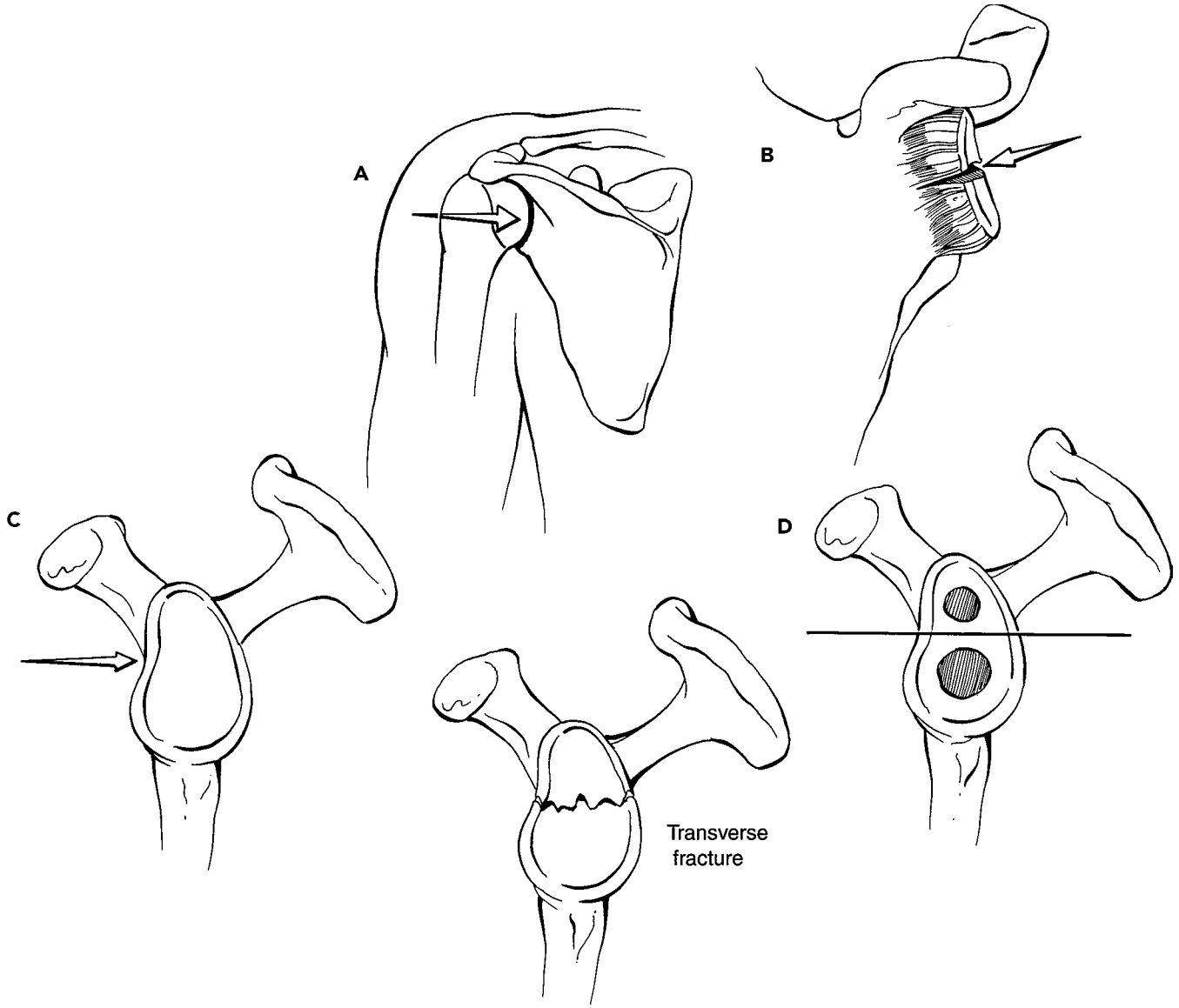


Figure 26-25 Illustrations depicting a transverse disruption of the glenoid cavity and the factors responsible for this orientation: **(A)** the glenoid's concave shape concentrates forces across its central region; **(B)** the subchondral trabeculae are oriented in the transverse plane; **(C)** the "crook" along the anterior rim is a stress riser where fractures tend to originate; **(D)** formed from a superior and an inferior ossification center, the glenoid cavity may have a persistently weak central zone.

therapist and the patient are initiated immediately, in hope that the movement of the humeral head will mold the articular fragments into a maximally congruous position. By 2 weeks, healing is sufficient to allow protection of all such injuries in a sling and swathe immobilizer. Exercises designed to gradually increase range of motion and progressive functional use of the shoulder out of the sling within clearly defined limits are prescribed during the subsequent 4 weeks. At 6 weeks, these fractures are sufficiently healed to allow discontinuation of all external protection. Functional use of the shoulder is encouraged and physiotherapy continues until range of motion and strength are maximized. These injuries obviously have the highest potential for posttraumatic degenerative joint disease and glenohumeral instability.

Results

Bauer et al.[12] reviewed 20 patients treated surgically for significantly displaced fractures of the scapula (6.1-year average follow-up) and reported greater than 70% good or very good results based on a Constant score. They recommended early ORIF for grossly displaced fractures of the glenoid fossa as well as those of the glenoid rim, glenoid neck, and coracoid–acromial processes. Hardegger et al.[71] reported that "if there is significant displacement, conserva-

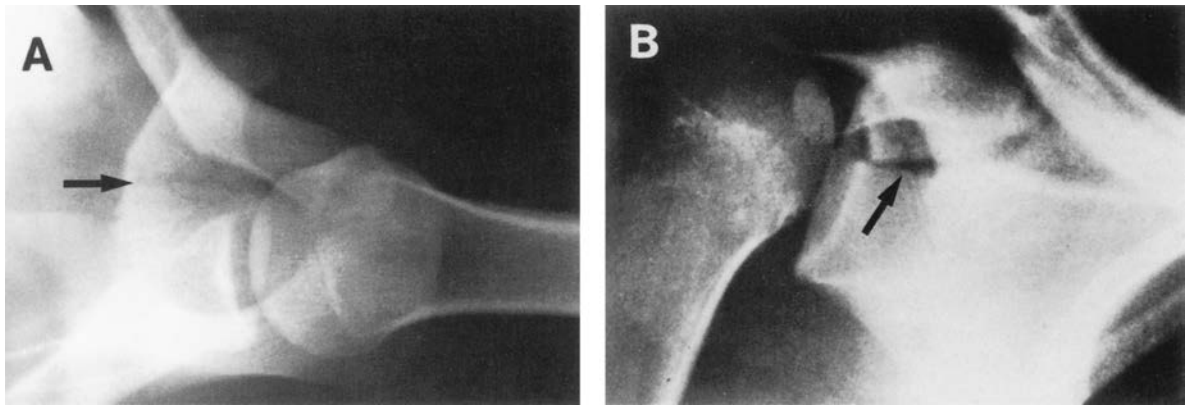


Figure 26-26 Proximal coracoid fractures (two types): **(A)** an axillary radiograph showing an undisplaced fracture of the base of the coracoid process (*arrow*); and **(B)** an anteroposterior radiograph showing a type III fracture of the glenoid cavity (*arrow*) with some medial displacement (from a functional standpoint, this could be considered a very proximal coracoid process fracture).

tive treatment alone cannot restore congruence" and that stiffness and pain may result and "for this reason, open reduction and stabilization are indicated." Kavanagh et al.[94] presented their experience at the Mayo Clinic with 10 displaced intraarticular fractures of the glenoid cavity treated with ORIF. They found ORIF to be "a useful and safe technique" that can "restore excellent function of the shoulder." In their series, the major articular fragments were displaced 4 to 8 mm. They emphasized that it remained uncertain how much incongruity of the glenoid articular surface could be accepted without risking the long-term sequelae of pain, stiffness, and/or traumatic osteoarthritis. Soslowsky et al.[181] found the maximal depth of the glenoid articular cartilage to be 5 mm. Consequently, if displacement at a glenoid fossa fracture site is 5 mm or more, subchondral bone is exposed. Schandelmaier and coauthors reported a series of 22 fractures of the glenoid fossa treated with ORIF. They stated that "if the postoperative courses are uneventful, excellent to good results can be expected."[174] Case reports by Aulicino et al.[8] and Aston and Gregory[7] lend support to the role of surgery in the management of significantly displaced glenoid fossa fractures. Ferraz et al. described a type IV glenoid fossa fracture that progressed to nonunion. When explored surgically, less than

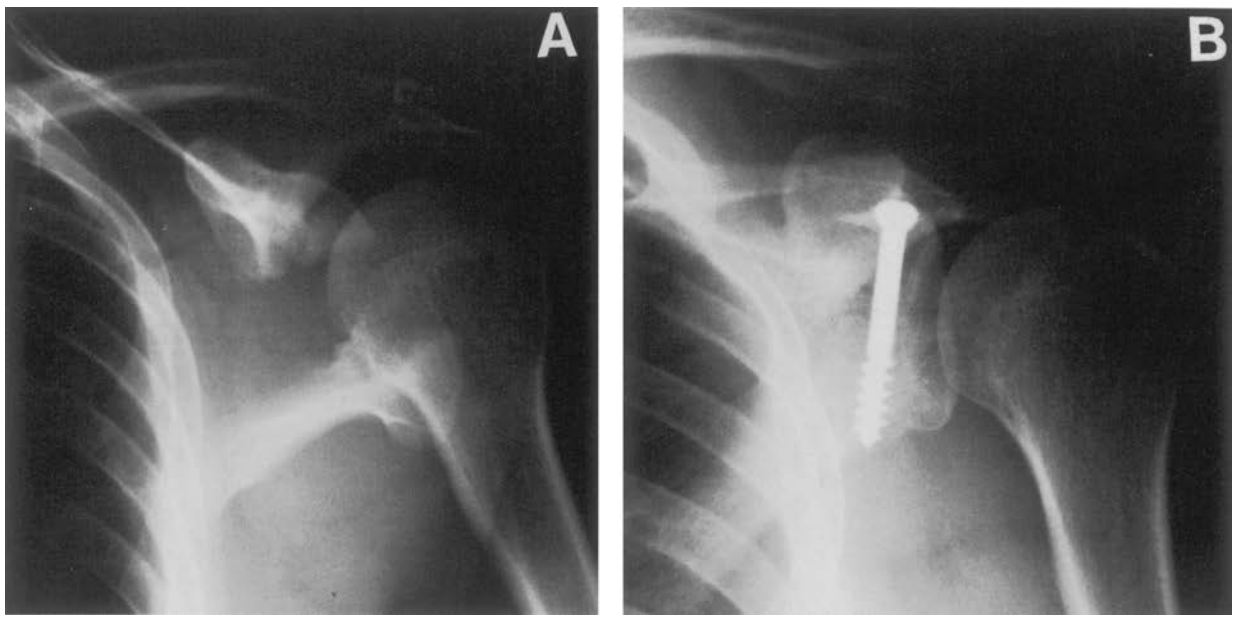


Figure 26-27 Radiographs of a patient who sustained a type IV fracture of the glenoid cavity: **(A)** a preoperative anteroposterior (AP) radiograph showing severe separation of the superior and inferior portions of the glenoid fossa and scapular body; and **(B)** postoperative AP radiograph showing anatomic reduction and stabilization of the superior and inferior portions of the glenoid fossa and scapular body, with restoration of articular congruity.

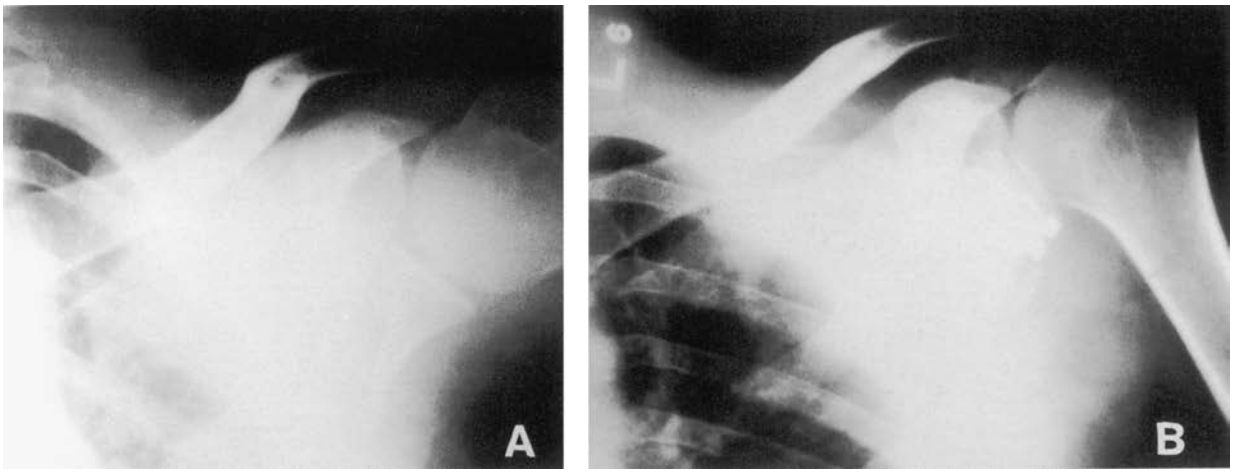


Figure 26-28 Radiographs of a patient who sustained a type II fracture of the glenoid cavity: **(A)** preoperative anteroposterior (AP) radiograph showing significant displacement of the inferior glenoid fragment and a severe articular step-off; and **(B)** postoperative AP radiograph showing anatomic reduction and stabilization of the inferior glenoid fragment, with restoration of articular congruity.

2 years after injury, a 7-mm articular step-off was noted, as well as grade III cartilaginous erosion of the humeral head.[40] Lee and colleagues reported the case of a child who sustained a type II fracture that required ORIF.[111] Ruedi and Chapman[171] stated that "grossly displaced intraarticular fractures of the glenoid that render the joint incongruent and unstable profit from operative reconstruction and internal fixation as incongruities will result in osteoarthritic changes." Rowe[169] has advocated surgical management of severely displaced injuries. Leung et al. reviewed 14 displaced

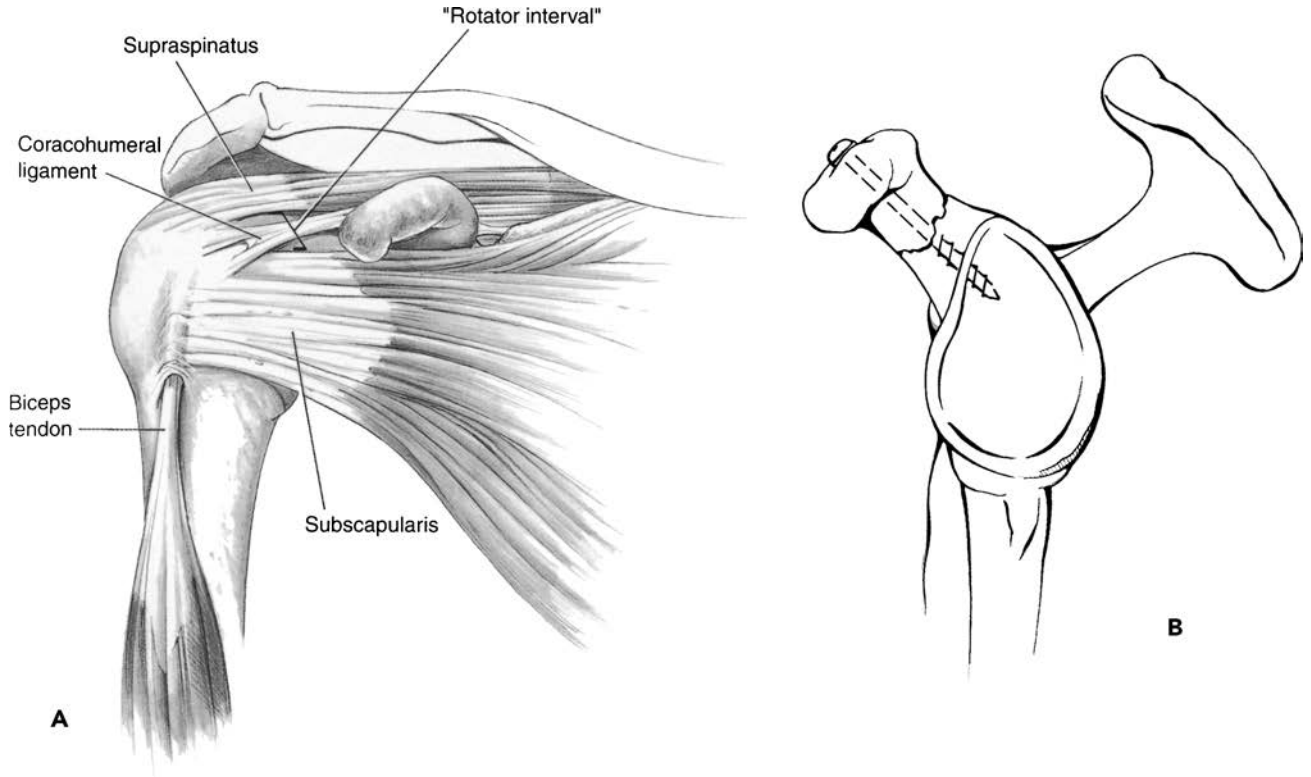


Figure 26-29 Illustrations depicting **(A)** the rotator interval, which can be incised, and the surrounding soft tissues retracted to expose the coracoid process including its junction with the glenoid process; and **(B)** a base of the coracoid process fracture reduced and stabilized with an interfragmentary compression screw.

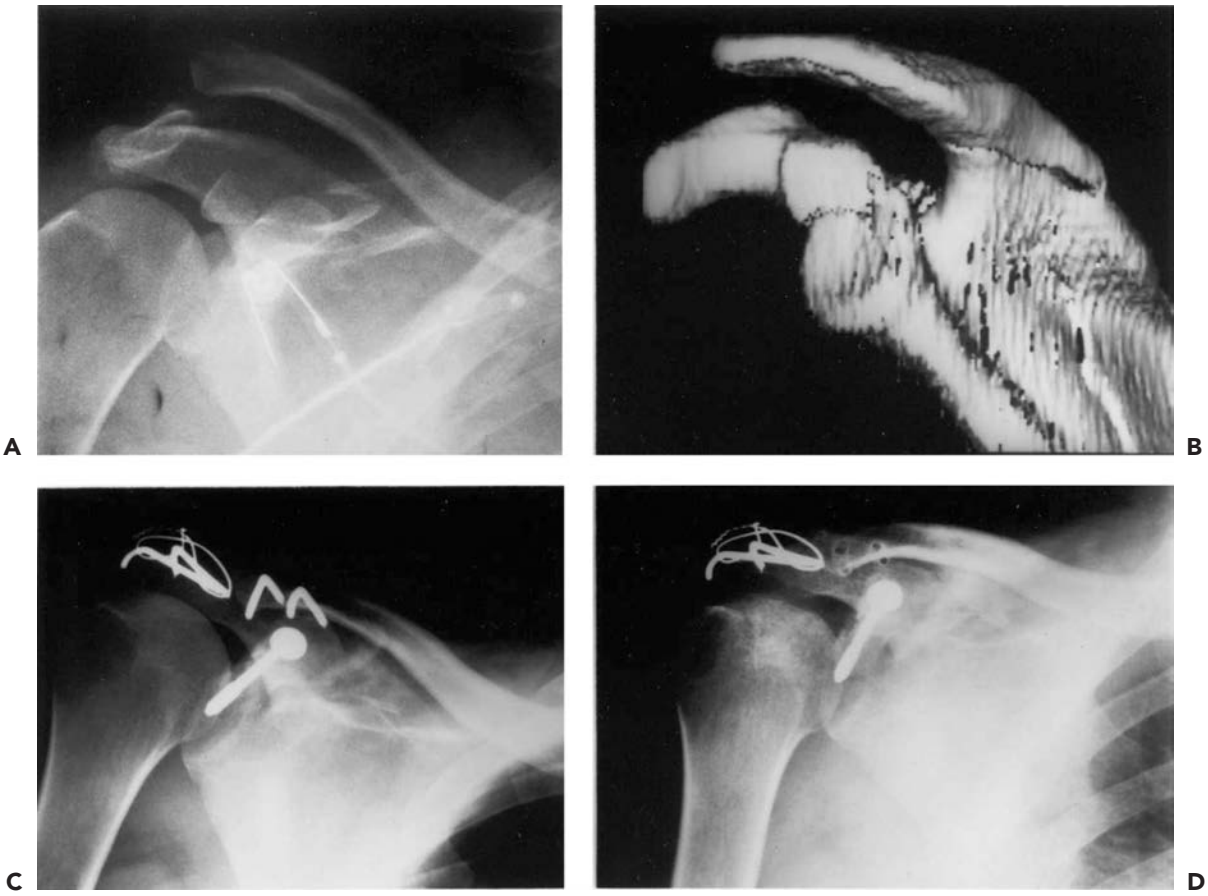


Figure 26-30 An individual who sustained a fracture of his acromion, a type III acromioclavicular (AC) joint disruption, and a type III glenoid cavity fracture: **(A)** preoperative anteroposterior (AP) radiograph; **(B)** preoperative three-dimensional computed tomographic radiograph; **(C)** postoperative AP radiograph showing the acromial fracture reduced and stabilized with a tension band construct, the type III glenoid cavity fracture reduced and stabilized with a compression screw, and the AC joint disruption reduced and stabilized with K-wires passed through the clavicle and into the acromial process; and **(D)** postoperative AP radiograph showing maintenance of the normal clavicular–scapular relationships after removal of the clavicular–acromial K-wires. (From Goss TP. Fractures of the scapula. In: Rockwood CA, Matsen FA, Wirth MA, Lippitt SB, eds. *The shoulder,* 3rd ed. Philadelphia: Saunders, 2004:413, with permission.).

intraarticular fractures of the glenoid treated with ORIF (30.5-month average follow-up) and reported nine excellent and five good results.[114] On the basis of these reports, it seems reasonable to conclude that there is a definite role for surgery in the treatment of glenoid fossa fractures. An injury with an articular step-off of 5 mm or more should be considered for surgical intervention to restore articular congruity, and displacement of 10 mm or more is an absolute indication to avoid posttraumatic osteoarthritis.

Fractures of the Glenoid Neck

Incidence, Mechanism, and Classification

Fractures of the glenoid neck make up 25% of scapular fractures. It is somewhat surprising that these injuries are not more common, since this portion of the glenoid process is quite narrow. Fractures of the glenoid neck may be caused by (a) a direct blow over the anterior or posterior aspect of the shoulder; (b) a fall on an outstretched arm, with impaction of the humeral head against the glenoid process; or in rare cases, (c) a force applied to the superior aspect of the shoulder complex.

The glenoid neck is that portion of the glenoid process that lies between the scapular body and the glenoid cavity. The coracoid process arises from its superior aspect. Its stability is primarily osseous, specifically its junction medially with the scapular body. Secondary support is provided by its attachment superiorly to the clavicular–AC joint–acromial strut via the clavicular–CC ligamentous–coracoid (C-4) linkage (Fig. 26-32) and the coracoacromial (CA) ligament. Tertiary soft tissue support is provided anteriorly by the subscapularis muscle, superiorly by the supraspinatus muscle, and posteriorly by the infraspinatus and teres minor

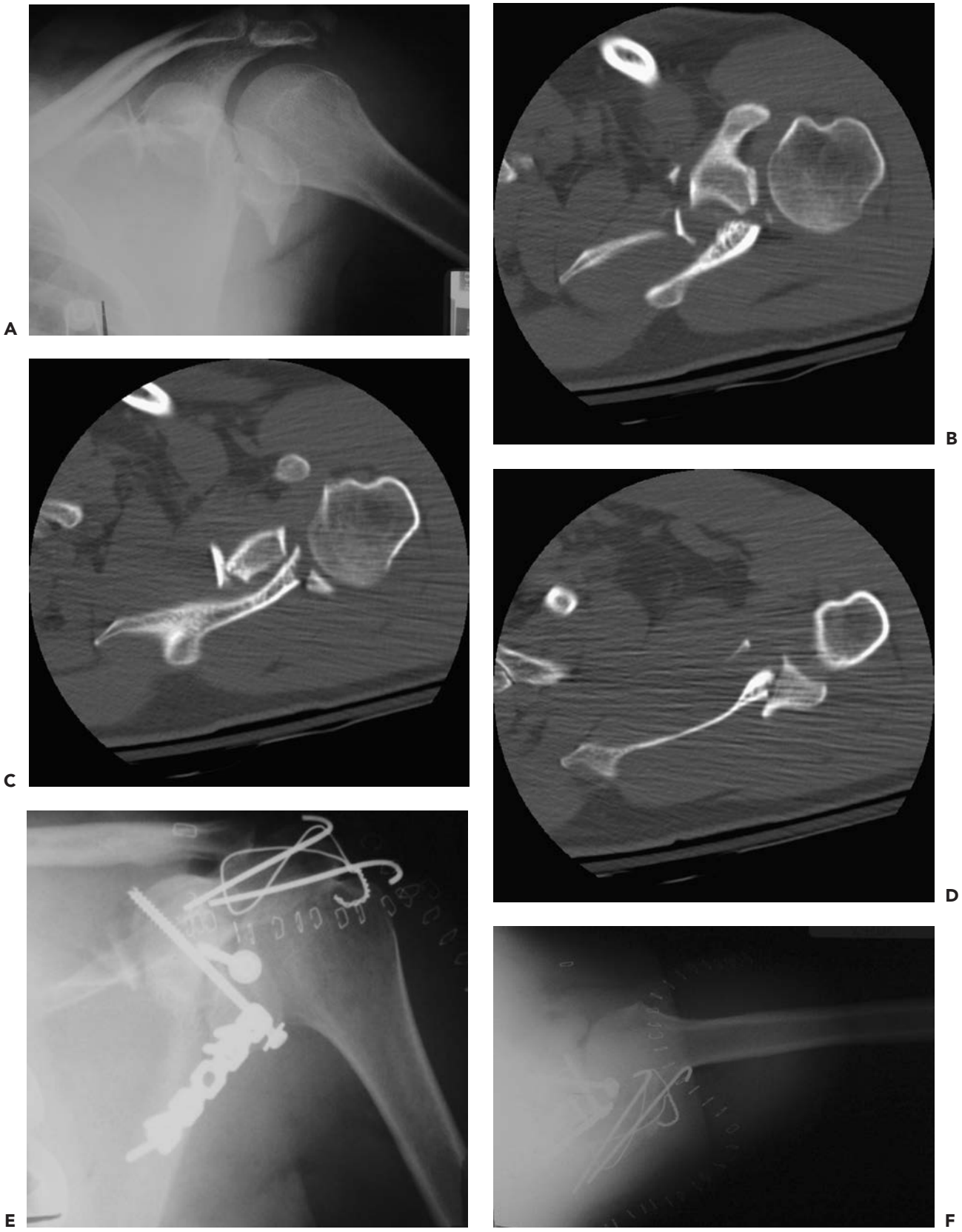


Figure 26-31 Radiographs of a patient involved in a motor vehicle accident who sustained a type Vc fracture of the glenoid cavity: **(A)** anteroposterior (AP) radiograph showing the glenoid cavity fracture; **(B)** axillary computed tomographic (CT) image showing a large anterosuperior glenoid fragment including the coracoid process; **(C)** axillary CT image showing the lateral aspect of the scapular body lying between the two glenoid fragments, abutting the humeral head; **(D)** axillary CT image showing a large posteroinferior cavity fragment; **(E)** postoperative AP and **(F)** axillary radiographs showing the glenoid cavity fragments secured together with cannulated screws and the glenoid unit secured to the scapular body with a malleable reconstruction plate (the acromial fracture was reduced and stabilized with a tension band construct.)

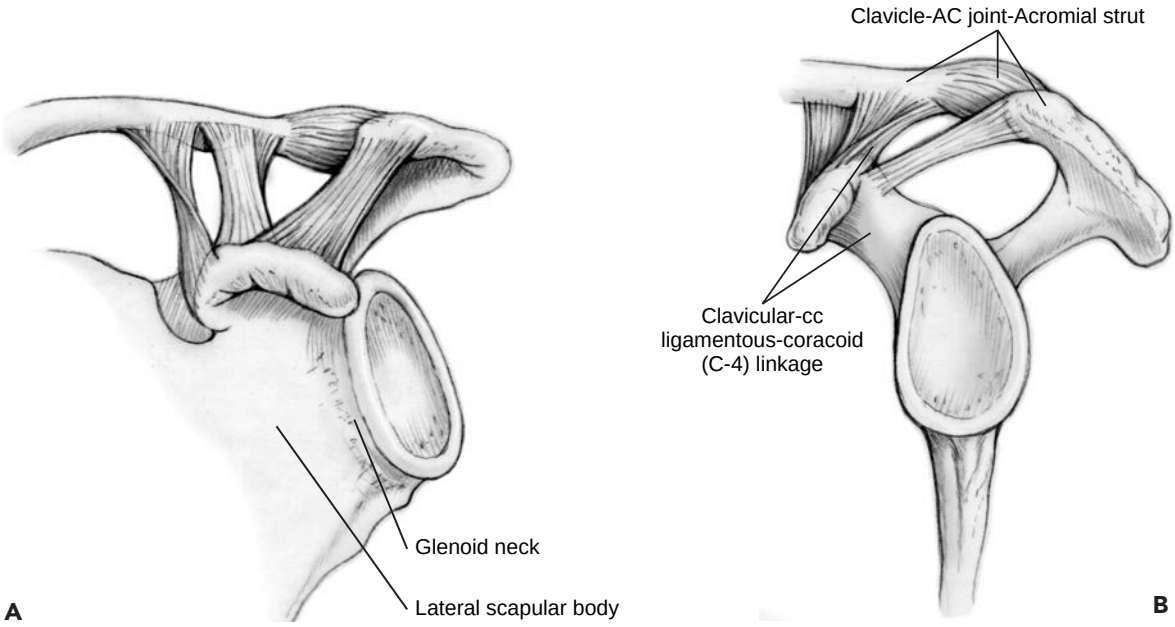


Figure 26-32 Illustrations depicting structures providing stability to the glenoid process in the region of the glenoid neck: **(A)** lateral aspect of the scapular body; and **(B)** the clavicular–acromioclavicular joint–acromial strut via the clavicular– coracoclavicular ligamentous–coracoid (C-4) linkage and the coracoacromial ligament. (Adapted from Goss TP. Fractures of the scapula. In: Rockwood CA, Matsen FA, Wirth MA, Lippitt SB, eds. *The shoulder,* 3rd ed. Philadelphia: Saunders, 2004:413, with permission).

muscles. To be a glenoid neck fracture, the disruption must be complete—exiting along the lateral scapular border and the superior scapular margin, either just lateral or just medial to the coracoid process (Fig. 26-33A,B). Displacement may then occur. If in addition its secondary support is compromised (a coracoid process fracture, a coracoclavicular ligamentous disruption with or without a CA ligamentous injury, a clavicle fracture, an AC joint disruption, or an acromial fracture—that is, a double disruption of the SSSC), there is the potential for severe displacement.[55]

Type I fractures include all insignificantly displaced injuries and constitute 90+% of the total. Management is nonoperative, and a good to excellent functional result can be expected.

Type II fractures include all significantly displaced injuries, and significant displacement is defined as translational displacement of the glenoid fragment greater than or equal to 1 cm or angulatory displacement of the fragment greater than or equal to 40 degrees in either the coronal or the sagittal plane (Fig. 26-34).[60]

One centimeter of translational displacement was chosen by Zdravkovic and Damholt,[207] Nordquvist and Petersson,[148] and Miller and Ada[1,135] as separating major from minor injuries. Bateman[11] believed that this degree of displacement could interfere with abduction. Hardegger et al.[71] pointed out that significant translational displacement changed the normal relations between the glenohumeral articulation and the undersurface of the distal clavicle, AC joint, and acromial process, thereby altering the mechanics

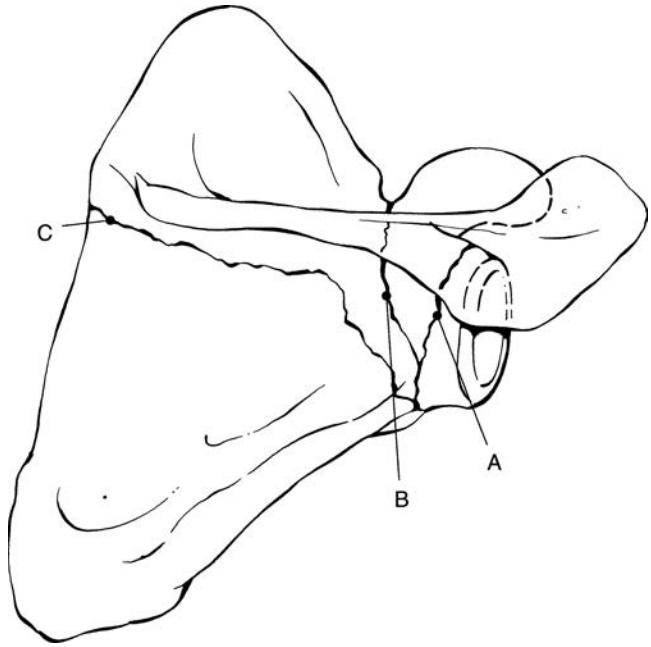


Figure 26-33 Illustration depicting three basic fracture patterns involving the glenoid neck: **(A)** a fracture through the anatomic neck; **(B)** a fracture through the surgical neck; and **(C)** a fracture involving the inferior glenoid neck, which then courses medially to exit through the scapular body (this is managed as a scapular body fracture).

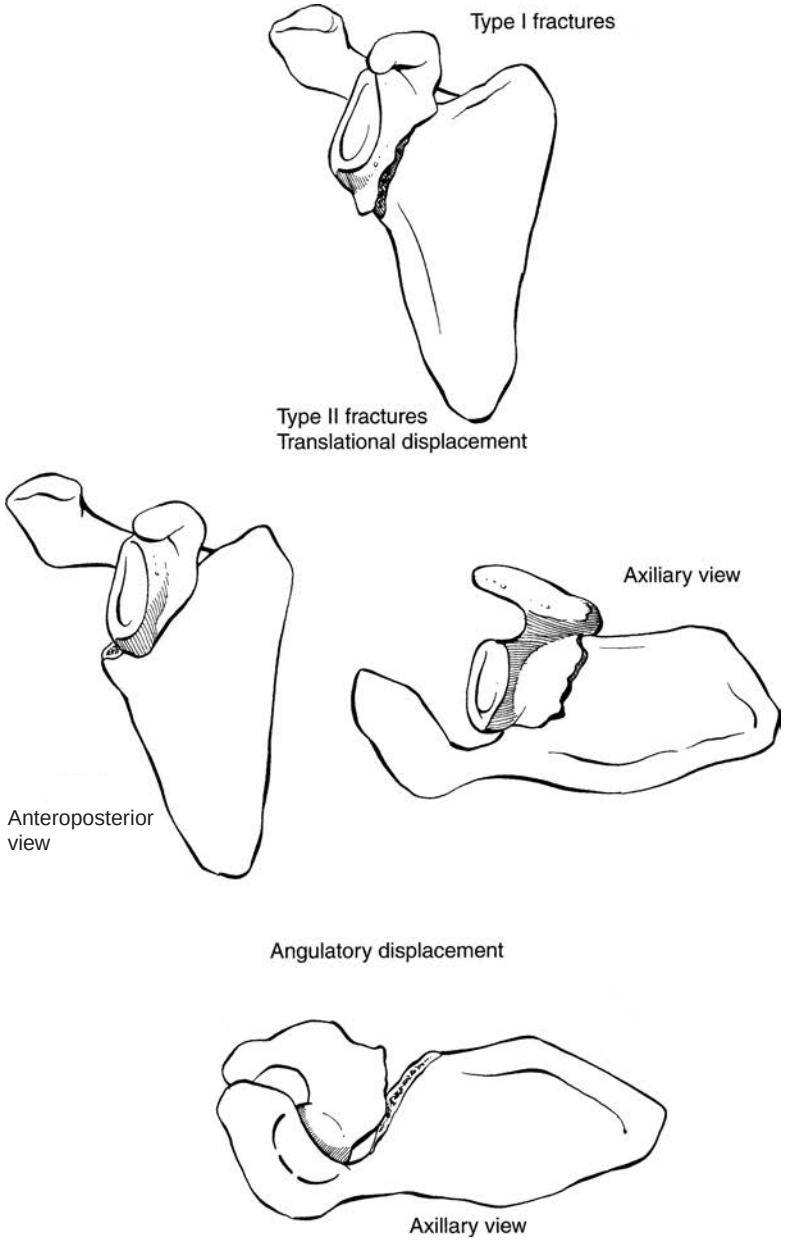


Figure 26-34 Classification scheme for fractures of the glenoid neck.

of nearby musculotendinous units, resulting in a functional imbalance of the shoulder complex as a whole: a "disorganization of the coracoacromial arch." Miller and Ada[1,135] believed that resultant weakness (especially abductor weakness), decreased range of motion, and pain (especially subacromial pain) were largely due to rotator cuff dysfunction. The premise that significant translational displacement of the glenoid process can lead to shoulder discomfort and dysfunction certainly makes sense intuitively—the complex bony relations in the glenohumeral region are clearly altered, as are the mechanics of the musculotendinous structures that pass from the scapula to the proximal humerus (the deltoid muscle and the rotator cuff in particular). The fracture line usually exits the superior scapular border medial to the coracoid process (the surgical neck region). The glenoid fragment is then drawn distally by the weight of the arm and anteromedially by adjacent muscle forces or posteromedially, in which case it is usually forced inferiorly by the scapular spine (Fig. 26-35). Hardegger et al.[71] described a rare case in which the fracture line exited the superior scapular border lateral to the coracoid process (the anatomic neck), allowing the glenoid fragment to be displaced laterally and distally by the pull of the long head of the triceps muscle. Arts and Louette described a similar injury. They believed that such fractures were inherently unstable and in need of ORIF, as opposed to fractures of the surgical neck, which require associated injuries to be rendered unstable and in need of ORIF.[6]

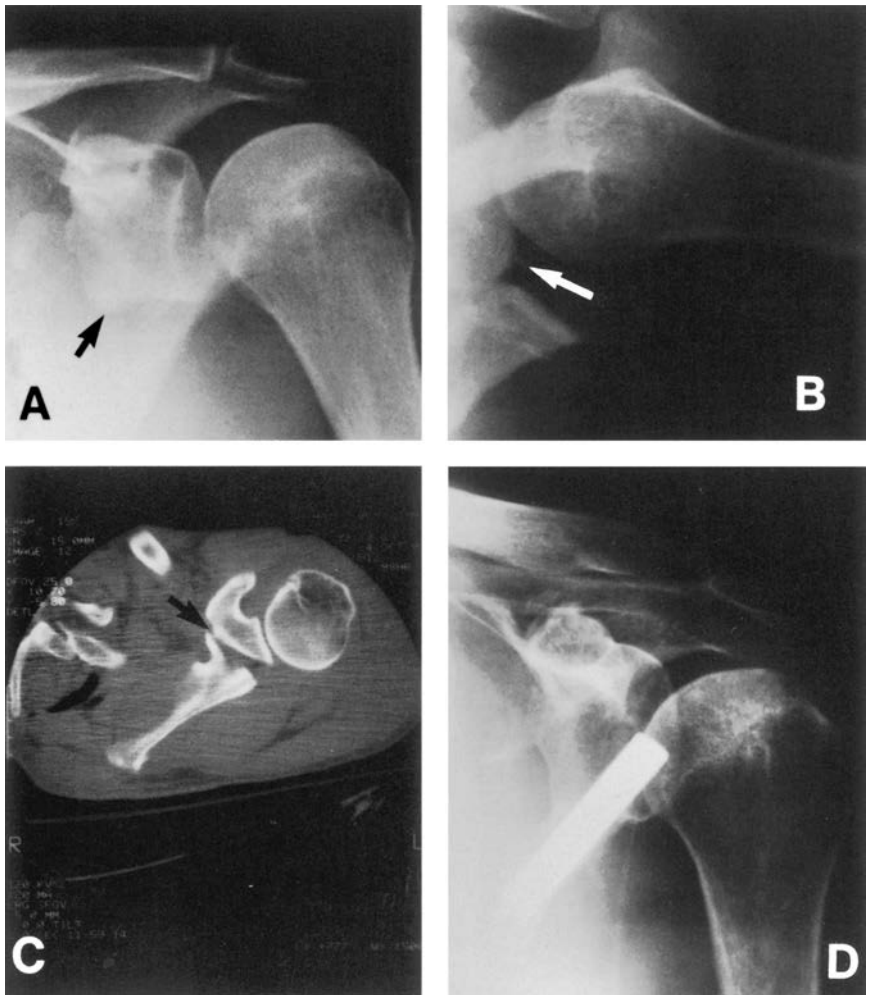


Figure 26-35 Radiographs of a patient who sustained a type II fracture of the glenoid neck with significant translational displacement of the glenoid fragment: **(A,)** a preoperative anteroposterior (AP) radiograph showing the glenoid neck fracture and medial translation of the glenoid fragment; **(B)** a preoperative axillary radiograph revealing that the unstable glenoid fragment is severely displaced anteromedially (the glenohumeral joint is medial to the most lateral aspect of the scapular body), probably owing to an associated disruption of the coracoclavicular ligaments (a violation of the C-4 linkage and, therefore, a double disruption of the superior shoulder suspensory complex) and possibly the coracoacromial ligament; **(C)** a preoperative axial computed tomographic image revealing the glenoid neck fracture to be complete, exiting the superior scapular border medial to the intact coracoid process (a surgical neck fracture); and **(D)** a postoperative AP radiograph showing anatomic reduction and stabilization of the glenoid neck fracture and glenoid fragment.

Bateman[11] and DePalma[32] believed that excessive angulation of the glenoid fragment could result in glenohumeral instability (anterior, posterior, or inferior). Normally, the glenoid cavity faces 15 degrees superiorly and is retroverted 6 degrees relative to the plane of the scapular body. With increasing angulation, the humeral head loses the normal bony support provided by the glenoid cavity (bony instability), which translates into glenohumeral discomfort and dysfunction.[1,11,32,135] Miller and Ada[1,135] felt that angular displacement greater than or equal to 40 degrees in either the coronal or transverse plane was unacceptable (Fig. 26-36). They felt that this degree of displacement adversely altered not only glenohumeral but also other bony relations as well as musculotendinous dynamics, particularly those of the rotator cuff, resulting in pain and overall shoulder dysfunction (diminished range of motion and loss of strength).

Miller and Ada[1,135] retrospectively reviewed 16 displaced glenoid neck fractures (greater than or equal to 1 cm of translational displacement or greater than or equal to 40 degrees of angulation in either the transverse or coronal plane) managed nonoperatively (36-month average follow-up). They found that 20% had decreased range of motion, 50% had pain (75% night pain), 40% had weakness with exertion, and 25% noted "popping." In particular, these patients frequently had shoulder abductor weakness and subacromial pain, due at least in part to rotator cuff dysfunction. They recommended ORIF of glenoid neck fractures with this degree of displacement.

Diagnosis

Diagnosis is ultimately radiographic. Plain radiographs are helpful, but because of the complex bony anatomy in the area, CT scanning is generally necessary to determine whether a glenoid neck fracture is indeed complete, to determine the degree of displacement, if any, and to identify injuries to adjacent bony structures and articulations. One must not confuse these injuries with the more common fractures that course through the inferior glenoid neck and the scapular body (see Fig. 26-33C). CT scanning readily reveals that the latter are not complete disruptions of the glenoid process, since the superior aspect of the glenoid neck is intact. These are essentially fractures of the scapular body and do quite well with nonoperative care

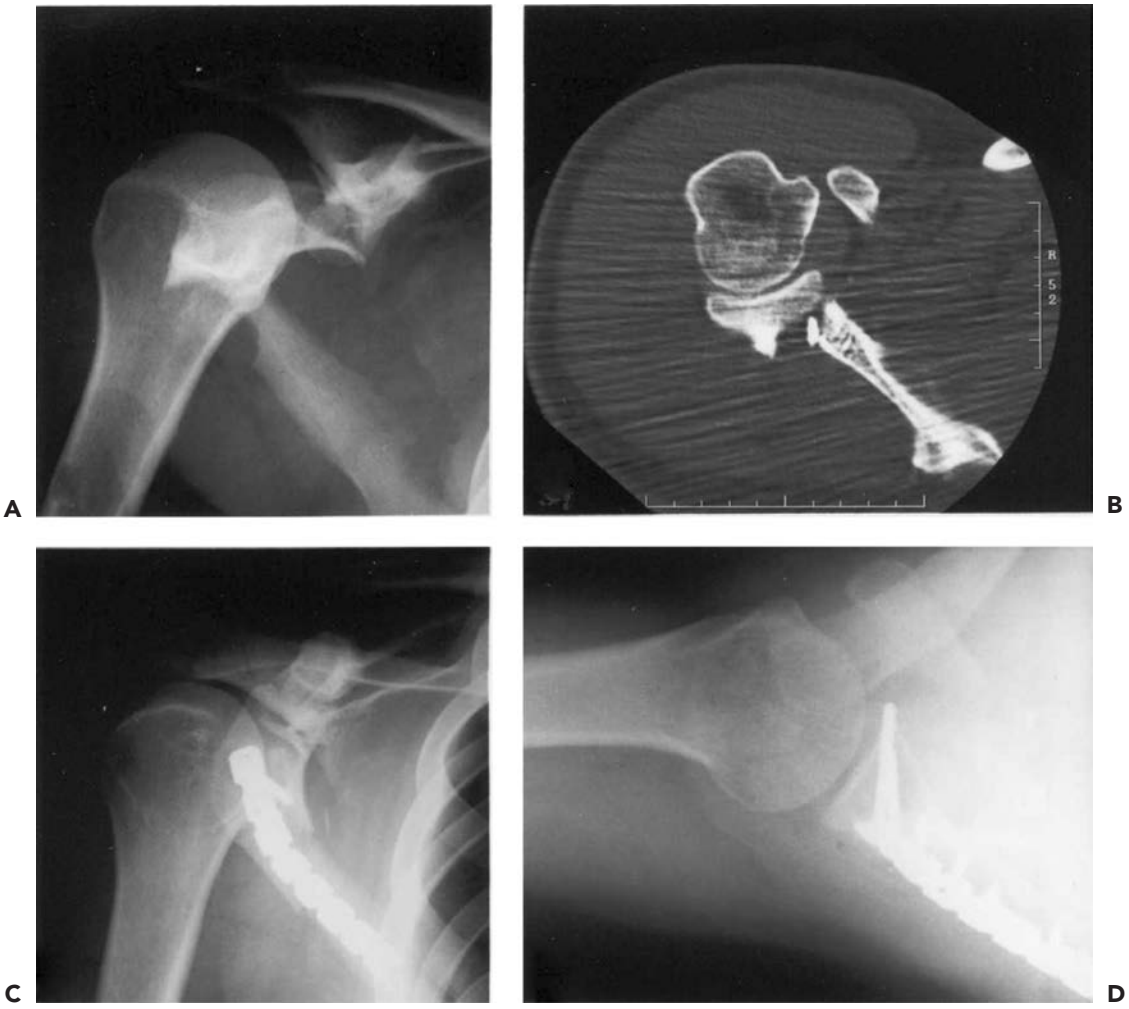


Figure 26-36 A patient who sustained a type II fracture of the glenoid neck with significant angulatory displacement of the glenoid fragment: **(A)** a preoperative anteroposterior (AP) radiograph showing the glenoid neck fracture with severe angulation of the glenoid fragment and a fractured coracoid process—also note the fracture of the scapular body with displacement of the lateral scapular border; **(B)** a preoperative axillary computed tomography projection showing the coracoid process fracture (a violation of the C-4 linkage) that further destabilized the glenoid fragment and allowed severe angulatory displacement to occur (a double disruption of the superior shoulder suspensory complex); **(C)** and **(D)** postoperative AP and axillary radiographs showing the glenoid fragment reduced and stabilized with a contoured reconstruction plate (the coracoid process fracture was allowed to heal spontaneously). (From Goss TP. Fractures of the scapula. In: Rockwood CA, Matsen FA, Wirth MA, Lippitt SB, eds. *The shoulder,* 3rd ed. Philadelphia: Saunders, 2004:413, with permission.)

because the normal relations between the glenohumeral articulation and the distal clavicle–acromion are unaltered (Fig. 26-37).[60] McAdams et al.[124] found that CT scanning did not improve the evaluation of glenoid fractures over plain films but did aid in the identification of associated injuries to the superior shoulder suspensory complex.

Nonoperative Versus Operative Treatment

Imatani,[88] McGahan et al.,[127] and Lindholm and Leven[116] recommended nonsurgical treatment for all glenoid neck fractures, but their studies give few details to justify this conclusion. A long-term follow-up study by Zdravkovic and Damholt[207] included 20 to 30 patients (it is difficult to determine the exact number from the text) with displaced glenoid neck fractures and noted that nonoperative treatment yielded satisfactory results. Nordquvist and Petersson,[148] however, evaluated 37 glenoid neck fractures treated without surgery (10- to 20-year follow-up) and found the functional result to be either fair or poor in 32%. They believed that for some fractures, early ORIF might have improved the result. Although somewhat ambivalent in their recommendations, three studies do mention surgical management as an option in selected cases. Armstrong and Vanderspuy[5] believed that although most of these individuals do well, more aggressive treat-

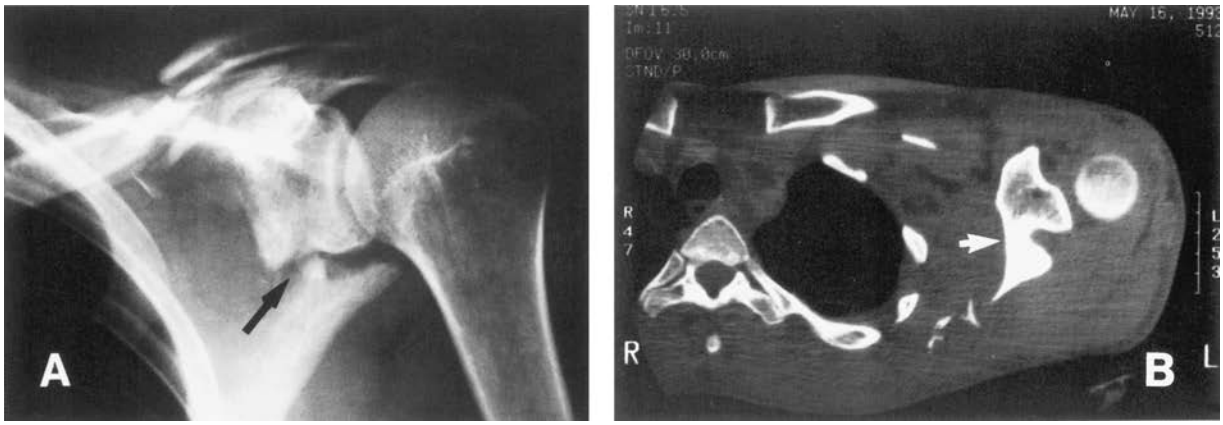


Figure 26-37 Radiographs showing what initially, but erroneously, appeared to be a complete fracture of the glenoid neck: **(A)** An anteroposterior radiograph of the shoulder showing a fracture involving the inferior aspect of the glenoid neck; and **(B)** an axial computed tomographic image showing the superior portion of the glenoid neck to be uninvolved (the fracture exited through the scapular body).

ment including ORIF may be indicated in patients who are young and fit. DeBeer et al.[29] described the operative treatment of a professional cyclist who was able to return to competition at 6 weeks and urged more aggressive treatment in high-demand patients. Wilbur and Evans[199] believed that ORIF might be indicated if the glenoid fragment is markedly displaced or angulated, but did not believe that they had enough information or experience to warrant definitive surgical indications.

Hardegger et al.[71] reported 79% good to excellent results in five displaced glenoid neck fractures treated surgically (6.5-year follow-up). They agreed with Judet,[91] Magerl,[119] Ganz and Noesberger,[48] and Tscherne and Christ[190] that operative management of displaced glenoid neck fractures avoids late disability and yields better results. Gagney et al.[47] found a good result in only 1 of 12 displaced fractures treated nonoperatively. They believed that such injuries "could disorganize the coracoacromial arch," and recommended open reduction. Neer,[142] as well as Rockwood[143] and Butters,[23] presented the recommendations of other investigators, as did a review article by Guttentag and Rechtine.[68] Boerger and Limb reported the case of a patient with a fracture of the glenoid neck and acromial and coracoid processes, a dislocated AC joint, and incomplete paralysis of the infraspinatus muscle treated by ORIF of the acromial and glenoid neck fractures.[19] Clearly, all these investigators agree that the vast majority of glenoid neck fractures can and should be treated without surgery. However, most authors believe that more aggressive treatment, including ORIF, is at the very least a consideration, if not clearly indicated, when the glenoid fragment is severely displaced (i.e., type II injuries). Some might argue with 1 cm of translational displacement being an indication for surgery. However, the decision to proceed becomes easier with increasing degrees of displacement, especially if the glenoid rim lies medial to the lateral margin of the scapular body (see Fig. 26-35). Forty degrees (or certainly more) of angular displacement of the glenoid fragment also seems to be a reasonable indication for operative management.

Surgical Indications and Management

Type II fractures should at the very least be considered for surgical management.

The glenoid process and fracture site are approached posteriorly. The interval between the infraspinatus and teres minor is developed to expose the posteroinferior glenoid neck and lateral scapular border. A superior approach can be added to gain control over the free glenoid fragment. Once a satisfactory reduction has been achieved, temporary fixation can be provided by placing K-wires between the glenoid fragment and the adjacent bony structures (e.g., through the glenoid fragment and into the scapular body; through the acromial process and into the glenoid fragment; and so on). Firm fixation is generally achieved by means of a contoured 3.5-mm malleable reconstruction plate applied along the lateral border of the scapula and the posterior aspect of the glenoid process. Supplemental fixation can be provided by K-wires or lag screws. K-wires providing temporary fixation can be retained or used for the placement of 3.5-mm cannulated lag screws (Fig. 26-38). Conceivably, comminution of the scapular body and spine may be so severe or the size of the glenoid fragment so small as to preclude plate fixation. In these cases, K-wire or lag screw fixation of the reduced glenoid fragment to adjacent intact bony structures (i.e., the acromial process, the distal clavicle, etc.) may be all that can be provided. If a disruption of the clavicular–AC joint–acromial strut is also present, fixation of that injury may indirectly reduce and stabilize the glenoid neck fracture. If significant displacement persists, however, the glenoid neck fracture must also be addressed.[60] Conversely,

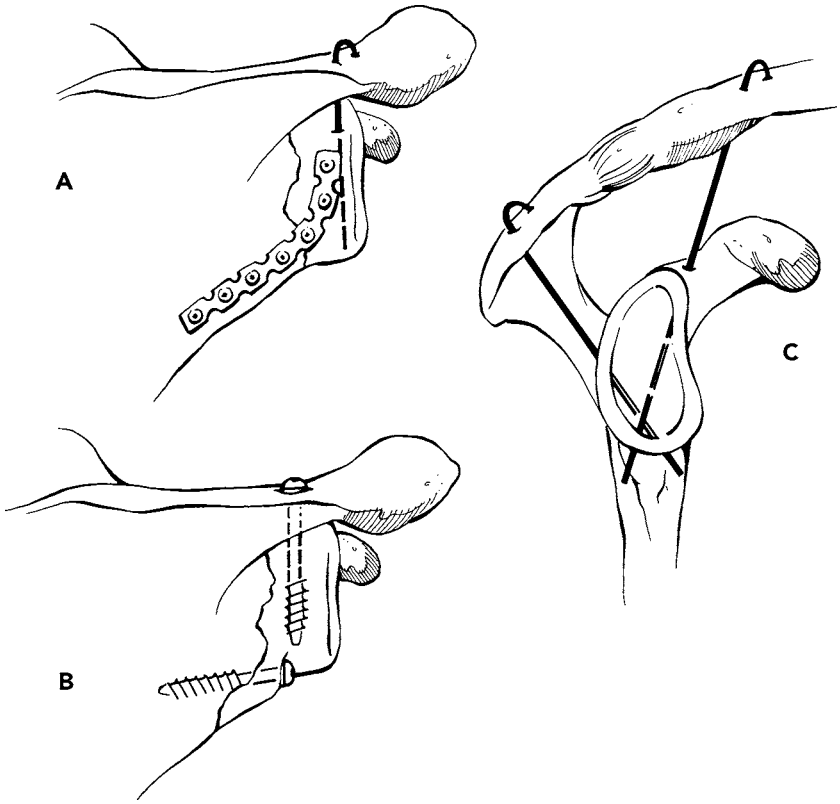


Figure 26-38 Illustrations depicting fixation techniques available for stabilization of glenoid neck fractures: **(A)** stabilization with a 3.5-mm malleable reconstruction plate (note the K-wire running from the acromial process to the glenoid process that can be used for either temporary or permanent fixation); **(B)** stabilization with 3.5-mm cannulated interfragmentary screws; and **(C)** stabilization with K-wires (in this case, K-wires passed from the acromion and clavicle into the glenoid process).

open reduction and internal fixation of the glenoid neck fracture may satisfactorily reduce and stabilize the second disruption. If not, that associated disruption must be addressed.[60] A fracture of the ipsilateral glenoid neck and midshaft clavicle has been termed a "floating shoulder." Disruptions of the clavicular–CC ligamentous–coracoid (C-4) linkage are usually managed indirectly by reducing and stabilizing the glenoid neck fracture and any injuries compromising the integrity of the clavicular–AC joint–acromial strut. Rarely, if the scapular body–spine, acromial process, and distal clavicle are all severely comminuted, overhead olecranon pin traction must be considered or displacement of the glenoid neck fracture must be accepted and managed nonoperatively.

Postoperative Management and Prognosis

The postoperative management of glenoid process fractures depends upon the fixation–stability achieved. Immobilization in a sling and swathe bandage is prescribed during the first 24 to 48 hours following surgery. If fixation is rigid, dependent circular and pendulum movements are then initiated as well as external rotation of the shoulder to, but not past, neutral. During postoperative weeks 3 to 6, progressive range-of-motion exercises in all directions (especially forward flexion, internal rotation up the back, and external rotation) are prescribed, seeking to achieve full range of motion by the end of the 6-week period. The patient is allowed to use his or her arm actively in a progressive manner within clearly defined limits (moving the weight of his or her upper extremity alone when sitting in a protected setting during weeks 3 and 4 and when up and about indoors during weeks 5 and 6). The patient is followed clinically and radiographically every 2 weeks to make sure displacement does not occur at the fracture site and to monitor and update the rehabilitation program. At 6 weeks, healing is sufficient so as to discontinue all external protection and encourage progressive functional use of the extremity. K-wires spanning bones that move relative to each other are removed at this time, as are those passing through soft tissues (K-wires imbedded within a single osseous structure [bent at their entry site to prevent migration] may be left in place). Physiotherapy continues to focus on regaining range of motion as progressive strengthening exercises are added. The patient's rehabilitation program continues until range of motion, strength, and overall function are maximized. Light use of the shoulder is emphasized through postoperative week 12, whereas heavy physical use of the shoulder, including athletic activities, is prohibited until the 4- to 6-month point.

If surgical fixation is less than rigid, the shoulder may need to be protected in a sling and swathe immobilizer, an abduction brace, or even overhead olecranon pin traction for 7, 10, or 14 days (dependent on the clinical situation) before the aforementioned physiotherapy program is prescribed. The patient must be encouraged to work diligently

on his or her rehabilitation program because range of motion and strength can improve, and the end result is often not achieved for approximately 6 months to 1 year after injury. Hard work, perseverance, and dedication on the part of the patient, the physician, and the physical therapist are critical to an optimal functional result.

Although the literature remains somewhat deficient owing to the rarity of these injuries, interest has increased in recent years, resulting in a growing number of case reports and personal series. Although more data are needed, it is reasonable to anticipate a good to excellent functional result if: (a) surgical management restores normal or near-normal glenoid anatomy and articular congruity, as well as glenohumeral stability; (b) the fixation is secure; and (c) a well-structured, closely monitored postoperative rehabilitation program is prescribed.

DOUBLE DISRUPTIONS OF THE SUPERIOR SHOULDER SUSPENSORY COMPLEX

Biomechanics

The SSSC is a bony soft tissue ring at the end of a superior and an inferior bony strut (Fig. 26-39). This ring is composed of the glenoid process, the coracoid process, the coracoclavicular ligament, the distal clavicle, the AC joint, and the acromial process. The superior strut is the middle third of the clavicle, whereas the inferior strut is the junction of the most lateral portion of the scapular body and the most medial portion of the glenoid neck. The complex can be subdivided into three units: (a) the clavicular–AC joint–acromial strut; (b) the three-process–scapular body junction; and (c) the clavicular–CC ligamentous–coracoid (C-4) linkage (Fig. 26-40), with secondary support provided by the CA ligament. The SSSC is an extremely important structure as regards the biomechanics of the shoulder complex: (a) each of its components has its own individual function(s); (b) it serves as a point of attachment for a variety of musculotendinous and ligamentous structures; (c) it allows limited, but very important motion to occur through the coracoclavicular ligament and the AC articulation; and (d) it maintains a normal, stable relationship between the upper extremity and the axial skeleton (the clavicle is the only bony connection between the upper extremity and the axial skeleton and the scapula is "hung" or suspended from the clavicle by the coracoclavicular ligaments and the acromioclavicular articulation).

The "double disruption" concept is a principle that underlies and allows one to understand a variety of difficult-to-treat injuries to the shoulder complex, which have previously been described in isolation, but are actually united by a single biomechanical theme. This "double disruption" concept also has a predictive value for certain injuries that are encountered only rarely.[55]

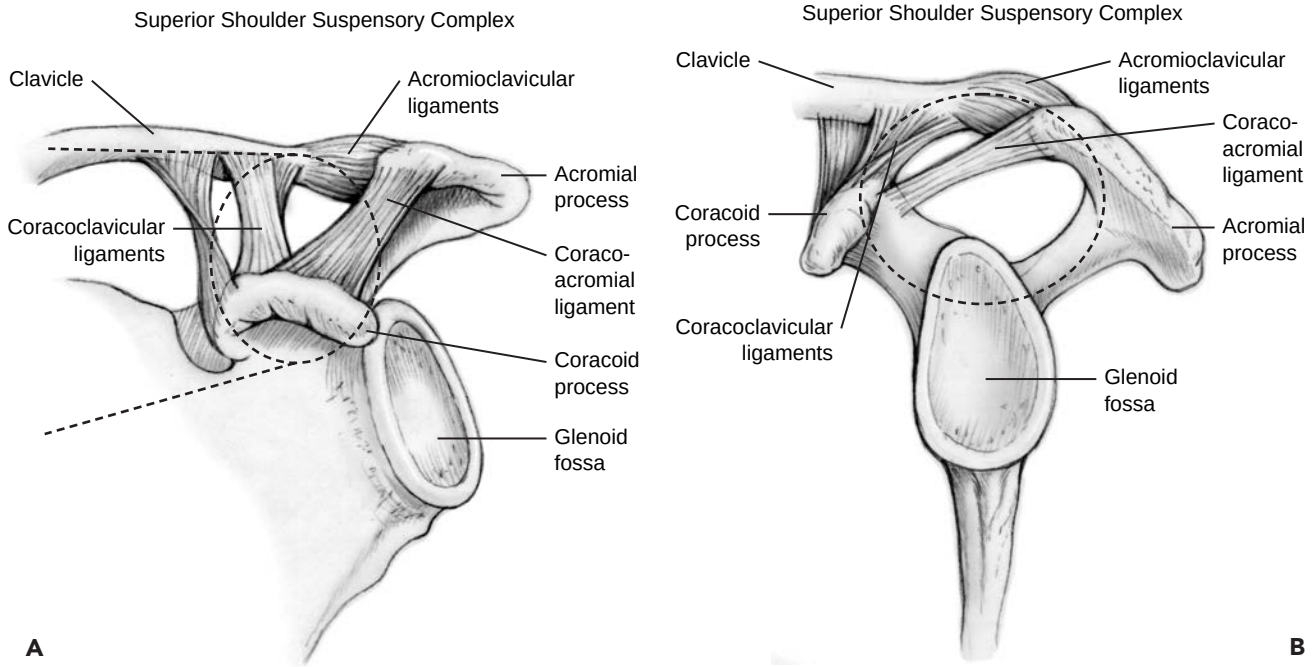


Figure 26-39 Illustrations depicting the superior shoulder suspensory complex: **(A)** an anteroposterior view of the bony–soft tissue ring and the superior and inferior bony struts and **(B)** a lateral view of the bony–soft tissue ring. (Adapted from Goss TP. Fractures of the scapula. In: Rockwood CA, Matsen FA, Wirth MA, Lippitt SB, eds. *The shoulder,* 3rd ed. Philadelphia: Saunders, 2004:413, with permission.)

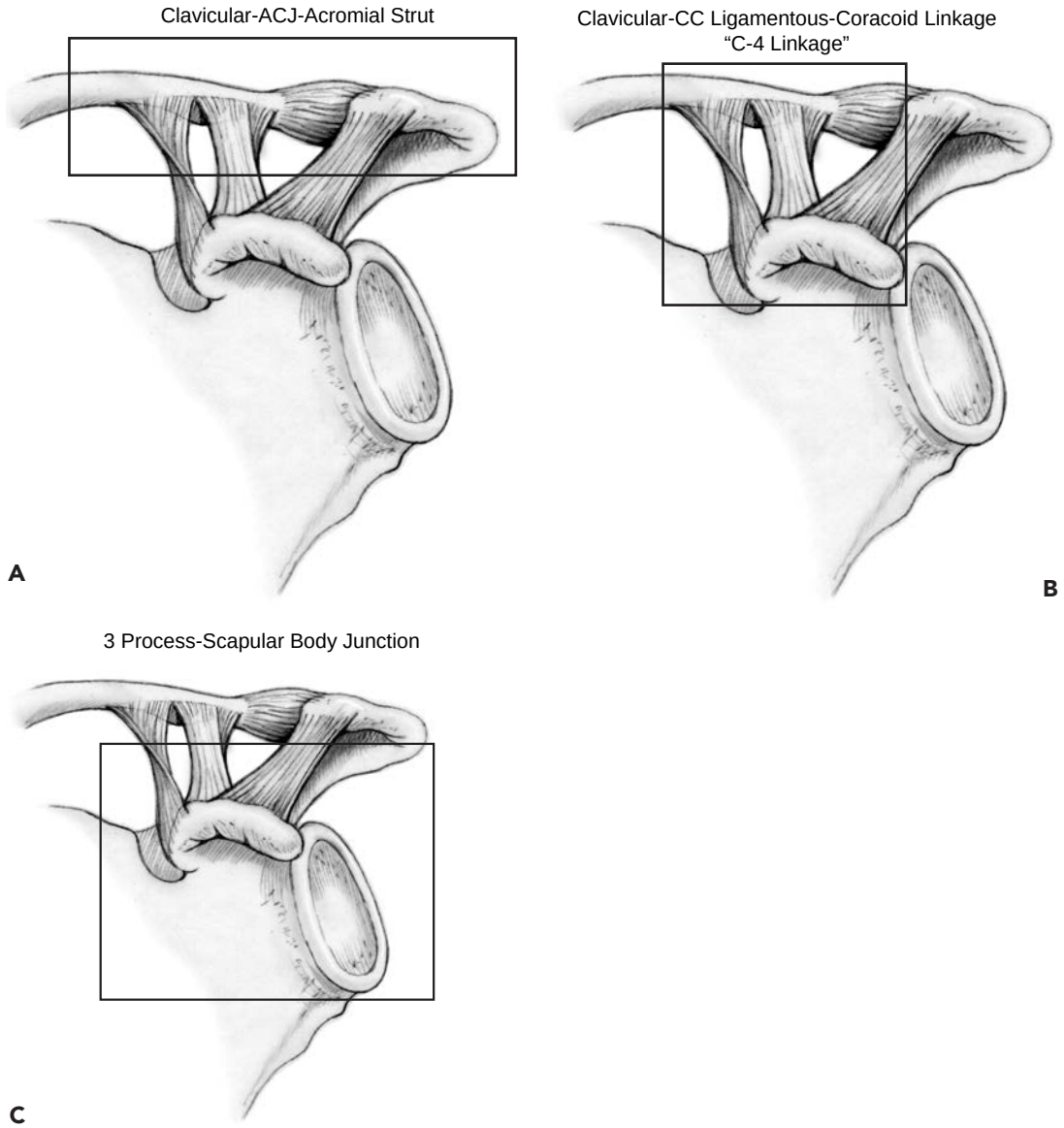


Figure 26-40 The three components of the superior shoulder suspensory complex: **(A)** the clavicular–acromioclavicular joint–acromial strut; **(B)** the clavicular– coracoclavicular ligamentous–coracoid (C-4) linkage; and **(C)** the three process–scapular body junction. (Adapted from Goss TP. Fractures of the scapula. In: Rockwood CA, Matsen FA, Wirth MA, Lippitt SB, eds. *The shoulder,* 3rd ed. Philadelphia: Saunders, 2004:413, with permission.)

Single traumatic disruptions of the SSSC are common (e.g., a type I fracture of the distal clavicle). These are anatomically stable situations because the overall integrity of the complex is not significantly violated and nonoperative management will generally yield a good to excellent functional result.

When the complex is disrupted in two places, however (a "double disruption"), the integrity of the SSSC is clearly compromised, creating a potentially unstable anatomic situation. Significant displacement can occur at either or both sites, resulting in bony healing problems (delayed unions, malunions, and nonunions) as well as adverse long-term functional difficulties (subacromial impingement, decreased strength and muscle fatigue discomfort, neurovascular compromise due to a "drooping" shoulder, and degenerative joint disease) depending on the particular injury.

Double disruptions may take a variety of forms: two fractures of the bony soft tissue ring; two ligamentous disruptions of the ring; a fracture and a ligamentous disruption of the ring; fractures of both bony struts; or a fracture of one strut combined with a ring disruption (either a fracture or a ligamentous disruption; Fig. 26-41). Because the glenoid, acromial, and coracoid processes are all components of the SSSC, many double-disruption injuries involve the scapula. Also, many, if not most, significantly displaced coracoid and acromial fractures are part of a double

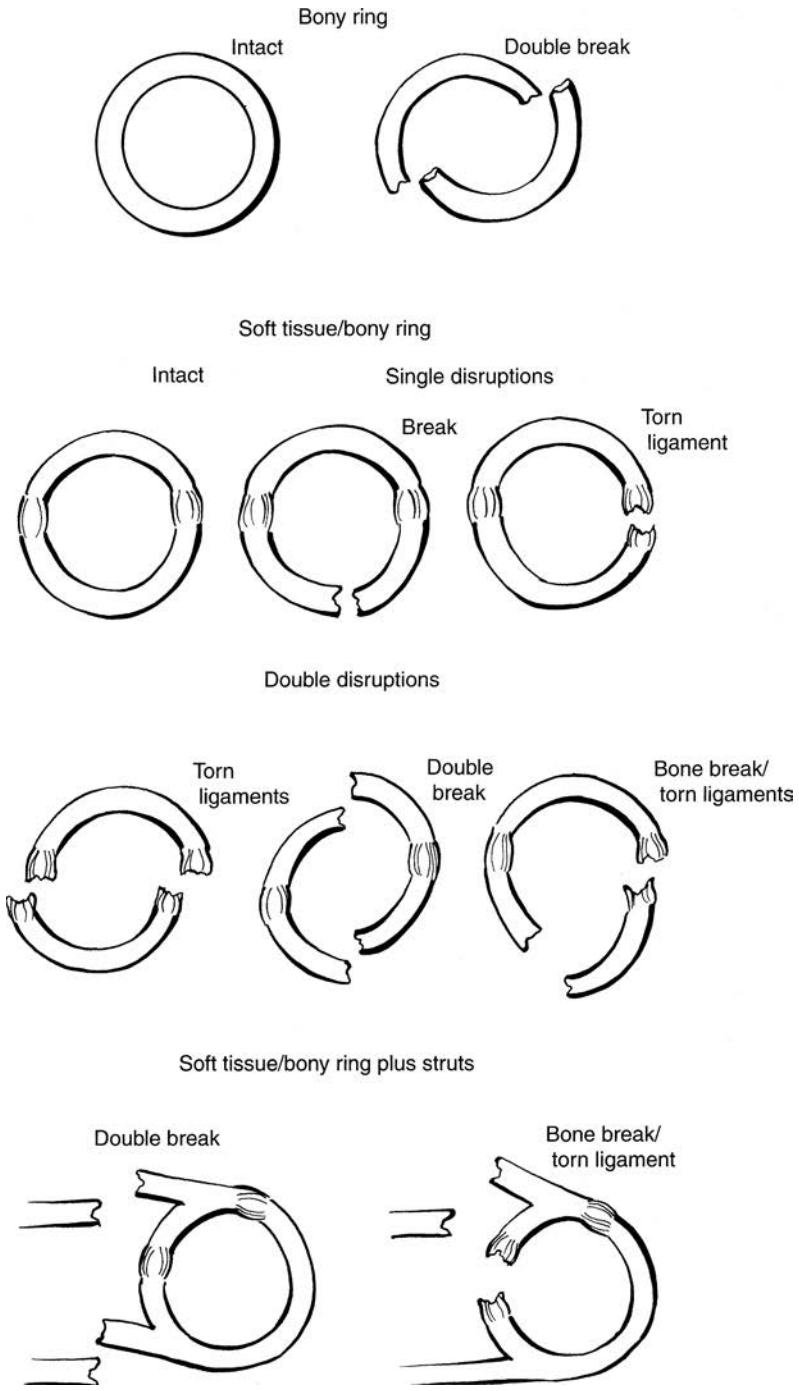


Figure 26-41 Illustrations depicting the many possible traumatic ring–strut disruptions.

disruption. When particularly severe forces are involved, there is the potential for complex injury patterns due to multiple ring and strut disruptions.[20]

Diagnosis

If a single disruption is noted on routine radiographs (a true AP view of the shoulder, a true axillary view of the glenohumeral joint, and a weight-bearing AP of the shoulder to evaluate the integrity of the clavicular–scapular linkage), one should look carefully for a second disruption (CT scanning is often necessary owing to the complex bony anatomy in the area).

Surgical Indications, Management Principles, and Results

If two disruptions are present, one must decide whether displacement at one or both sites is "unacceptable" (a relative term, dependent on the particular clinical situation) and if so, surgical management is generally necessary. Reducing and stabilizing one of the disruptions will frequently

indirectly reduce and stabilize the other disruption satisfactorily (whichever injury is easiest to manage is chosen). If unsuccessful, both disruptions may need to be addressed (see sections dealing with specific injuries). Results, as always, are dependent on the adequacy of the reduction, the quality of the fixation, and the rigor of the postoperative rehabilitation program.

The Floating Shoulder (Ipsilateral Fractures of the Midshaft Clavicle and Glenoid Neck)

Surgical Indications, Techniques, and Results

These injuries represent a double disruption of the SSSC. In isolation, each fracture is generally minimally displaced and managed nonoperatively. In combination, however, each disruption can make the other unstable (i.e., the glenoid neck fracture often allows severe displacement to occur at the clavicular fracture site and vice versa, although to a lesser degree). The situation is rendered even more unstable if an additional disruption of the clavicular–AC joint–acromial strut is present or if the C-4 linkage is violated (Fig. 26-42). Hardegger et al.[71] felt these injuries represented a "functional imbalance" owing to the "altered glenohumeral–acromial relationships." They and Butters[23] recommended surgery to reduce and stabilize the injury. Surgical reduction and stabilization of the clavicular fracture site (most commonly with plate fixation) is advisable if displacement is unacceptable to avoid a nonunion, alleviate tensile forces on the brachial plexus, restore normal anatomic relationships, and ensure restoration of normal shoulder function.[79,104] The glenoid neck fracture will generally reduce and stabilize secondarily; however, if significant displacement persists, it may also require surgical management[78,112] (see Fractures of the Glenoid Neck). Additional injuries to the clavicular–AC joint–acromial strut may require operative treatment, whereas associated injuries of the C-4 linkage will usually heal satisfactorily if the glenoid neck and clavicular fracture

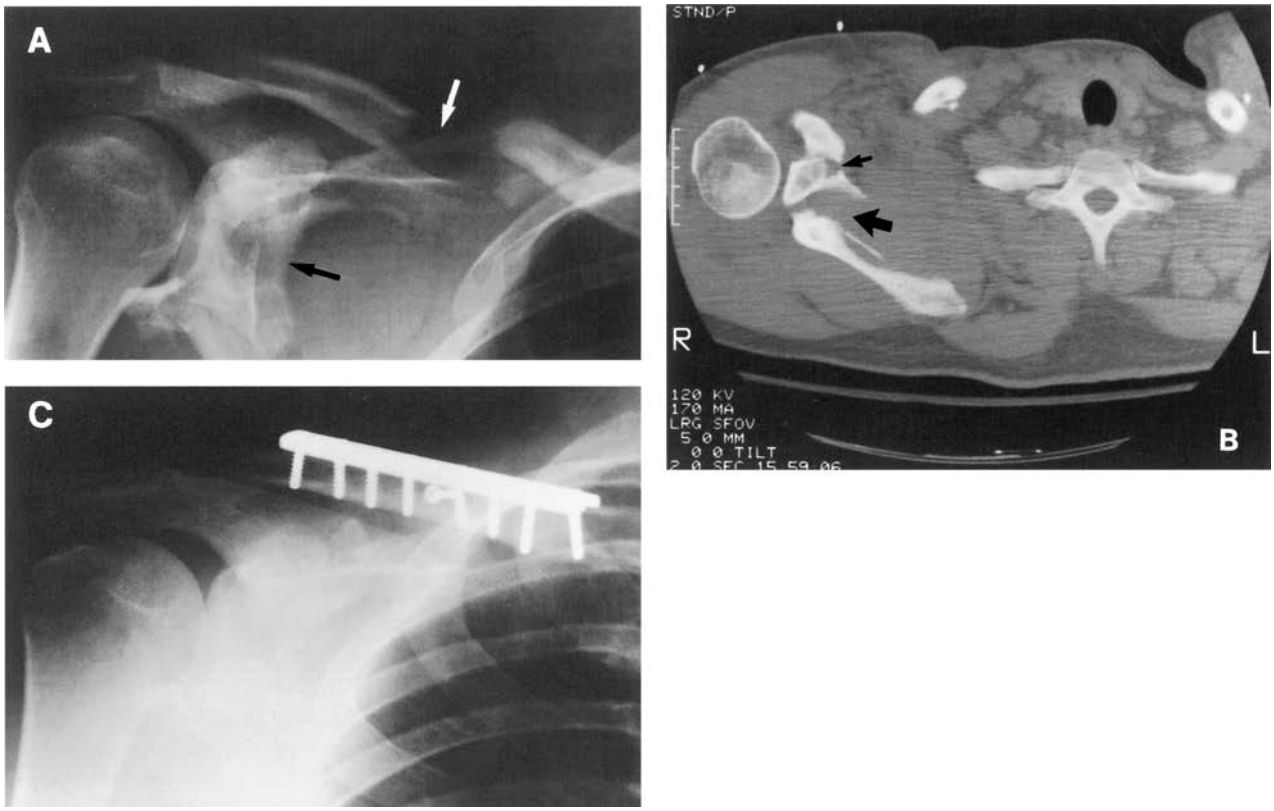


Figure 26-42 Radiographs of an individual who sustained a double disruption of the superior shoulder suspensory complex resulting in a "floating shoulder": **(A)** a preoperative anteroposterior (AP) radiograph showing a fracture of the glenoid neck with medial translation and a severely displaced fracture of the middle third of the clavicle; **(B)** a preoperative axial computed tomographic image showing the glenoid neck fracture to be complete, with the glenoid fragment and the entire superior shoulder suspensory complex rendered particularly unstable by an associated fracture of the coracoid process; and **(C)** a postoperative AP radiograph showing anatomic reduction and stabilization of the clavicle fracture. The glenoid neck fracture was managed nonoperatively, although a strong case could have been made for open reduction and internal fixation in light of its persistent medial translational displacement (the glenoid fragment remained unstable owing to the fractured coracoid process; a double disruption of the superior shoulder suspensory complex was still present).

sites are treated appropriately. Leung and Lam[113] reported on 15 patients treated surgically (average follow-up period 25 months). In 14 of the 15 patients, the fractures healed with a good or excellent functional result. Herscovici et al.[80] reported the results of nine patients with ipsilateral clavicular and glenoid neck fractures (average follow-up period 48.5 months). Seven patients were treated surgically with plate fixation of the clavicular fracture and achieved excellent results. Two patients were treated without surgery and had decreased range of motion as well as "drooping" of the involved shoulder. The authors strongly recommended ORIF of the clavicle to prevent a glenoid neck malunion. Simpson and Jupiter in a review article indicated that these injuries frequently require operative treatment.[177] Rikli et al. expanded this concept somewhat, saying that a fracture of the glenoid neck combined with either a fracture of the clavicle or a disruption of the AC joint or the sternoclavicular (SC) joint results in an "unstable shoulder girdle." They reviewed 13 cases (12 patients) in which the clavicular injury was surgically stabilized and reported excellent results in nearly all.[164]

There has been a great deal of interest recently as regards "floating shoulders," including both clinical and basic science studies. Edwards et al.[37] reported excellent results with conservative treatment of consecutive patients with ipsilateral clavicle and scapular fractures; however, in 5 of the 20 individuals, the scapula fracture did not involve the glenoid neck. Hashiguchi and Ito[74] reported excellent results in five patients with ipsilateral clavicle and glenoid neck fractures in whom fixation of the clavicle alone was performed. Van Noort et al.[193] reported a multicenter study of 46 patients with ipsilateral clavicle and glenoid neck fractures treated both operatively and nonoperatively, with mixed results in both groups. Egol et al.[38] reviewed their results in 19 patients (some treated surgically based on surgeon preference and some treated nonoperatively) and reported good results with each approach. Ramos et al. also reported on a series of patients treated nonoperatively who did quite well.[161] Williams et al. conducted a cadaveric study to determine the stability afforded by specific structures. Using a model with ipsilateral glenoid neck and clavicular fractures, they found that instability of the glenoid segment occurred only with subsequent coracoacromial and coracoclavicular ligamentous sectioning. They concluded that "floating shoulders" only become unstable when there is an associated disruption of the CA and AC ligaments.[200] Perhaps an associated coracoid process fracture would result in instability as well since it serves as a point of attachment for the CA and CC ligaments. Current experience would seem to indicate that (a) the mere presence of a clavicular and a glenoid neck fracture does not demand operative treatment and many/most do well nonoperatively; (b) the more displacement there is at one or both sites, the greater the need for ORIF is; and (c) another disruption of the SSSC may be necessary for significant displacement to occur.

Coracoid Process Fracture as One Component of a Double Disruption of the Superior Shoulder Suspensory Complex

The coracoid process is a vital part of the SSSC serving as one of the bony components of the clavicular–CC ligamentous–coracoid (C-4) linkage that joins the scapula to the clavicular–AC joint–acromial strut. Consequently, if a coracoid process fracture is present and associated with another SSSC injury, the potential adverse consequences of a "double disruption" must be considered and treatment tailored accordingly.[55] Ogawa and coworkers believed that such fractures were usually deep to the CC ligament. They classified them as type I fractures and they thought that they represented a dissociation between the scapula and the clavicle and as such often required ORIF.[152] The following are examples:

A fracture of the coracoid process and a grade III disruption of the AC joint. On occasion, especially in young adults, a force that would otherwise cause a grade III sprain of the AC joint results in an avulsion fracture of the base of the coracoid process or the bony attachment of the CC ligament to the angle of the coracoid process instead of a disruption of the CC ligament (Fig. 26-43). A weight-bearing AP projection of the shoulder complex will show displacement of the distal end of the clavicle above the superior border of the acromion but the CC interval remains normal. Treatment of injuries with a small bony avulsion fracture of the angle of the coracoid process follows the principles developed for grade III AC joint disruptions—specifically, ORIF of the AC joint and coracoid should be considered in young individuals engaged in athletics or heavy manual physical work. Disruptions associated with a fracture of the base of the coracoid process (especially if significantly displaced) should be considered for surgical ORIF of both sites to avoid the adverse long-term effects of a grade III AC separation and a nonunion of the coracoid process. Reports in the literature have described both operative and nonoperative management of these injuries.[15,26,39,69,81,103,110,122,137,159,179,196,208]

Fractures of the ipsilateral coracoid and acromial processes. Isolated fractures of the acromial and coracoid processes (Fig. 26-44) are almost always minimally displaced and, therefore, managed nonoperatively. When they occur together, however, they constitute a "double disruption" of the superior shoulder suspensory complex, a potentially unstable anatomic situation.[25,209] If displacement at either or both sites is unacceptable, surgical management is indicated. ORIF of the acromial fracture may be all that is required, since this will often indirectly reduce and stabilize the coracoid fracture satisfactorily and is technically less difficult than addressing the coracoid injury. If not, however, the coracoid

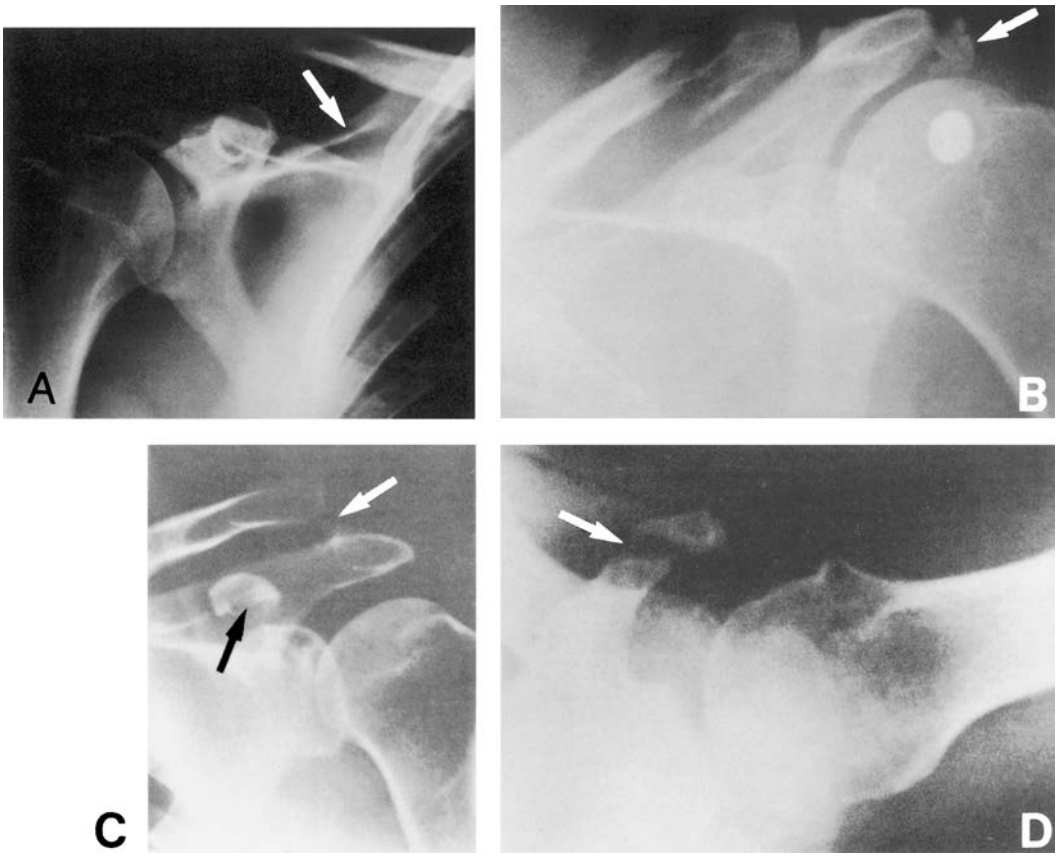


Figure 26-43 Radiographs showing a variety of scapular avulsion fractures: **(A)** a minimally displaced fracture of the superior angle of the scapula (attachment of the levator scapulae; *arrow*); **(B)** a displaced fracture of the lateral margin of the acromial process (origin of the deltoid muscle; *arrow*); **(C)** a type III disruption of the acromioclavicular joint (*white arrow*) with an associated avulsion fracture at the base of the coracoid process; and **(D)** displaced fracture of the tip of the coracoid process (attachment of the conjoined tendon; *arrow*).

fracture may need to be reduced and stabilized as well. Lim and coworkers described such an injury managed with ORIF of both sites.[115]

A fracture of the base of the coracoid process and a fracture of the glenoid neck. A fracture of the glenoid neck can only displace if it is complete (i.e., if the fracture line exits the lateral scapular border and the superior scapular margin adjacent to the coracoid process). Even so, if continuity of the glenoid fragment with the clavicular–AC joint–acromial strut via the C-4 linkage is intact, displacement is usually minimal. If, however, this linkage is disrupted (e.g., a fracture at the base of the coracoid process), significant displacement of the glenoid fragment is particularly likely (translational displacement greater than or equal to 1 cm and/or angular displacement greater than or equal to 40 degrees) and surgical management must be considered.[60] Operative treatment consists of ORIF of the glenoid neck fracture through a posterior or posterosuperior approach. The coracoid fracture will usually heal without direct intervention.

A fracture of the coracoid process and a type I fracture of the distal third of the clavicle (Fig. 26-45). Fractures of the distal third of the clavicle can displace unacceptably if the continuity of the coracoclavicular ligament between the coracoid process and the proximal clavicular segment is disrupted (type II and V fractures[141,142]). The same situation can occur with type I fractures if the coracoid process is fractured. (It would probably make most sense to call all of these injuries type II fractures, that is, situations in which the distal third of the clavicle is fractured, and the linkage between the proximal segment and the scapula [the C-4 linkage] is disrupted.) If displacement at the clavicular fracture site is of such a degree that it makes a delayed union or a nonunion likely, treatment consists of surgical reduction and stabilization of the injury (usually by means of tension band fixation). The coracoid fracture may reduce secondarily and heal uneventfully. If not, the coracoid fracture may also need to be addressed surgically.

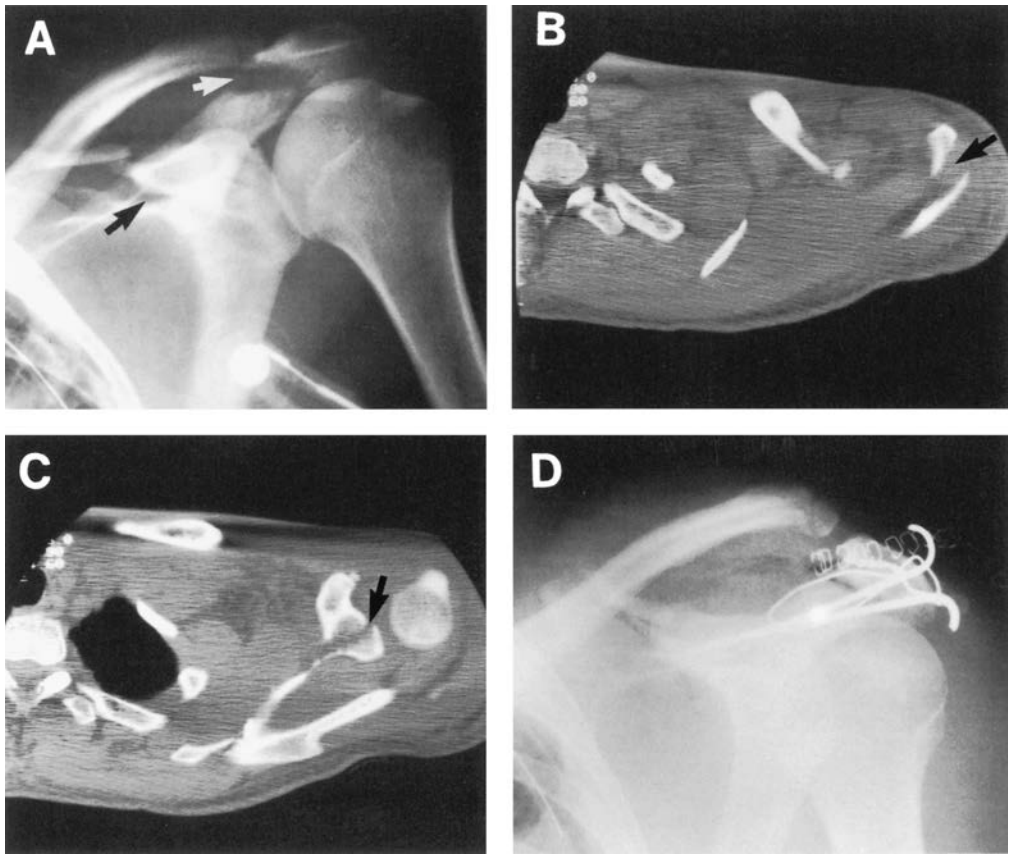


Figure 26-44 A patient who sustained ipsilateral fractures of the coracoid and acromial processes and distal clavicle: **(A)** a preoperative anteroposterior (AP) radiograph of the involved area (*white arrow,* acromial fracture; *black arrow,* coracoid fracture); **(B)** a preoperative axillary computed tomographic (CT) image showing wide separation at the acromial fracture site (*arrow*) owing to the associated coracoid process fracture; **(C)** a preoperative axillary CT image showing the fractured coracoid process (*arrow*); and **(D)** a postoperative AP radiograph of the shoulder showing reduction and stabilization of the acromial fracture using a tension band construct (the coracoid process fracture and distal clavicle fracture were not addressed and healed spontaneously).

Acromial Fracture as One Component of a Double Disruption of the Superior Shoulder Suspensory Complex

Acromial fractures that are significantly displaced are usually the result of the instability created when other fractures or ligamentous disruptions of the SSSC are present.[55] These injuries are generally the result of high-energy trauma and often require surgical intervention. Ogawa and Naniwa divided acromial fractures into two types: type I (lateral to the spinoglenoid notch) and type II (descending into the spinoglenoid notch). They believed that the mechanism of each was different and that type I injuries were more likely to be associated with other injuries to the SSSC (i.e., a "double disruption"), were more likely to be significantly displaced, and were more likely to require ORIF.[151] The following are examples:

Ipsilateral acromial and coracoid process fractures (discussed earlier).

An acromial fracture and a grade III disruption of the acromioclavicular joint. This combination creates a free-floating acromial fragment and can lead to a nonunion as well as the well-described adverse long-term functional consequences associated with AC joint disruptions. One case report describes an individual with an associated axillary nerve deficit.[126] Acutely, isolated grade III AC joint disruptions are usually managed nonoperatively; however, in this situation, if displacement at the acromial fracture site is unacceptable, surgical reduction and stabilization of both injuries is indicated. Kurdy and Shah described a patient treated nonoperatively who realized a "satisfactory" outcome; however, he was 74 years old and an acromial nonunion occurred.[106] Gorczyca and coauthors described an injury that gradually displaced over time and eventually required ORIF.[53] Torrens and colleagues described a combined type VI AC joint disruption and an

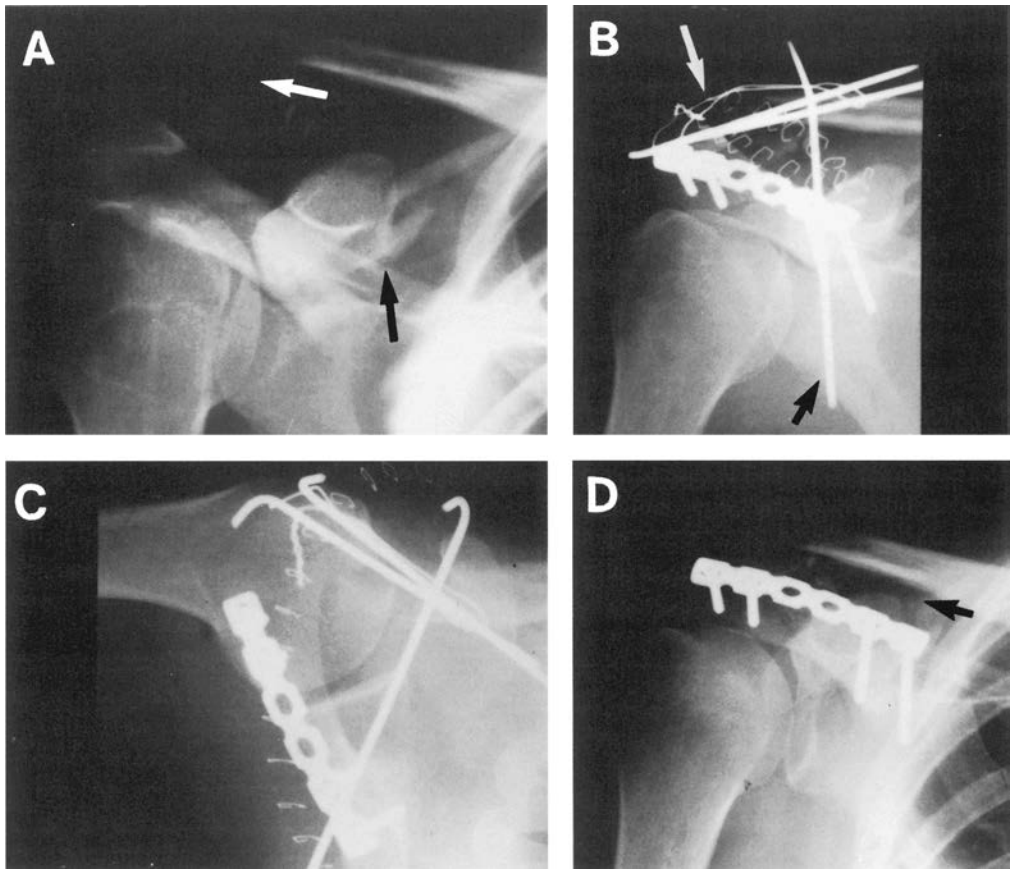


Figure 26-45 A patient who sustained a fracture of the base of the coracoid process, a type I fracture of the distal clavicle, and a nondisplaced fracture of the acromion: **(A)** a preoperative anteroposterior (AP) radiograph showing severe displacement at the distal clavicular fracture site (*white arrow*) and the coracoid fracture (*black arrow*); **(B,C)** postoperative AP and axillary radiographs showing reduction and stabilization of the clavicular fracture using a tension band construct (*white arrow*) and reduction and stabilization of the coracoid process fracture by means of a transfixing K-wire passed into the glenoid process (a cannulated interfragmentary screw would now be used; the acromial fracture was plated, *black arrow*); and **(D)** AP radiograph taken 3 months postoperatively following removal of hardware showing the distal clavicle and coracoid fractures to be healed and the superior shoulder suspensory relationships reestablished (*arrow*).

acromial fracture requiring ORIF of both injury sites.[189]

A segmental fracture of the acromion. As with segmental fractures of other bones, two acromial disruptions create an unstable intermediate segment. If displacement at one or both sites is unacceptable and a nonunion likely, ORIF through a posterolateral approach using plate fixation and/or a tension band construct is performed (Fig. 26-46).

Type III, Vb, and Vc Glenoid Cavity Fractures and Another Disruption of the Superior Shoulder Suspensory Complex

See Fig. 26-30.

ISOLATED FRACTURES OF THE CORACOID PROCESS

Classification and Mechanism of Injury

The coracoid process develops from two constant ossification centers: one at its base, which also forms the upper third of the glenoid process; and one that becomes its main body. In addition, there are at least two inconstant centers: one at its angle where the coracoclavicular ligament attaches and one at its tip where the conjoined tendon is located. The regions at which the centers finally unite are relatively weak, especially in young adults, making fractures more likely to occur when direct or indirect forces are applied.[137,159] The coracoid process, which has been called the "lighthouse of the anterior shoulder," has three basic functions: (a) it serves as a point of attachment

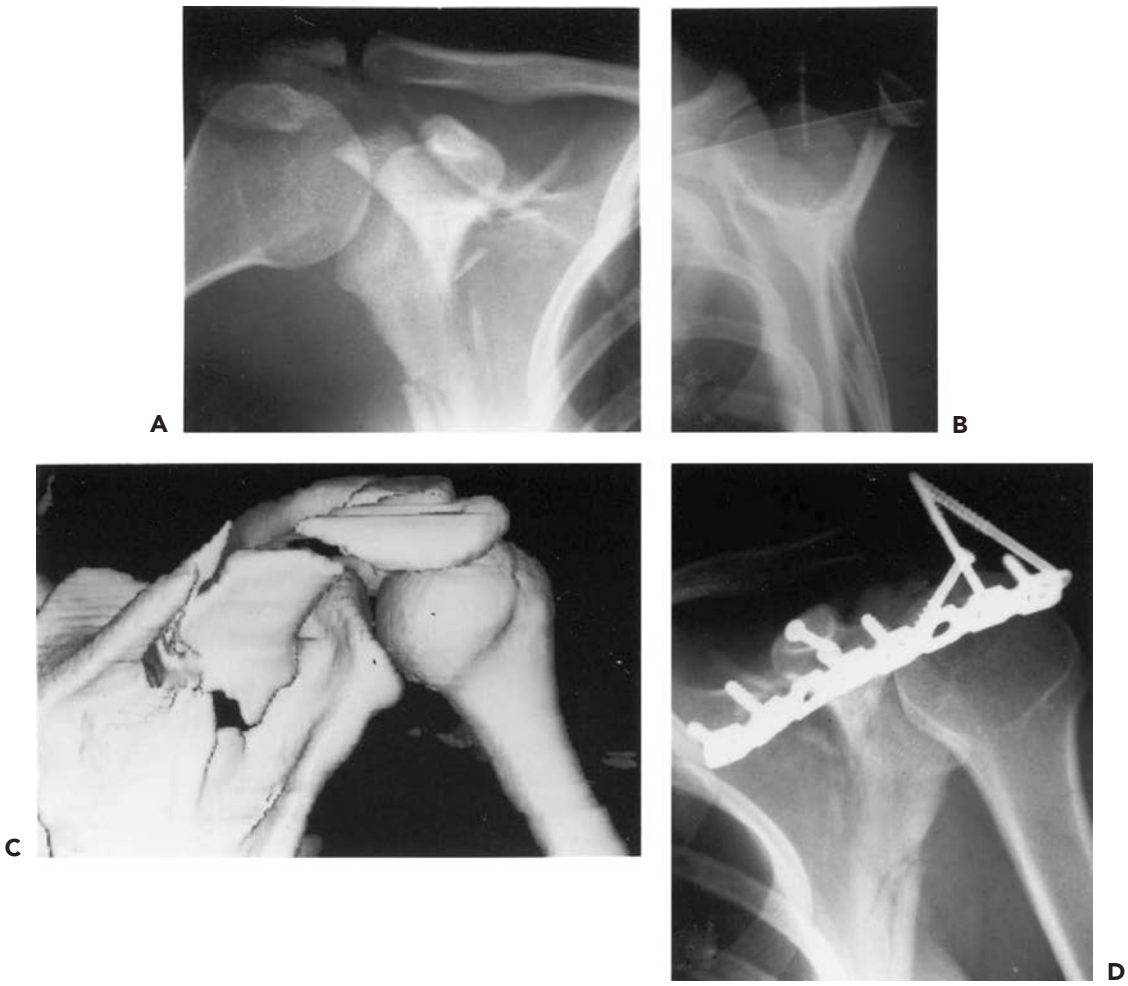


Figure 26-46 Segmental fracture of the acromial process as well as a comminuted fracture of the scapular body: **(A)** preoperative anteroposterior (AP) radiograph; **(B)** preoperative lateral scapular radiograph; **(C)** preoperative three-dimensional computed tomographic radiograph; and **(D)** postoperative AP radiograph showing the acromial fractures reduced and stabilized with a malleable reconstruction plate. (From Goss TP. The scapula: coracoid, acromial, and avulsion fractures. *Am J Orthop* 1996;25:106, with permission.)

for a number of musculotendinous and ligamentous structures (Fig. 26-47), (b) it provides the glenohumeral joint with some anterosuperior stability, and (c) it is an integral part of the SSSC (the scapular–clavicular linkage system), serving as one of the bony components of the clavicular–CC ligamentous–coracoid [C-4] linkage. Coracoid fractures may be caused by a blow from the outside, contact with a dislocating humeral head, or indirect forces applied through the musculotendinous and ligamentous structures at their attachment sites (avulsion fractures fall into this category[27,41,172]). Fatigue fractures have also been described.[21,173] Nontraumatic causes include (a) fractures associated with coracoclavicular tape fixation used in AC joint reconstructions[136] and (b) fractures associated with massive rotator cuff tears. Despite the relative scarcity of isolated coracoid fractures, several types have been described.[39,64] These injuries can be anatomically divided into (a) fractures of the tip of the coracoid (see Fig. 26-43D), (b) fractures between the CC and CA ligaments, and (c) fractures at the base of the coracoid process. Fractures of the coracoid tip are avulsion injuries—the result of an indirect force applied to the conjoined tendon and concentrated over its attachment to the coracoid process (see Fig. 26-43D). Fractures between the CC ligament and the CA ligament may be the result of either a direct or an indirect force.[30] The distal coracoid fragment is usually significantly displaced, drawn distally by the pull of the conjoined tendon and rotated laterally by the tethering effect of the coracoacromial ligament (Fig. 26-48). Fractures of the base of the coracoid process are the most common coracoid fractures. They may be caused by a direct blow from the outside or a dislocating humeral head.[13] Avulsion fractures caused by strong traction forces are also possible.[120,160,167]

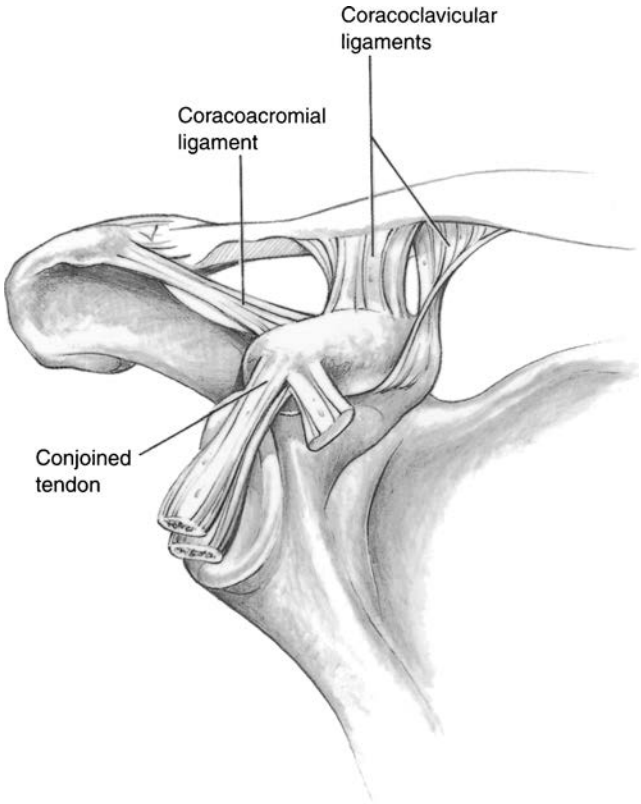


Figure 26-47 Illustration showing the coracoid process as the point of attachment for the conjoined tendon, the coracoacromial ligament, and the coracoclavicular ligament.

Diagnosis

Diagnosis is ultimately radiographic. True AP and axillary projections of the glenohumeral joint will disclose, or at least suggest, the presence of most coracoid fractures. Because of the complex bony anatomy in the area, "tilt"–oblique views,[45,52] or even CT scanning,[99] may be necessary to detect and accurately define some fractures as well as injuries to adjacent bony and articular structures. Accessory ossification centers and epiphyseal lines may complicate the evaluation. A weight-bearing AP view of the shoulder should be obtained if there is concern over the integrity of the scapular–clavicular linkage.

Management

Fractures of the Coracoid Tip

Displacement may be quite marked, but nonsurgical treatment is usually in order.[205,209] ORIF has been advocated in athletes, especially those participating in sports that require optimal upper extremity function, and in persons who perform heavy, manual, physical work. Wong-Chung and Quinlan described a case in which a fractured coracoid tip prevented the closed reduction of an anterior glenohumeral dislocation.[204] Late surgical treatment may be necessary if the displaced bony fragment causes irritation of the surrounding soft tissues.[14] Surgical management (either acute or late if a symptomatic nonunion occurs[49]) takes two forms: (a) ORIF of the bony fragment if sufficiently large and noncomminuted, or (b) excision of the fragment and suture fixation of the conjoined tendon to the remaining coracoid process (Fig. 26-49).

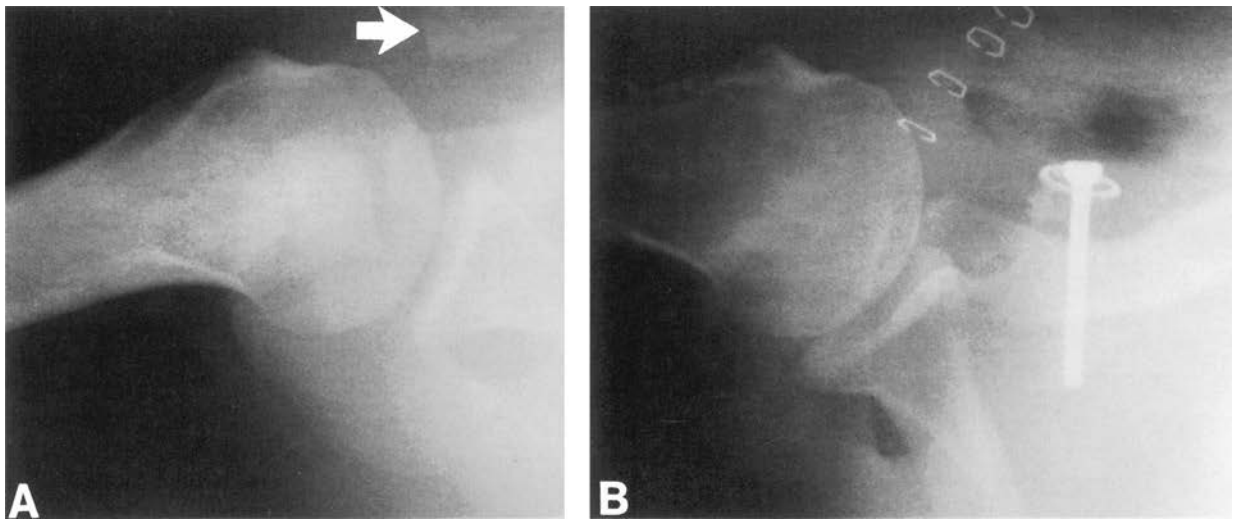


Figure 26-48 A patient who sustained a fracture of the distal coracoid process between the coracoclavicular ligament and the coracoacromial ligament: **(A)** preoperative axillary radiograph showing the significantly displaced distal portion of the coracoid process (*arrow*); and **(B)** postoperative axillary radiograph showing the bony fragment reduced and stabilized using an interfragmentary screw with a ligament washer.

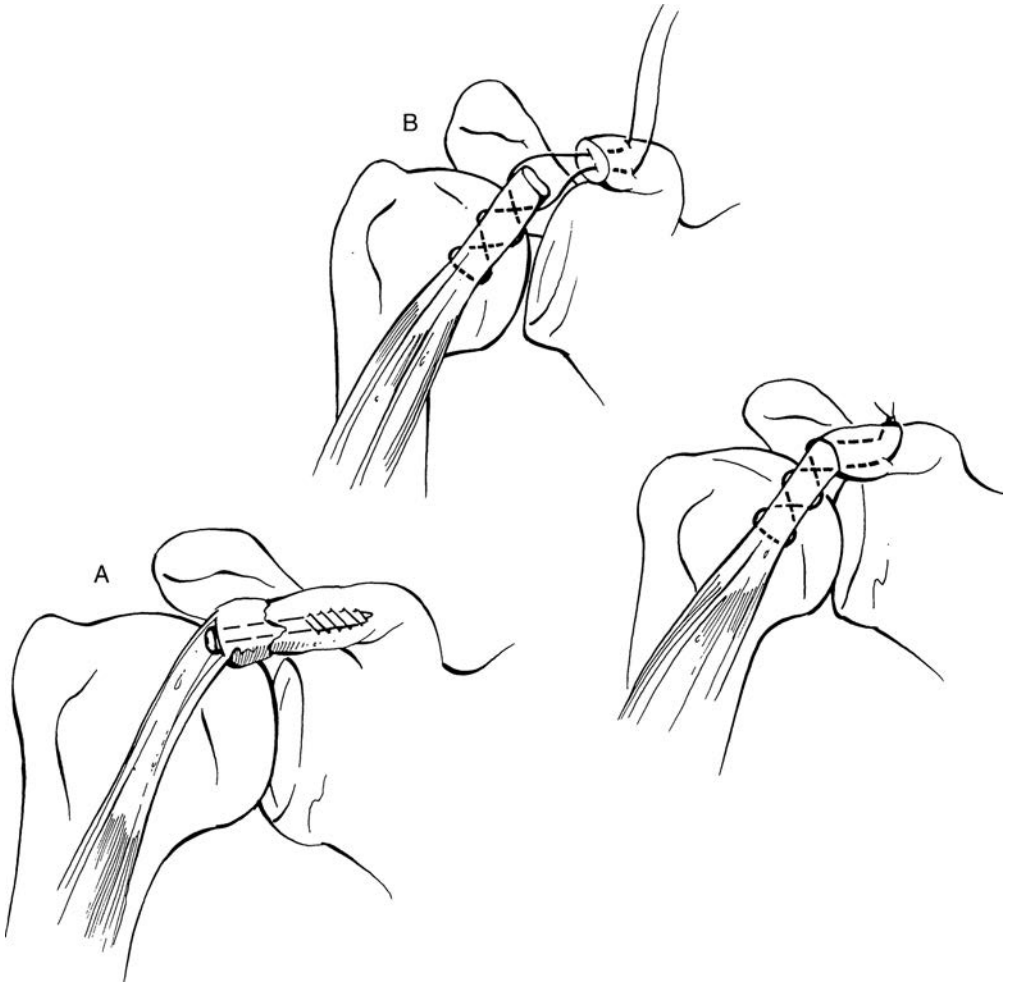


Figure 26-49 Illustrations showing two surgical techniques for managing coracoid process fractures: **(A)** interfragmentary screw fixation (if the fragment is sufficiently large and noncomminuted); and **(B)** excision of the distal fragment (if small and/or comminuted) and suture fixation of the conjoined tendon to the remaining coracoid process.

Fractures Between the Coracoclavicular Ligament and the Coracoacromial Ligament

Initial treatment may be nonsurgical or surgical, following the same reasoning described for significantly displaced avulsion fractures of the coracoid tip. Because the fragment is larger, however, symptomatic irritation of the local soft tissues is more frequent and late surgical management is more likely. The size of the fragment generally makes it amenable to interfragmentary screw fixation. Cannulated 3.5- and 4.0-mm compression screws are particularly useful. As with all coracoid process fractures managed surgically, an anterior deltopectoral interval approach is used, and the rotator interval is opened as need be for optimal exposure of the fracture site (see Fig. 26-29).

Fractures of the Base of the Coracoid Process

These injuries are generally minimally displaced owing to the stabilizing effect of the surrounding soft tissues, in particular the coracoclavicular ligament (see Fig. 26-26). Symptomatic nonsurgical care is usually sufficient and union occurs within 6 weeks.[50,205,209] McLaughlin felt that fibrous union is not uncommon, but is rarely associated with discomfort.[129] If symptomatic, however, bone grafting and compression screw fixation must be considered. This is accomplished by an anterior deltoid-splitting approach, opening the rotator interval for adequate exposure.

ISOLATED FRACTURES OF THE ACROMIAL PROCESS

Classification and Mechanism of Injury

The acromial process is formed from two ossification centers: one for most of its anterior end and one for its posterolateral tip (its base is actually an extension of the scapular body and spine). The acromial process has four

basic functions: (a) it provides one side of the acromioclavicular articulation; (b) it serves as a point of attachment for various musculotendinous and ligamentous structures; (c) it lends posterosuperior stability to the glenohumeral joint; and (d) it is an important component of the SSSC (the scapular–clavicular linkage).

Acromial fractures may be caused by a direct blow from the outside or a force transmitted via the humeral head. Avulsion fractures are the result of purely indirect forces. Even stress and fatigue fractures have been reported.[197] These injuries may be minimally or significantly displaced.

Kuhn et al. proposed a classification scheme that drew some discussion.[101,102,134,186] They emphasized the need for ORIF if an acromial fragment is displaced inferiorly by the pull of the deltoid muscle, compromising the subacromial space, thereby resulting in impingement symptoms and interfering with rotator cuff function.[100]

Diagnosis

Diagnosis is radiographic. True AP and lateral views of the scapula and a true axillary projection of the glenohumeral joint will detect most acromial fractures. The presence of an os acromionale may complicate the evaluation. On occasion, however, CT scanning may be needed to precisely define the injury and disclose involvement of adjacent bony and/or articular structures. A weight-bearing AP projection is obtained if a disruption of the scapular–clavicular linkage is suspected. Arthrography to evaluate the rotator cuff should be considered if the acromial fracture is the result of traumatic superior displacement of the humeral head or chronic superior migration of the humeral head, as seen in longstanding rotator cuff disease (e.g., cuff tear arthropathy with a stress fracture of the acromion). Madhaven et al. described the case of an individual with an acromial fracture associated with an avulsed subscapularis tendon.[118]

Management

Although significantly displaced isolated nonavulsion acromial fractures have been described, the vast majority are nondisplaced or minimally displaced. Symptomatic, nonoperative care will reliably lead to union and a good to excellent functional result. If surgical reduction and stabilization is necessary, a tension band construct is usually chosen for distal disruptions where the acromial process is quite thin, whereas 3.5-mm malleable reconstruction plates are usually chosen for more proximal injuries (Fig. 26-50).[51] Symptomatic acromial nonunion, although uncommon, has been reported in the literature.[54] No more than a small fragment should ever be excised. The presence of a large fragment requires surgical stabilization and bone grafting.[64]

Significantly displaced avulsion fractures may occur wherever musculotendinous or ligamentous structures (the deltoid and trapezius muscles as well as the coracoacromial and acromioclavicular ligaments) attach to the acromion. Two varieties have been described in the literature:

1. An avulsion fracture of the origin of the deltoid muscle.[81] The deltoid is the most important dynamic structure about the glenohumeral joint. Consequently, if a fracture is significantly displaced, surgical reattachment is indicated. This is rather simple and accomplished with multiple nonabsorbable sutures passed in a horizontal mattress fashion through the deltoid and drill holes made along the periphery of the acromial process.

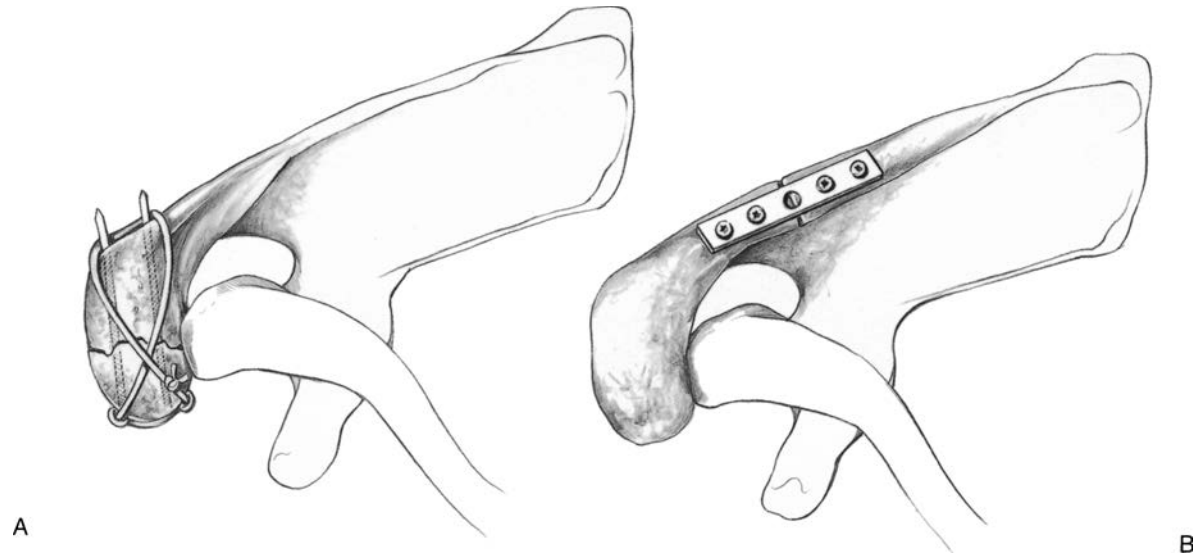


Figure 26-50 Illustrations showing two surgical techniques for managing fractures of the acromial process: **(A)** a tension band construct (most appropriate for fractures through the distal portion of the acromion); and **(B)** plate–screw fixation (most appropriate for proximal fractures).

2. An avulsion fracture through the main body of the acromion.[139,163] Two cases have been described. Both were caused by significant forces transmitted through the surrounding musculature, especially the deltoid. One was treated nonsurgically, whereas the second was managed surgically. Bony union and satisfactory return of function was realized in both.

AVULSION FRACTURES OF THE SCAPULA

Classification and Mechanism of Injury

Most of the scapula is formed by intramembranous ossification, but it also has at least six or seven secondary ossification centers. The scapula has three basic functions: (a) it provides a semistable, yet fairly mobile, platform for the humeral head and the upper extremity to work against; (b) it serves as a point of attachment for a variety of soft tissue structures (musculotendinous and ligamentous; Fig. 26-51); and (c) it takes part in three articulations: the glenohumeral joint, the acromioclavicular joint, and the scapulothoracic articulation. As with other scapular fractures, avulsion injuries are uncommon. By definition, they are caused by indirect forces applied to the surrounding musculotendinous and ligamentous soft tissues and concentrated at their scapular attachment sites. Three mechanisms are possible: (a) severe, uncontrolled muscular contraction caused by electroconvulsive treatment, electric shock, or epileptic seizure;[16,33,77,81,92,96,123,157,176,185,198] (b) strong indirect forces associated with a single traumatic event;[17,31,208] and (c) gradual bony failure caused by lesser but repetitive traumatic events (stress[22] or fatigue[197] fractures). The potential varieties are numerous and many have been described.

Diagnosis

True AP and lateral projections of the scapula and a true axillary view of the glenohumeral joint constitute the diagnostic trauma series. This may be supplemented as need be by CT scanning and a weight-bearing view of the shoulder complex.

Management

Treatment is by and large symptomatic/nonoperative; however, if the fracture is significantly displaced and of functional importance, surgical ORIF must be considered.[64] The following are examples of those injuries that are managed quite successfully nonoperatively:

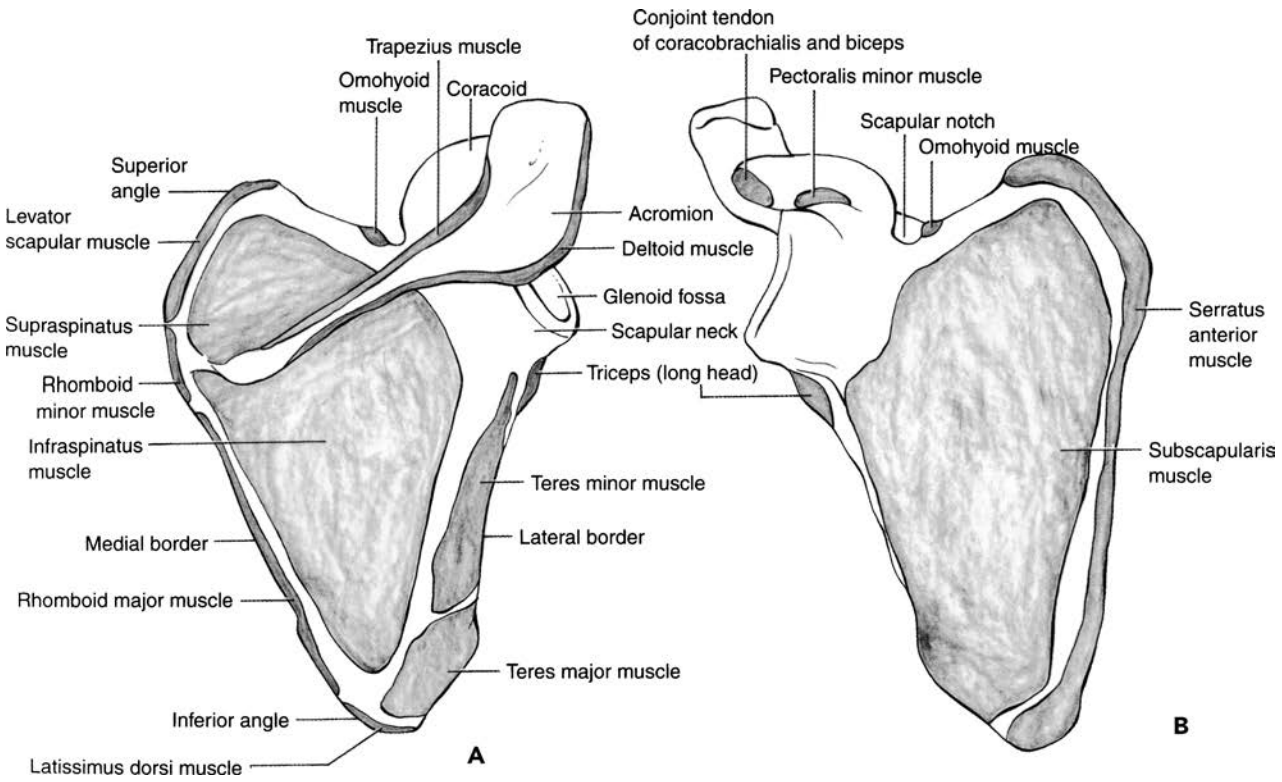


Figure 26-51 Illustrations showing the many scapular musculotendinous and ligamentous attachment sites: **(A)** posterior or dorsal surface of the scapula; and **(B)** anterior or costal surface of the scapula.

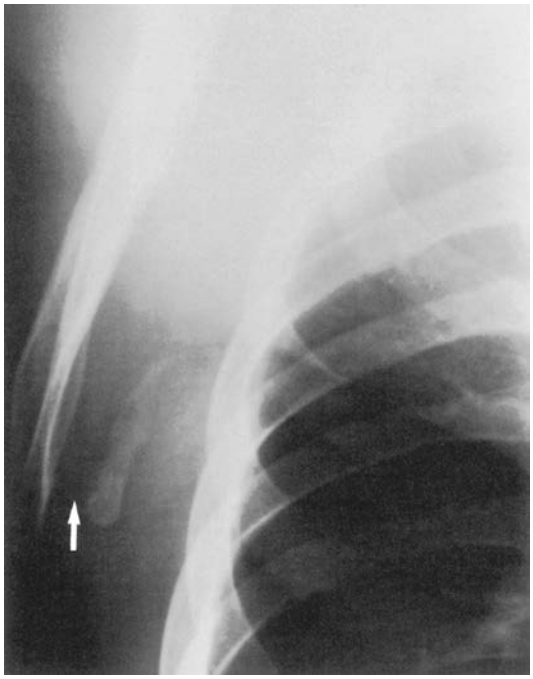


Figure 26-52 A true lateral radiograph of the scapula showing a severely displaced avulsion fracture of the inferior angle (the insertion of a serratus anterior muscle).

- Avulsion fracture of the superior angle of the scapula[54] (insertion of the levator scapulae; see Fig. 26-43A)
- Avulsion fracture of the superior border of the scapula. This injury is often associated with a fracture of the base of the coracoid process and an AC joint disruption.[7,10] Some have attributed the fracture to indirect stresses applied via the omohyoid muscle,[4,89,201] whereas others have considered it an extension of the coracoid fracture.[203]
- Avulsion fracture through the body of the scapula caused by an accidental electric shock[81,185]
- Avulsion fracture of the infraglenoid tubercle (origin of the long head of the triceps) and the lateral border of the scapula (origin of the teres major and minor muscles[81])
- Avulsion fracture of the infraspinatus fossa (origin of the infraspinatus muscle[10] [this must be differentiated from a developmental anomaly [162]])

The following are examples of avulsion fractures that, at the very least, deserve operative consideration:

- Avulsion fracture of the lateral margin of the acromial process (origin of the deltoid muscle;[81] see Fig. 26-43B)
- Avulsion fracture of the distal coracoid process (attachment of the conjoined tendon; see Fig. 26-47)
- Avulsion fracture of the superior angle of the coracoid process (attachment of the coracoclavicular ligament[137]) or the base of the coracoid process in association with a disruption of the acromioclavicular joint (see Fig. 26-43C)
- Avulsion fracture of the inferior angle of the scapula (Fig. 26-52; insertion of the serratus anterior muscle):[24] If significantly displaced, this rare injury causes winging of the scapula and can significantly compromise shoulder function—surgical ORIF is indicated.[75] (This may be the fracture Longabaugh described in 1924.[117])
- Avulsion fracture of the supraglenoid tubercle (origin of the long head of the biceps muscle) indicative of a displaced, possibly symptomatic SLAP lesion[84]
- Avulsion fracture through the body of the acromion: Two cases have been described. Both were caused by a significant force transmitted through the surrounding musculature, especially the deltoid. One was treated nonsurgically whereas the second was managed surgically. Bony union and satisfactory return of function were realized in both.[139,163]

SCAPULOTHORACIC DISSOCIATION

Lateral Dislocation of the Scapula

Scapulothoracic dissociation is a rare traumatic disruption of the scapulothoracic articulation caused by a severe direct force over the shoulder accompanied by traction applied to the upper extremity.[3,34,35,62,82,90,95,109,140,153,170,191] Although the skin remains intact, the scapula is torn away from the posterior chest wall, prompting some to call this injury a "closed traumatic forequarter amputation." Because of the violent forces involved, any of the three bones in the shoulder complex (the clavicle, the scapula, and the proximal humerus) may be fractured, and any of the remaining three articulations (the glenohumeral, AC, and sternoclavicular joints) may be disrupted. Neurovascular injury is common. Disruption of the subclavian and axillary artery (most frequently the former) and a complete or partial disruption of the brachial plexus are well described. In addition, there may be severe damage to the soft tissue supporting structures, especially those that run from the chest wall to the scapula or the chest wall to the humerus. Complete and partial tears of the trapezius, levator scapulae, rhomboids, pectoralis minor, and latissimus dorsi all have been reported. A presumptive diagnosis is based on a history of violent trauma in the presence of massive soft tissue swelling over the shoulder girdle. A pulseless upper extremity, indicating a complete vascular disruption, and a complete or partial neurologic deficit, indicating injury to the brachial plexus, are quite suggestive. Significant lateral displacement of the scapula seen on a nonrotated chest radiograph confirms the diagnosis. As with all rare injuries, awareness of the clinical entity is critical to making the correct diagnosis. Treatment recommendations have focused on care of the accompanying neurovascular injury. If the vascular integrity of the extremity is in question, an

emergency arteriogram is performed followed by surgical repair if necessary. The brachial plexus is explored at the same time. If a neurologic deficit is present, electromyographic testing is performed 3 weeks after injury to determine the extent of damage and to assess the degree of recovery, if any. Cervical myelography can be performed at 6 weeks. If nerve root avulsions or a complete neurologic deficit is present, the prognosis for a functional recovery is poor.[207] Partial plexus injuries, however, have a good prognosis and most patients achieve complete recovery or regain functional use of the extremity. If some portions of the plexus are intact and others are disrupted, neurologic repair is a possibility. Late reconstructive efforts are guided by the degree of neurologic return, and musculotendinous transfers are performed as needed. Care of the surrounding soft tissue supportive structures (musculotendinous and ligamentous) has been nonoperative, consisting of immobilization of the shoulder complex for 6 weeks to allow healing, followed by a closely monitored physical therapy program designed to restore range of motion initially, followed by strength. Magnetic resonance imaging (MRI) of the involved area now offers the ability to visualize important disruptions that may be amenable to surgical repair.

Injury to the sternal–clavicular–acromial linkage (a disruption of the sternoclavicular or AC joint or a fracture of the clavicle) is frequently, if not invariably, present for posterolateral displacement of the scapula to occur. This component of scapulothoracic dissociation has been largely ignored, both in terms of diagnosis and treatment. Of the three possible disruptions, a fracture of the clavicle seems to be the most common. This constitutes a very unstable anatomic situation—the clavicular injury allows maximal displacement of the scapula, whereas the unstable scapulothoracic articulation often leads to significant displacement at the clavicular fracture site. Consequently, surgical ORIF of the clavicle (screw plate fixation for fractures of the middle third and tension band fixation for fractures of the distal third) should be considered (a) to avoid a delayed union or a nonunion; (b) to restore as much stability as possible to the shoulder complex to avoid adverse long-term functional consequences; and (c) to protect the brachial plexus and subclavian and axillary vessels from further injury caused by tensile forces (Fig. 26-53). The upper extremity is protected for 6 weeks while a progressive rehabilitation program is instituted. Uhl and Hospeder described a lesser injury characterized by progressive subluxation of the scapulothoracic articulation and a clavicle fracture (no neurovascular involvement) requiring ORIF of the latter.[192] Similar therapeutic reasoning would apply to scapulothoracic dissociations accompanied by a disruption of the AC or the sternoclavicular joint, although in the latter, metallic fixation devices must be avoided.

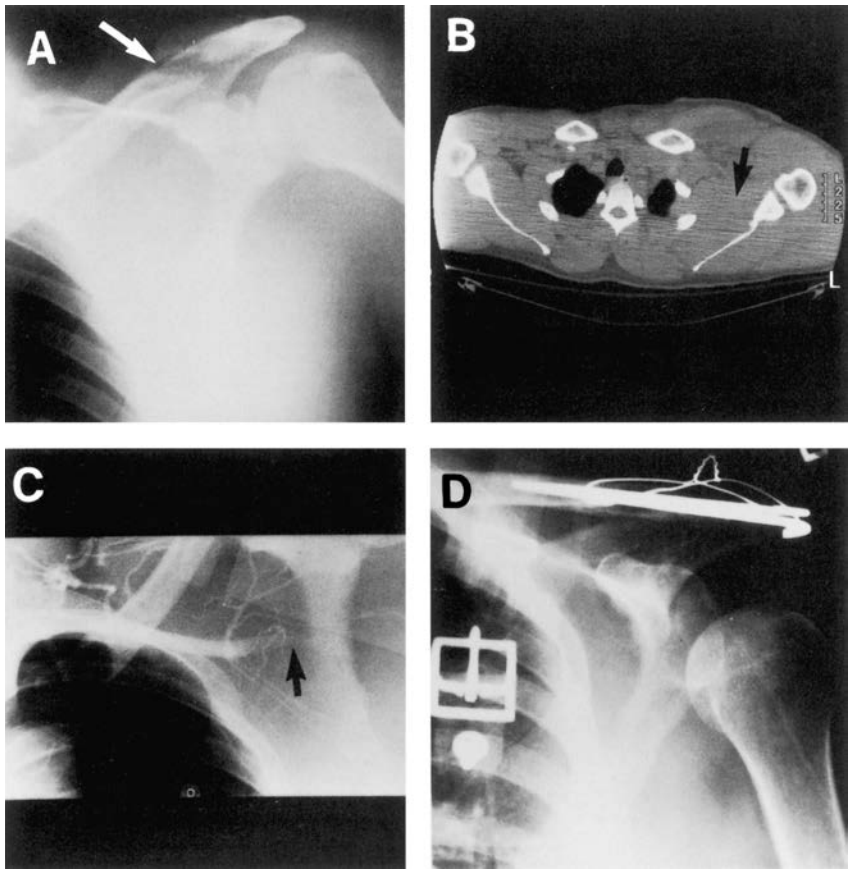


Figure 26-53 A patient who sustained a left scapulothoracic dissociation: **(A)** preoperative anteroposterior (AP) radiograph showing significant lateral displacement of the scapula and a significantly displaced fracture of the distal clavicle; **(B)** computed tomographic image showing significantly increased distance between the left scapula and the rib cage as compared with the opposite (uninjured) side; **(C)** arteriogram showing disruption of the subclavian artery; and **(D)** postoperative AP radiograph showing reduction and stabilization of the distal clavicle fracture (and secondarily the scapulothoracic articulation) by means of a tension band construct.

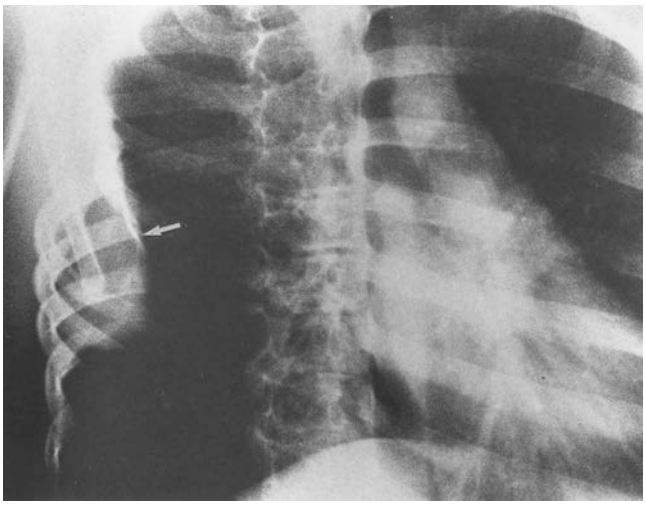


Figure 26-54 A right anterior oblique radiograph taken of a patient who sustained an intrathoracic dislocation of his scapula. (From Nettrour LF, Krufty LE, Mueller RF, et al. Locked scapula: intrathoracic dislocation of the inferior angle. *J Bone Joint Surg Am* 1972;54:413, with permission. Copyright is owned by The Journal of Bone and Joint Surgery, Inc.)

Intrathoracic Dislocation of the Scapula

Intrathoracic dislocation of the scapula is extremely rare. Cases associated with minimal violence and a preexisting factor (generalized laxity or locking osteochondroma) have been described. The scapula becomes locked within the posterior aspect of one of the upper intercostal spaces (Fig. 26-54).[144] A second type is caused by more violent trauma, either a direct blow applied over the posterior aspect of the scapula or a violent outward distractive force applied to the arm. The scapular body is displaced anterolaterally, and its inferior angle becomes lodged between the ribs. The severity of the event usually causes a fracture of the scapula and ribs as well as marked disruption of the periscapular soft tissues.[97] Pell and Whipple described an individual who sustained a fracture of the scapular body with the inferior angle locked within the fourth intercostal space that was managed successfully by closed reduction.[156] The diagnosis may be missed initially because of associated injuries and/or inadequate radiographic projections. Displacement of the scapula may not be readily apparent on routine AP chest radiographs. Tangential views (anterior oblique or lateral scapular projections[144]) or a chest CT may be necessary to establish the diagnosis.

Acute injuries are reduced in a closed fashion under anesthesia by hyperabducting the arm and manually manipulating the scapula (rotating the scapula forward and pushing it backward[32]) while steady traction is applied to the arm. The reduction is usually stable, but securing the scapula to the chest wall with adhesive tape and immobilizing the arm in a sling and swathe are advisable for comfort and soft tissue healing.[97] The dressing and immobilizer are changed at 7 to 10 days and discontinued 2 weeks thereafter. Unprotected and progressive functional use of the shoulder and arm is then permitted and encouraged. In long-standing cases, open reduction with soft tissue detachment may be necessary followed by reconstruction of the periscapular tissues to reestablish stability.[144]

COMPLICATIONS

The most significant complications associated with scapular fractures are those that result from accompanying injuries to adjacent and distant osseous and soft tissue structures. Because of the severe traumatic forces frequently involved, these patients have an average of 3.9 additional injuries, with the most common sites being the ipsilateral shoulder girdle,[87] upper extremity, lung, and chest wall. Twenty-five to 45% of patients have accompanying rib fractures; 15% to 40% have fractures of the clavicle; 15% to 55% have pulmonary injuries[130] (hemopneumothorax, pulmonary contusion, etc.); 12% have humeral fractures; and 5% to 10% sustain injuries to the brachial plexus and peripheral nerves.[34,188] Fractures of the skull are found in approximately 25% of patients, cerebral contusions in 10% to 40%, central neurologic deficits in 5%, tibial and fibular fractures in 11%, major vascular injuries in 11%,[70,182,188] and splenic injuries that result in splenectomy in 8%. Two percent of these patients die. A variety of other cardiothoracic, genitourinary, and gastrointestinal injuries have been described. Stephens et al. reviewed 173 blunt trauma patients (92 with scapular fractures and 81 controls) and concluded that scapular fractures were not a significant marker for greater mortality or neurovascular morbidity.[184] Veysi et al. also showed no increase in mortality; however, they did confirm significantly greater injury severity scores (ISSs) in trauma patients sustaining scapular fractures.[195]

Complications related to the scapular fractures themselves are relatively uncommon. Nonunion, although possible, is quite rare.[25,40,67,93] Malunion can occur in a variety of forms, depending on the particular fracture type. Malunion of a scapular body fracture is generally well tolerated; however, painful scapulothoracic crepitus has been described on occasion. Fractures of the glenoid cavity may result in symptomatic glenohumeral degenerative joint disease. Shoulder instability can occur following significantly displaced fractures of the glenoid neck (angulatory displacement) and fractures of the glenoid rim. Fractures of the glenoid neck with significant translational displacement may give rise to glenohumeral pain and dysfunction related to altered mechanics of the surrounding soft tissues.

Various complications associated with surgical management are possible, for example, infection (both superficial

and deep), intraoperative neurovascular injury, and loss of fixation owing to poor surgical technique. A poorly supervised postoperative physiotherapy–rehabilitation program may lead to unnecessary postoperative shoulder stiffness.

Finally, complications related to poor patient compliance may occur. Examples would include suboptimal shoulder range of motion caused by unwillingness to follow the postoperative physiotherapy program and hardware failure associated with failure to observe postoperative instructions.

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27 Fractures of the Proximal Humerus: Classification, Diagnosis, and Nonoperative Management

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ANATOMY

Development

In the newborn infant, the spherical proximal humeral epiphysis contains the primary ossification center and the two secondary centers that will form the greater and lesser tuberosities. The primary ossification center appears within 4 to 6 months of birth, the greater tuberosity at approximately 3 years, and the lesser tuberosity by 5 years. These coalesce between 4 and 7 years and fuse to the shaft between 17 and 20 years, with earlier physeal closure in girls than in boys[44,72,112] (Fig. 27-1).

The physeal plate of the proximal humerus is concave inferiorly and almost spherical, with its apex posterior and medial to its center. This asymmetrical metaphyseal dome and the strong thick attachment of periosteum along the posterior surface explain why anterior, rather than posterior,

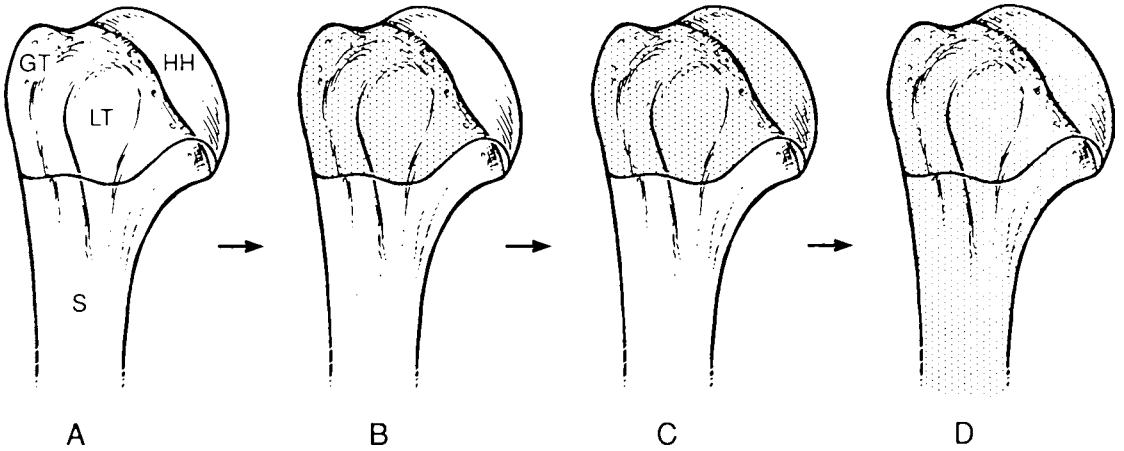


Figure 27-1 **(A)** The humeral head (*HH*) ossification center appears at 4 to 6 months of age, the greater tuberosity (*GT*) at 1 to 3 years of age, the lesser tuberosity (*LT*) at 3 to 5 years of age. **(B)** Coalescence of the GT with the LT occurs at approximately 3 years of age; **(C)** coalescence of the primary ossification center (*HH*) with the greater tuberosity and lesser tuberosity between 4 and 7 years of age; and **(D)** fusion to the shaft between 17 and 20 years of age.

displacement of the metaphysis occurs in fractures (Fig. 27-2). Dameron and Reibel demonstrated, in stillborn infants, that the metaphysis could easily be displaced anteriorly at the epiphyseal plate with the humerus extended and adducted; yet posterior displacement was difficult to achieve.[44] Approximately 80% of humeral growth occurs at the proximal physis, giving this region great remodeling potential following fracture.[49]

Vascular and Neurologic Anatomy

The proximity of the nerves of the brachial plexus and vascular structures to the proximal humerus puts them at risk of injury from proximal humerus fractures and dislocations, as well as during surgical approaches.

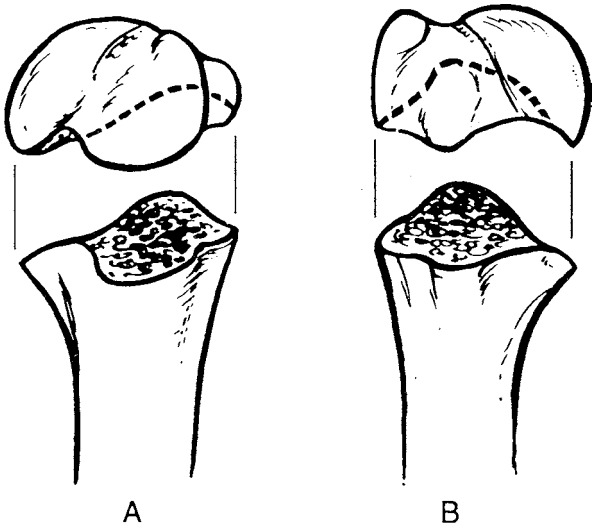


Figure 27-2 **(A)** Lateral and **(B)** anteroposterior view of the proximal humerus, demonstrating the asymmetrical physeal plate and metaphyseal dome with an apex posterior and medial to its center.

Vascular Anatomy

The rich vascular anatomy of the proximal humerus has been further delineated in the recent literature to help explain osteonecrosis of the humeral head. Laing's[93] and Gerber et al.'s[60] studies showed that the anterior lateral branch of the anterior humeral circumflex artery is the primary blood supply to the proximal humerus. The anterior humeral circumflex artery arises from the lateral side of the third division of the axillary artery approximately 1 cm distal to the inferior border of the pectoralis minor muscle (just above the teres major muscle) and courses laterally behind the coracobrachialis to reach the surgical neck of the humerus at the lower border of the subscapularis.[24,60,118] The anterolateral branch sends twigs to the lesser tuberosity, crosses under the biceps tendon, and then arches superiorly adjacent to the lateral side of the intertubercular groove. The vessel then penetrates bone at the cephalad portion of the transition from greater tuberosity to intertubercular groove, staying distal to the position of the old epiphysial plate. The intraosseous portion of this artery has been named the arcuate artery by Laing because of its posteromedial course after entering the humeral head. Multiple branches radiate to supply the subchondral bone of the humeral head[24] (Fig. 27-3).

The importance of both the extra- and intraosseous arterial anastomoses has recently been described. Laing noted abundant *extraosseous* anastomoses between the anterior humeral circumflex artery and posterior humeral circumflex arteries, as well as with the thoracoacromial, subscapular, suprascapular, and profunda brachii arteries.[93] The anterior and posterior circumflex vessels connect with the

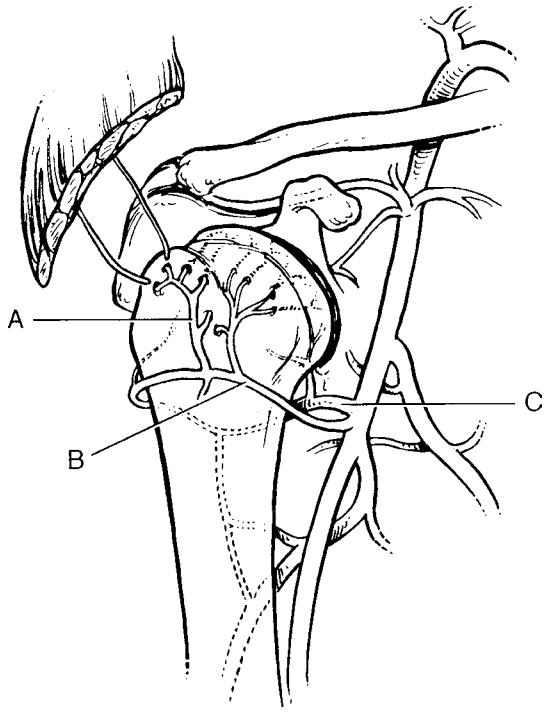


Figure 27-3 The rich vascular anatomy of the proximal humerus: **(A)** The anterior lateral branch of **(B)** the anterior humeral circumflex artery is the primary blood supply. **(C)** The posterior humeral circumflex artery arises close to the anterior humeral circumflex and gives off posterior medial branches to help supply the head.

profunda brachii artery through ascending deltoid vessels. Anastomosis of the profunda brachii artery occurs with the radial (anterior) and medial (posterior) collaterals. This rich collateral circulation about the shoulder can provide adequate circulation to maintain viability of the extremity despite axillary artery disruption after, for example, a dislocation.[53] Gerber et al. also identified abundant extraosseous anastomoses; however, vascularization of the entire humeral head was possible only through the anterior lateral branch of the anterior humeral circumflex artery. Laing noted inconsistent contributions to the humeral head from both the lesser and greater tuberosities.[93] Gerber et al. could not confirm that vessels of the rotator cuff directly vascularized the underlying bone.[60] The posterior humeral circumflex artery arises closely juxtaposed to the anterior humeral circumflex artery and is much larger in diameter.[118] Gerber found that the posteromedial vessels arising from the posterior humeral circumflex artery supplied the posterior portion of the greater tuberosity and a small posterior inferior part of the head. The vessels then formed an anastomosis to the arcuate artery on and within the greater tuberosity and on the joint capsule.[60] Brookes et al. demonstrated that these posteromedial vessels pass beneath the humeral capsular attachment (which at this site extends from 1 cm onto the surgical neck) and run toward the humeral head before entering the bone just below the articular margin.[24]

The study by Brookes et al. emphasized the importance of the *intraosseous* anastomoses and, unlike Gerber et al.'s study, showed that the humeral head could be completely profused after ligation of the anterior humeral circumflex artery at its entry site into the humeral head. They found large metaphyseal arteries that passed through the fused growth plate to anastomose with the arcuate artery in six of the eight normal and control specimens. In addition, significant intraosseous anastomoses occurred between the arcuate artery and the posterior humeral circumflex artery through the posteromedial vessels described in the foregoing and the vessels of the greater and lesser tuberosities.[24]

Knowledge of the vascular anatomy may help predict osteonecrosis of the humeral head. Gerber et al. highlighted the clinical relevance of the intraosseous anastomosis in a case report of a shoulder dislocation with both circumflex arteries interrupted and the extraosseous network compromised. Osteonecrosis did not develop in this patient, suggesting that the *intraosseous* anastomoses are important.[59] A *classic four-part* fracture will lose the blood supply to the head from disruption of the anterior humeral circumflex artery, greater and lesser tuberosities, and any metaphyseal arterial anastomosis. Thus, a high rate of osteonecrosis is expected.[113] Yet, Brookes et al. has shown that perfusion of the humeral head by the arcuate artery may continue if the head fragment includes part of the medial aspect of the upper part of the neck where the posteromedial arteries enter.[24] The so called *four-part valgus-impacted fracture* with limited lateral displacement of the head fragment may retain its vascularity from the posterior medial vessels, thus accounting for the lower incidence of osteonecrosis reported compared with classic four-part fractures.[79] The vascular anatomy also supports the observation that minimally displaced fractures of the anatomic neck, in which the fracture line is at the junction between the articular surface and neck, may be complicated by osteonecrosis as a result of disruption of both the extra- and intraosseous blood supply.[59]

Knowledge of the extensive collateral circulation around the shoulder helps explain the mechanism whereby the viability of the upper extremity can be maintained despite disruption of the axillary artery.

Muscles and Nerves

The proximal humerus is composed of the lesser tuberosity, which is the site of insertion of the subscapularis; the greater tuberosity, which has three facets for insertion of the supraspinatus, infraspinatus, and teres minor; the humeral head; and the shaft. The bicipital groove, a useful surgical landmark, is located between the tuberosities and contains the tendon of the long head of the biceps. The anatomic neck is at the junction of the articular margin of the humeral head and tuberosities, and the surgical neck is the area below the greater and lesser tuberosities.[11,118]

The important muscles and their innervations about the proximal humerus include the subscapularis muscle that arises from the medial two-thirds of the costal surface of the scapula. It is innervated on its costal surface by the upper and lower subscapular nerves. The supraspinatus muscle arises from the medial two-thirds of the bony walls of the supraspinatus fossa of the scapula, and from the dense fascia that covers the muscle. It is innervated from its underside by the suprascapular nerve. This nerve is relatively fixed at its origin from the superior trunk of the brachial plexus and where it passes under the superior transverse scapular ligament. Although rare, a traction injury can occur. The infraspinatus muscle takes its origin from the infraspinatus fossa of the scapula (except its lateral fourth) and from the dense overlying infraspinatus fascia. The suprascapular nerve passes through the notch of the scapular neck and under the inferior transverse scapular ligament to enter the upper part of the infraspinatus muscle.[118]

The teres minor arises from the upper two-thirds of the lateral border of the scapula and from adjacent intermuscular septa. The deltoid takes origin from the lateral third of the clavicle, the anterior and lateral border of the acromion, and the lower lip of the crest of the spine of the scapula. It inserts into the deltoid tuberosity of the humerus. Both of these muscles are supplied by the axillary nerve that arises from the posterior cord of the brachial plexus. An upper branch curves around the posterior surface of the humerus and courses from behind forward on the deep surface of the muscle, approximately 5 cm distal to the lateral border of the acromion, supplying muscular branches throughout its course. A lower branch supplies the teres minor as it ascends onto its lateral and superficial surface.[118] The axillary nerve is relatively fixed at its origin from the posterior cord and as it enters the deltoid. It also passes in close proximity to the inferior capsule, placing it at risk for a traction injury with downward motion of the proximal humerus, as in a dislocation. In fact, it is the most commonly injured nerve, especially from anterior dislocations.[13] This nerve can also be damaged with surgical approaches that split the deltoid fibers beyond 5 cm from the acromial edge or with percutaneous pins inserted from an inferior starting point.[74,97] The pectoralis major muscle has clavicular, sternocostal, and abdominal origins. It is innervated by both the lateral and medial pectoral nerves.[118]

The conjoined tendon is composed of the coracobrachialis and short head of the biceps brachii muscles, which take origin from the coracoid process. They are supplied by the musculocutaneous nerve, which is a branch of the lateral cord of the brachial plexus. This nerve enters the coracobrachialis muscle medially at a mean of 5.6 cm from the coracoid, but may be as close as 3.1 cm.[55] Therefore, retraction during anterior approaches places this nerve at risk for neurapraxia.[74]

The cords of the brachial plexus surround the axillary artery at the level of the humeral neck. Therefore, neurologic injury can result from fractures in this area. These injuries, though uncommon, are encountered more frequently than vascular injuries. Most commonly, they represent either a contusion or mild traction injury with a good prognosis for spontaneous recovery.[173] Electrophysiologic evidence of nerve injury is found in up to 45% of humeral neck fractures and primary dislocations, most commonly involving the axillary nerve, followed by the suprascapular, radial, and musculocutaneous nerves. Older patients and those with clinical evidence of hematomas have more neurologic injuries.[46] Although the neurologic injury does not have the limb-threatening potential of the vascular injury, long-term disabilities are determined by the brachial plexus injury. Although most patients with low-energy injuries recover partially or completely in less than 4 months,[46] permanent motor loss from brachial plexus injury will result in impairment of hand function. Treatment is focused on preservation of hand function, which is more important than shoulder motion.[149]

Muscle Forces

The attachment site of each muscle and the direction of pull are important determinants of fracture deformity and displacement.

The teres minor, infraspinatus, and supraspinatus muscles insert onto the greater tuberosity epiphysis, as does the subscapularis in the area of the lesser tuberosity. When the epiphysis is separated from the metaphysis as in Salter-Harris type I and II fractures, the muscular forces displace it into a position of flexion, abduction, and slight external rotation. The metaphysis often pierces the periosteum anterolateral to the biceps tendon and is pulled anteriorly and medially by the pectoralis major, which attaches to the metaphysis just below its junction with the physeal plate. It is also pulled proximally by the deltoid. The thick posterior periosteum remains intact[44,118] (Fig. 27-4). In the adult, Codman noted that after coalescence of the ossification centers and physeal plate, fractures tend to occur along the lines of the old epiphyseal plate scar, with patterns involving four important segments: the greater and lesser tuberosity, articular surface, and humeral shaft[34] (Fig. 27-5).

Neer emphasized the importance of the muscular attachment sites in determining fracture deformity and displacement[114,115] (Fig. 27-6A). In minimally displaced fractures (one-part), the periosteum, joint capsule, and rotator cuff attachments hold the fracture fragments together.[34] In a displaced surgical neck fracture (two-part), the humeral shaft is displaced forward and medially by the pectoralis major, while the head and attached tuberosities remain in neutral rotation because the rotator cuff is intact and balanced. In displaced fractures of the greater tuberosity

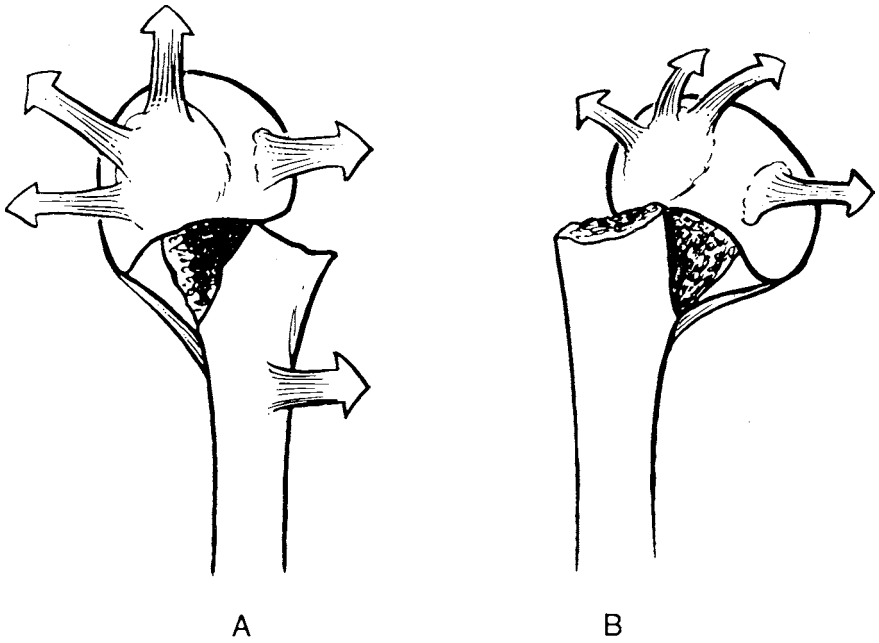


Figure 27-4 **(A)** Lateral and **(B)** anteroposterior view of proximal humerus, showing the muscle forces of the rotator cuff displacing the head and tuberosities into flexion abduction and slight external rotation. The metaphysis is pulled anteriorly and medially by the pectoralis major. The thick periosteum remains intact posteromedially.

(two-part), the supraspinatus and external rotators (infraspinatus and teres minor) actively retract fragments of the greater tuberosity superiorly and posteriorly. The direction may be more superior when the fragment is small and does not include all of the infraspinatus or more posterior with larger fragment size. When a displaced surgical neck component is also present (three-part), unopposed pull of the subscapularis internally rotates the articular segment, causing the articular surface to face posteriorly. This pattern of displacement is accompanied by a longitudinal tear of the rotator cuff (Fig. 27-6B). Similarly, a lesser tuberosity fracture will be displaced (two-part) medially by the subscapularis muscle. When this is accompanied by a displaced surgical neck fracture (three-part), the unopposed pull of the external rotators causes the articular segment to face anteriorly (Fig. 27-6C). Similarly, a longitudinal rotator cuff tear also occurs.

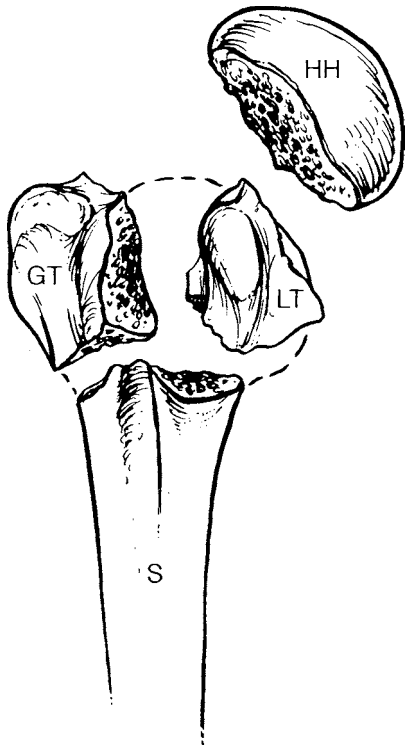


Figure 27-5 Proximal humerus fractures tend to occur along the lines of the old epiphyseal plate scar. The four important segments are the greater tuberosity (*GT*), lesser tuberosity (*LT*), articular surface (*HH*), and shaft (*S*).

If both tuberosities and the surgical neck are displaced (four-part), the greater tuberosity is retracted posteriorly and superiorly by the external rotators; the lesser tuberosity is retracted anteromedially by the subscapularis; and the pectoralis major retracts the shaft medially. The articular segment may be impacted on the upper shaft, displaced laterally, or dislocated anteriorly, posteriorly, or inferiorly. A longitudinal rotator cuff tear occurs between the subscapularis and supraspinatus in the rotator interval area (Fig. 27-6D).

In general, the deforming forces produced by the attached musculature prevent obtaining or maintaining a satisfactory closed reduction. Ultimately, this will determine fracture treatment.

CLASSIFICATION OF PROXIMAL HUMERAL FRACTURES

A classification system for proximal humerus fractures should provide a comprehensive means of describing

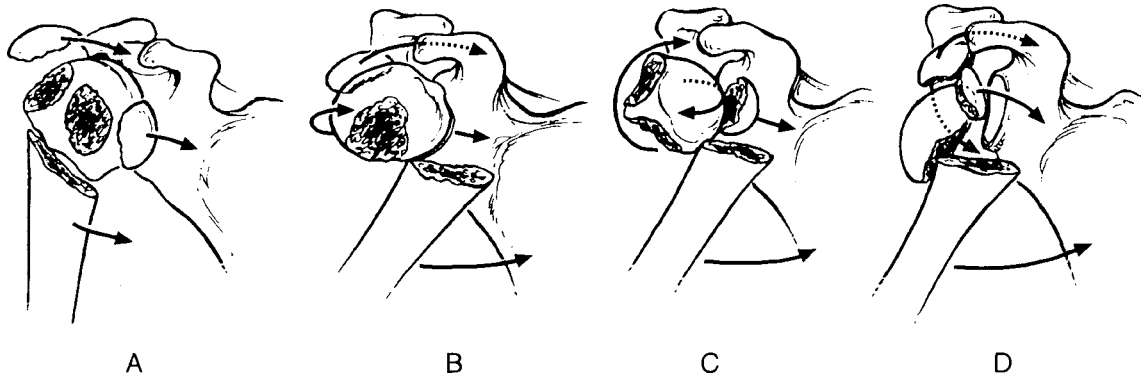


Figure 27-6 **(A)** *Arrows* demonstrating muscle forces on each segment of proximal humerus that determine fracture deformity and displacement. **(B)** Three-part greater tuberosity fracture demonstrates internal rotation of the articular segment and superior and posterior displacement of the greater tuberosity. **(C)** Three-part lesser tuberosity fracture demonstrates external rotation of the articular segment such that it faces anteriorly, and medial displacement of the lesser tuberosity. **(D)** Four-part fracture demonstrating posterior and superior displacement of the greater tuberosity, anterior medial retraction of the lesser tuberosity, and medial displacement of the shaft.

fracture fragment displacement and position, and the presence of dislocation. It should also assist in determining treatment, predicting long-term clinical outcomes, and providing an acceptable level of inter- and intraobserver reliability. And finally, the classification should be based on a standard radiographic evaluation that is easily and reproducibly obtained in the clinical setting.

Pediatric

Aitken classified fractures of the proximal humerus into three types. In his series of 11 cases, all fractures were of the first type: a fracture line through the layer of transitional cartilage and newly formed bone. There were no fractures through the epiphysis or evidence of epiphyseal "crush" injuries.[2]

Salter-Harris defined five types of epiphyseal injuries on the basis of radiologic and pathoanatomic patterns. Type I is a separation through the physis. Type II includes a metaphyseal fragment that is always posteromedial. Type III is a separation through the physis, with an extension of the fracture through the epiphysis and articular surface. Type IV is an intraarticular and transmetaphyseal fracture. Type V is physeal crush injury[136] (Fig. 27-7).

In a more pathophysiologic classification, Shapiro classified fractures of the physis as type A, B, or C. The neonatal epiphyseal separation of the proximal part of the humerus is considered a type A fracture (Salter-Harris I or Salter-Harris II), in which the separate epiphyseal and metaphyseal circulation remains intact and the longitudinal growth of the humerus is not arrested.[143] Neer and Horwitz further graded each fracture according to their initial displacement: grade I less than 5 mm; grade II less than one-third the width of the shaft; grade III up two-thirds of the shaft width; and grade IV more than two-thirds of the width.[112]

Adult

History

The classification of proximal humeral fractures has evolved over the last century parallel to our understanding

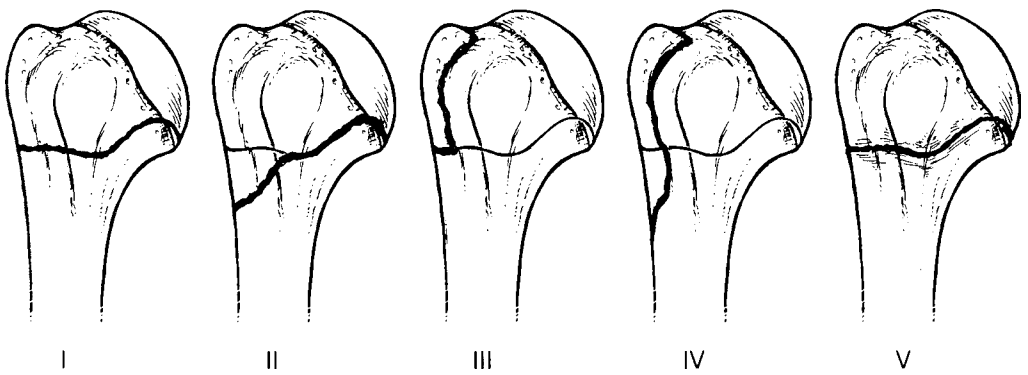


Figure 27-7 The Salter-Harris classification of physeal fractures of the proximal humerus (see text).

of these injuries. Major milestones in this evolution include Kocher's[83] classification that was based upon the different anatomic levels of fracture: anatomic neck, epiphysial region, and surgical neck. The disadvantages of this simplistic system included the lack of attention to important issues, such as the presence of fractures at multiple levels, the degree of fracture displacement, the presence of dislocation, or the mechanism of injury.

Recognizing that classification systems based solely on the anatomic level of fracture did not provide information about mechanism of injury nor assist in the choice of treatment, Watson-Jones[164] proposed a different classification system. He divided proximal humeral fractures into three types: contusion "crack" fractures, impacted adduction fractures, and impacted abduction fractures. He believed that each type was caused by a specific mechanism of injury and required a specific treatment approach. A major disadvantage of this system was that changes in humeral rotation altered the radiographic appearance of the fracture; specifically, the same fracture patterns could appear as an abduction- or adduction-type fracture, depending on the rotational position of the humerus when the x-ray film was obtained.[114] In 1934, Codman presented a classification system based on the epiphyseal regions of the proximal humerus.[34] This system identifies four possible fracture fragments: greater tuberosity, lesser tuberosity, anatomic head, and shaft. His appreciation that fractures occurred along the lines of the epiphyseal scars formed the basis for Neer's development of his classification system.

Neer Classification

Neer[113,114] proposed his classification of proximal humeral fractures in 1970, and since then it has become the most widely used system in clinical practice[13,64,90,106,141,146] (Fig. 27-8). This system is based on the anatomic relations of the four major anatomic segments: articular segment, greater tuberosity, lesser tuberosity, and the proximal shaft, beginning at the level of the surgical neck. Knowledge of the rotator cuff insertions and the effects of the muscular-deforming forces on the four segments is essential to understanding this classification system. Fracture types are based on the presence of displacement of one or more of the four segments. For a segment to be considered displaced, it must be either displaced more than 1 cm or angulated more than 45 degrees from its anatomic position. The number of

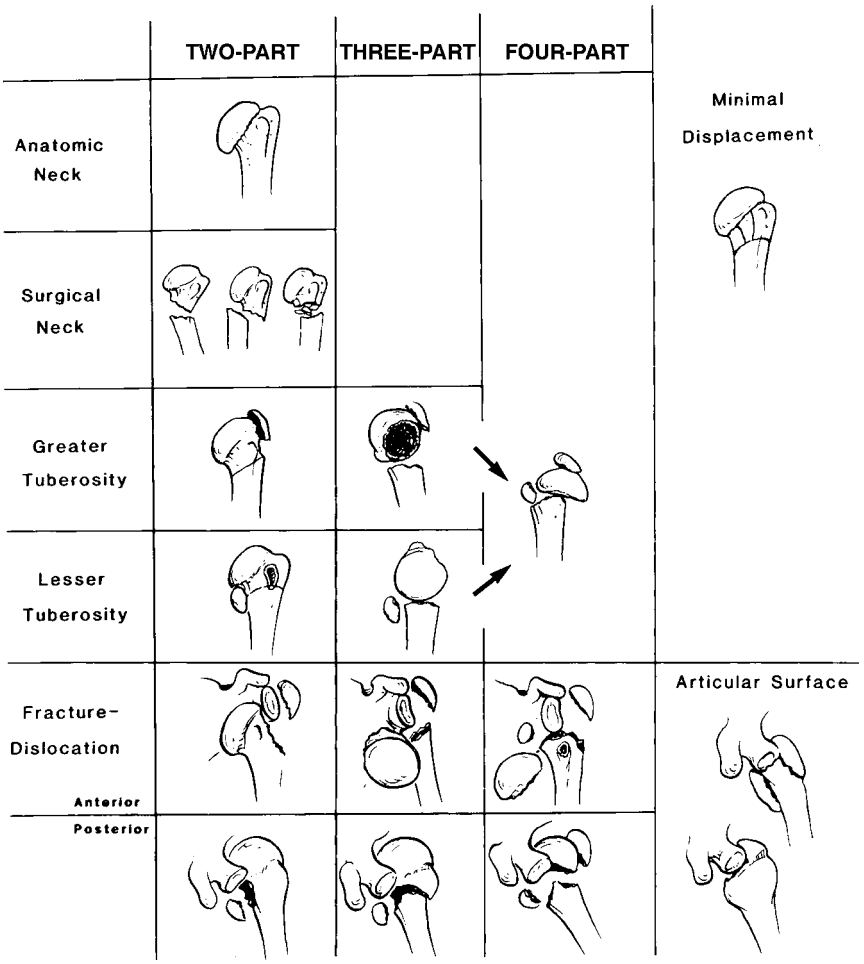


Figure 27-8 The Neer classification of proximal humeral fractures (see text).

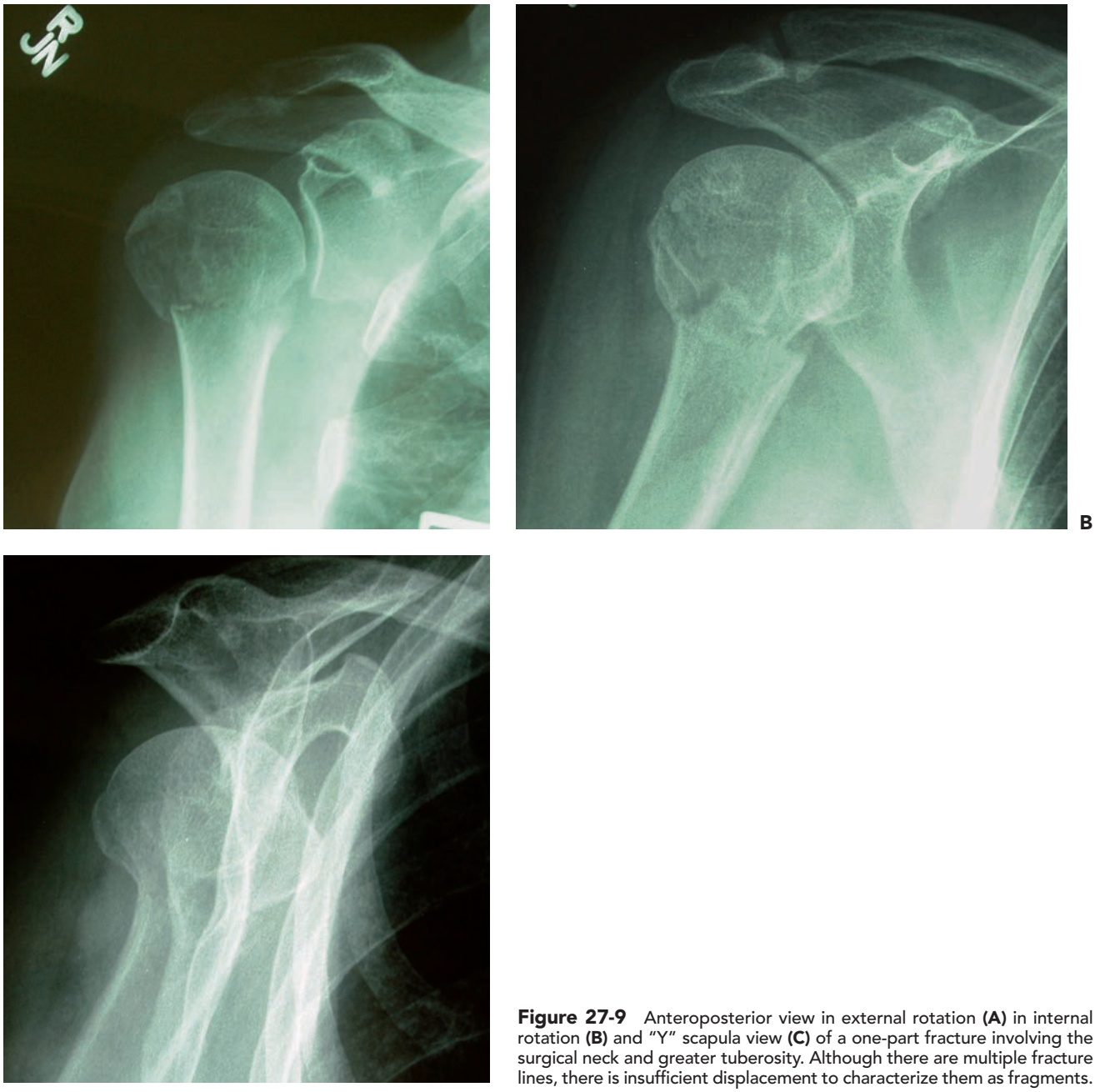


Figure 27-9 Anteroposterior view in external rotation **(A)** in internal rotation **(B)** and "Y" scapula view **(C)** of a one-part fracture involving the surgical neck and greater tuberosity. Although there are multiple fracture lines, there is insufficient displacement to characterize them as fragments.

fracture lines is not important in this classification system. For example, one-part fractures, or minimally displaced fractures, are the most common type of proximal humerus fractures and account for up to 85% of all proximal humerus fractures.[75,116,131] Although these fractures may have multiple fracture lines, they are characterized by the fact that none of the four segments fulfills the criteria for displacement. Hence, they are considered one part or minimally displaced (Fig. 27-9A,B).

Displaced fractures include two-part, three-part, and four-part fractures. A two-part fracture is characterized by displacement of one of the four segments, with the remaining three segments either not fractured or not fulfilling the criteria for displacement. Four types of two-part fractures can be encountered (greater tuberosity, lesser tuberosity, anatomic neck, and surgical neck). A three-part fracture is characterized by displacement of two of the segments from the remaining two nondisplaced segments. Two types of three-part fracture patterns are encountered. The more common pattern is characterized by displacement of the greater tuberosity and the shaft, with the lesser tuberosity remaining with the articular segment. The much less commonly encountered pattern is characterized by displacement of the lesser tuberosity and shaft, with the greater tuberosity

remaining with the articular segment. A four-part fracture is characterized by displacement of all four segments.

Neer also categorized fracture–dislocations, which are displaced proximal humerus fractures: two-part, three-part, or four-part, associated with either anterior or posterior dislocation of the articular segment. Therefore, six types of these fracture–dislocation patterns can occur. Neer also described articular surface fractures that were of two types: impression fractures or head-splitting fractures. Impression fractures of the articular surface most often occur in association with chronic dislocations. As such, they can be either anterior or posterior and involve variable amounts of articular surface.[13] Head-splitting fractures are usually associated with other displaced fractures of the proximal humerus in which the disruption or "splitting" of the articular surface is the most significant component.

Reliability of Neer Classification

As noted, the Neer classification is the most widely used classification system for proximal humerus fractures.[90,106,113,114] Recently, there have been several studies that have examined the reliability of the Neer classification.[1,9,21,86,135,146,151] Among four observers of varying expertise who evaluated a series of 100 proximal humerus fractures, Kristiansen et al.[86] found a low level of interobserver reliability using a condensed Neer classification system. The level of expertise was noted to be an important factor on predicting interobserver reliability. However, this study was limited by various factors. First, a complete trauma series was not used; rather, only anteroposterior (AP) and lateral radiographs were examined. Second, a condensed Neer classification was used that consisted of five categories (one-part fractures, two-part fractures, three-part fractures, four-part fractures, and all other fractures and fracture–dislocations), which were somewhat disparate with respect to fracture type, treatment options, and prognosis. And third, intraobserver reliability (reproducibility) was not assessed.

We conducted a study to assess the inter- and intraobserver reliability of the Neer classification system using the radiographs of 50 proximal humerus fractures.[146] Good-quality trauma series radiographs were available for each fracture, consisting of a scapular AP, scapular lateral, and axillary view. The radiographs were reviewed by an orthopedic shoulder specialist, an orthopedic traumatologist, a skeletal radiologist, an orthopedic resident in the fifth year of training, and an orthopedic resident in the second year of training. The radiographs were reviewed on two different occasions 6 months apart. Interobserver reliability was assessed by comparing the fracture classification determined by the five observers. Intraobserver reliability was assessed by comparing the fracture classification determined by each observer for the first and second reviews. Kappa-reliability coefficients were used to adjust the observed proportion of agreement between or among observers by correcting for the proportion of agreement that could have occurred by chance.

All five observers agreed on the final classification in 32% and 30% of cases for the first and second viewings, respectively. Paired comparisons between the five observers showed a mean corrected reliability coefficient of 0.50 (range 0.37 to 0.62) for both testings, which corresponds to a "moderate" level of reliability. An excellent level of reliability (kappa greater than 0.81) was not obtained for paired evaluation. Attending physicians demonstrated slightly higher interobserver reliability than orthopedic residents. Intraobserver reliability ranged from 0.83 (shoulder specialist) to 0.50 (skeletal radiologist) with a mean of 0.65, which corresponds to a "substantial" level of agreement.

Brien et al.[21] also questioned whether the use of plain films is reliable in the Neer classification of complex proximal humerus fractures. They found only fair intraobserver reliability in a 5-year retrospective analysis of 28 fractures with kappa values ranging from 0.37 to 1.00. Surgical neck fractures faired better than tuberosity fractures. The authors admitted to basing diagnosis on incomplete trauma series, with inclusion criteria of only two roentgenograms at 90-degree angles to each other (rather than the preferred three views). Also, there was no standardized radiographic procedure. They suggested that the addition of routine conventional radiographs and computed tomography (CT) scan may increase reliability.

The hypothesis that a better imaging technique may increase the reproducibility of proximal humerus fracture classification was explored by several authors. Bernstein et al.[9] found findings similar to ours for intraobserver reliability (kappa = 0.64) and interobserver reproducibility (kappa = 0.52) when fractures were classified on the basis of radiographs alone. When CT scans were added to plain radiographs, intraobserver reliability increased slightly (kappa = 0.72), but interobserver reproducibility did not improve (kappa = 0.50). They concluded that because even the shoulder experts had difficulty agreeing on which fragments were fractured, new imaging modalities rather than a new classification system may need to be developed.

Brorson et al.[25] assessed the effect of training on the improvement in interobserver variation of the Neer system. The orthopedic medical staff involved in the teaching was provided with a diagram of the Neer classification system and a 45-minute tutorial based on the original reports of the system. This session was repeated again 2 weeks later. They found formal training in the Neer system improved the mean kappa value in specialists from 0.3 to 0.79.

Sjoden et al.[151] investigated 26 proximal humerus fractures with both plain radiographs and CT. Similar to Bernstein, they found that CT scan together with plain radiographs did not make fracture classification more consistent. Sallay et al.[135] conducted a study for which two groups of observers (experts and nonexperts in shoulder surgery) reviewed the plain radiographs and the three-dimensional CT scans of 12 patients with proximal humerus fractures. Both groups of observers displayed suboptimal

reliability for the identification of displaced fracture fragments. The addition of three-dimensional CT scans did not improve the reliability or reproducibility. Unlike our study,[146] Sallay et al.[135] found that experience did not significantly improve the reliability of identifying specific fracture patterns. They recognized the limitations of their retrospective study and the small number of cases and a lack of standardized radiographs. They believed that it was not the classification system that was poor, but more likely the vague criteria for identifying the fractured segments and determining their displacement and angulation.

Burstein has questioned whether classification systems are useful at all.[30] Neer believes that the confusion is due to a bad use of the classification system that he believes is anatomically correct,[37] rather than to a bad classification system. Yet, Rockwood and even Neer stated that even the most experienced surgeon occasionally is in doubt and has to make the final classification at surgery.[30,37,39,117]

The classification of the fracture is critically important to the decision-making process. A fracture classified as minimally displaced by one observer and as a three-part by another may be treated differently by the two observers. Also, the results of treatment for a particular fracture in the literature may be inaccurate because of the difficulties of determining a reliable and reproducible classification.[39]

AO Classification

The AO group has modified the Neer classification, placing more emphasis on the vascular supply to the articular segment of the proximal humerus. Severity of the injury and risk of osteonecrosis forms the basis of the AO classification system (Fig. 27-10). In this system, it is accepted that if either tuberosity and its attached rotator cuff remain in continuity with the articular segment, the vascular supply is probably adequate. Proximal humerus fractures are separated into three types: extraarticular unifocal, extraarticular bifocal, and articular. Each of these types is further subdivided into different groups based on alignment, degree, and direction of the displacement; presence of impaction; and associated dislocation.

Type A fractures are extraarticular and involve one of the tuberosities with or without a concomitant metaphyseal fracture. Group Al fractures are the extraarticular unifocal tuberosity fractures; group A2 fractures are the extraarticular unifocal fractures with an impacted metaphyseal fracture; and group A3 fractures are the extraarticular unifocal fractures with a nonimpacted metaphyseal fracture. Osteonecrosis is unlikely in type A fractures.

Type B fractures are also extraarticular, but involve both tuberosities with a concomitant metaphyseal fracture or glenohumeral dislocation. Group B1 fractures are the extraarticular bifocal fractures associated with an impacted metaphyseal fracture; group B2 fractures are the extraarticular bifocal fractures with a nonimpacted metaphyseal fracture; and group B3 fractures are the extraarticular bifocal fractures with a glenohumeral dislocation. There is a low risk for osteonecrosis in type B fractures.

Type C fractures are extraarticular and involve vascular isolation of the articular segment. Group C1 fractures are

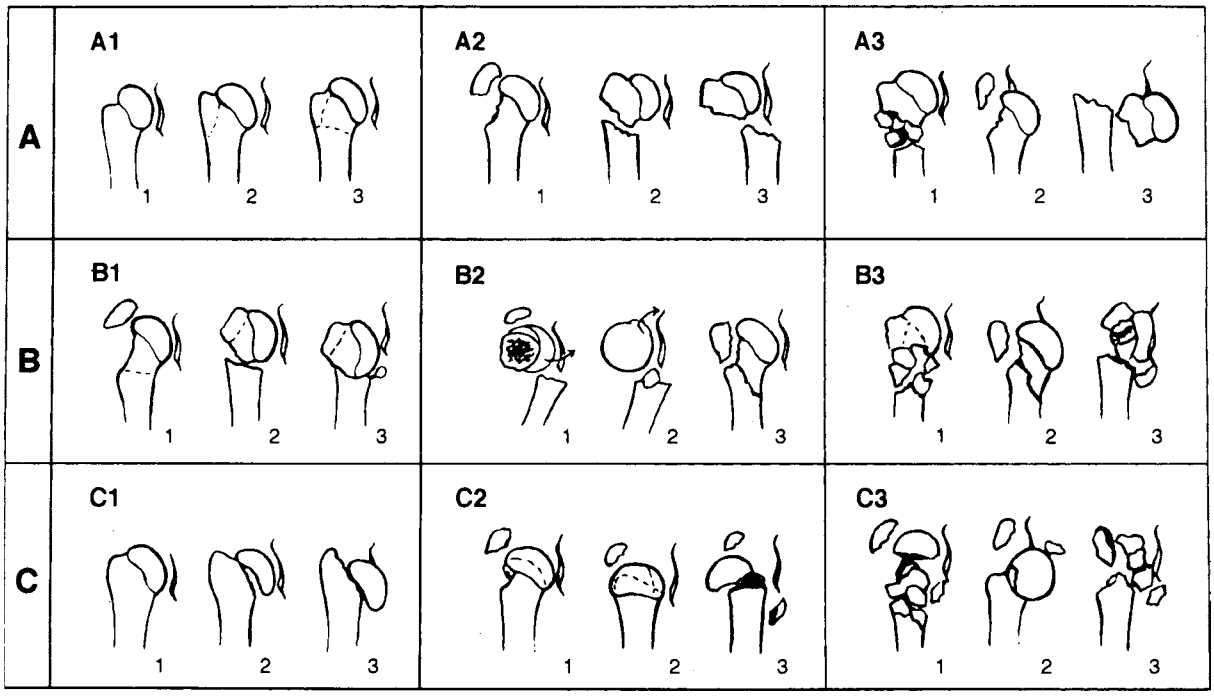


Figure 27-10 The AO classification of proximal humeral fractures (see text).

fractures with slight displacement; group C2 fractures are impacted fractures with marked displacement; and group C3 fractures are associated with a glenohumeral dislocation. There is a high risk of osteonecrosis in this type.

This more complex classification system theoretically should allow development of more detailed guidelines for treatment and prognosis. However, its complexity may actually preclude attaining the widespread utilization that the Neer classification currently commands. Thus far, clinical studies using the AO classification have been quite limited. Recent assessment of its interobserver reliability has not shown it to be significantly better than the Neer system.[78,147] Thus far, no long-term results of treatments based on the AO classification system have been presented. Siebenrock and Gerber's[147] study found that when five experts in shoulder surgery evaluated 95 proximal humerus fractures using simplified versions of both the Neer and AO/Association for the Study of Internal Fixation (ASIF) classification system, there was poor interobserver reliability and acceptable intraobserver reliability for both systems.

Valgus-Impacted

There is a specific type of four-part fracture described that is characterized by a valgus impaction of the humeral head and variable displacement of the tuberosities (Fig. 27-11). This valgus-impacted humeral head fracture pattern does not fit accurately into Neer's classification. The AO classification system classifies these fracture patterns as C2.1- and C2.2-type fractures.[45] However, because of the lower rate of osteonecrosis and the more favorable outcome compared with "classic" four-part fractures, Jakob et al.[79] felt this fracture pattern required special consideration, as will be discussed later.

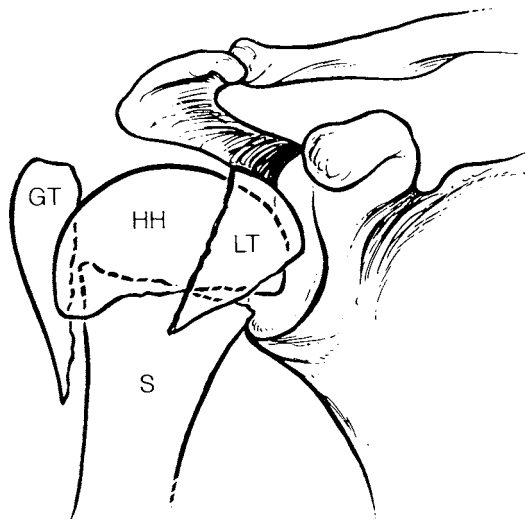


Figure 27-11 Valgus-impacted four-part fracture. The articular segment (*HH*) is impacted and angulated, rather than displaced as in the classic four-part fracture. GT = greater tuberosity; LT = lesser tuberosity; S = the shaft.

Incidence

Proximal humeral fractures account for 4% to 5% of all fractures in adults and less than 1% of children's fractures. Approximately 3% of physeal fractures occur through the proximal humerus.[11,121] Proximal humeral epiphyseal plate fractures can occur any time between birth and physeal closure at approximately 18 years of age. These are most common in adolescents between 10 and 16 years of age (owing to increased sports participation), followed by neonates who sustain birth trauma.[23,44,57,68,172] In patients younger than 17 years of age, Salter-Harris types I and II are most commonly seen with proximal humerus fractures. Salter-Harris type I injuries occur in newborn infants, whereas Salter-Harris type II injuries occur in adolescents.[2,3,44]

Salter-Harris III fractures are rare and usually occur with a dislocation in which the glenoid acts as a wedge to split the epiphysis.[161] Salter-Harris IV fractures are associated with open fractures.[3] No Salter-Harris V fractures have been reported in the literature.[44,138,144] The reason for this fracture distribution can be explained as follows. The proximal humeral epiphysial plate is very active, contributing about 80% of longitudinal humeral growth.[49] It remains open until approximately 19 years of age. The weakest area is distal to proliferating cartilage cells in the "zone of degenerative cartilage," where the cartilage is being converted to bone on the metaphyseal side of the plate.[112] Microscopic studies of fracture specimens in stillborn infants and experimental animals confirm the finding that the fracture through the plate occurs through the hypertrophied cartilage cells adjacent to the zone of provisional calcification, thus avoiding the proliferating cells and causing no harmful effects on growth.[19,44,136] The glenohumeral joint has the most mobility of any joint in the body, which limits the risk for crushing or epiphyseal splitting injuries.[2,112] Lesser tuberosity fractures occur with forced external rotation and abduction, while greater tuberosity fractures occur with dislocations, both being rare injuries.

Proximal humerus fractures account for over 75% of humerus fractures in patients older than age 40. After age 50, women have a much higher incidence than men. An exponential increase occurs after menopause, representing the typical characteristic of an osteoporotic fracture.[5,88,120,131] In patients younger than age 50, high-energy trauma is the most common cause of proximal humerus fractures; after age 50, minimal to moderate trauma is the most common cause.[80] Up to 85% of all proximal humerus fractures are one-part (nondisplaced or minimally displaced) fractures; 15% to 20% of all proximal humerus fractures are classified as displaced. Most two-part fractures are surgical neck fractures, with the majority of these fractures occurring in individuals older than 65 years.[77,165] Although up to 40% of proximal humerus fractures have some involvement of the greater tuberosity,[75,131] isolated two-part displaced greater tuberosity fractures are reported to comprise

less than 2% of all proximal humerus fractures.[77] However, it is believed that this fracture is underdiagnosed and may be more common than the literature suggests.[39] These fractures occur in association with anterior dislocation in 5% to 33% of cases.[62,132,140] Two-part anatomic neck fractures have not been reported to occur in children[44] and constitute only 0.54% of proximal humerus fractures.[77] Isolated lesser tuberosity fractures comprise 0.27% of all proximal humerus fractures and 0.5% of displaced fractures. Two-part lesser tuberosity fracture–dislocations comprise 1.3% of all displaced proximal humerus fractures. Three-part fractures comprise 3% or more of displaced proximal humerus fractures.[77,110,114] Three-part lesser tuberosity fractures occur much less frequently than those of greater tuberosity displacement. Four-part fractures comprise approximately 4% of proximal humerus fractures, and articular surface fractures comprise approximately 3%.[3]

Rating System

There are many different methods currently used to assess shoulder function. To some degree, all shoulder outcome instruments assess pain, function, range of motion, and strength. The problem is that each one emphasizes a different aspect of the shoulder evaluation. Some have a greater emphasis on range of motion, whereas others place more emphasis on pain and still others emphasize function. It is due to these differences that comparing instruments has become difficult if not impossible, and no individual instrument has become universally accepted.

Neer's shoulder grading scale was originally developed to assess shoulder arthroplasty for glenohumeral degenerative joint disease; however, it has been widely used to assess outcome following proximal humerus fractures. Neer used a system based on 100 units: 35 units were assigned for pain, 30 units for function, 25 units for range of motion, and 10 units for anatomy. A score of 89 or higher represents an excellent result; 80 to 88 units is a satisfactory result; 70 to 79 units is an unsatisfactory result; and less than 70 units represents a failure. Each assessment method places varying importance in the areas of pain, range of motion, and function. To facilitate communication between investigators, stimulate multicenter studies, and allow communication of useful and relevant outcome data to physicians, health care organizations, and the general public, a standardized method of assessing shoulder function, regardless of diagnosis, was established by the American Shoulder and Elbow Surgeons (ASES) in 1994.[127] Some of the other scoring systems include the Constant scoring system; the University of California, Los Angeles (UCLA) shoulder rating scale; the Shoulder Severity Index; the Simple Shoulder Test; the Disabilities of the Arm, Shoulder, and Hand Instrument (DASH); and PENN score. It is possible that one assessment system may indicate a good result, whereas another system may indicate a fair or poor result in the same patient. Furthermore, the outcome scores currently used may misrepresent patient perceptions of treatment outcomes. Patients may lack full range of motion and strength—objective measures often emphasized in outcome instruments—yet remain satisfied with a pain-free shoulder and restoration of a functional range of motion.

EVALUATION

Clinical Evaluation

Newborns with an epiphyseal separation of the proximal humerus present with pseudoparalysis with the arm held in extension, swelling at the apex of the shoulder, and occasionally fever. Infection, clavicle fracture, and Erb's palsy are included in the differential diagnosis. Pain with motion of the arm points toward a fracture.[3,22]

In eliciting a history from a patient who has sustained a proximal humerus fracture, determination of the mechanism of injury can be helpful. A child usually gives a history of falling backward onto an outstretched hand with the elbow extended and the wrist dorsiflexed.[3] A loss of consciousness or a history of falls may indicate a cardiac or neurologic cause in the older patient.[42,89]A direct mechanism can occur, such as a blow to the lateral aspect of the shoulder, in both young and older patients. However, in the elderly, the indirect mechanism is much more common and generally involves a fall onto the outstretched arm. In younger patients, this mechanism most commonly results in a dislocation. The indirect mechanism is usually associated with a greater degree of fracture displacement than the direct mechanism. Other indirect causes of proximal humerus fractures are seizures or electroconvulsive therapy without the use of muscle relaxants; both are often associated with posterior dislocations.[89]

The symptoms and signs associated with proximal humerus fractures can be quite variable. However, they most often correlate with the degree of fracture displacement and comminution. Pain, especially with any attempts at shoulder motion, is almost always present. Inspection of the shoulder usually reveals swelling and ecchymosis. The patient should be instructed that over the first 4 to 5 days following injury, the ecchymosis that develops may extend distally into the arm and forearm or even to the chest wall and breast area (Fig. 27-12).

Palpation of the shoulder will usually reveal tenderness about the proximal humerus. Crepitus may be evident with motion of the fracture fragments. The entire upper extremity should be examined. A fall on the outstretched arm can also result in a fracture of another area such as a distal radius fracture. The chest should also be examined, because rib fractures may also occur from a fall.[61,65,124,166]

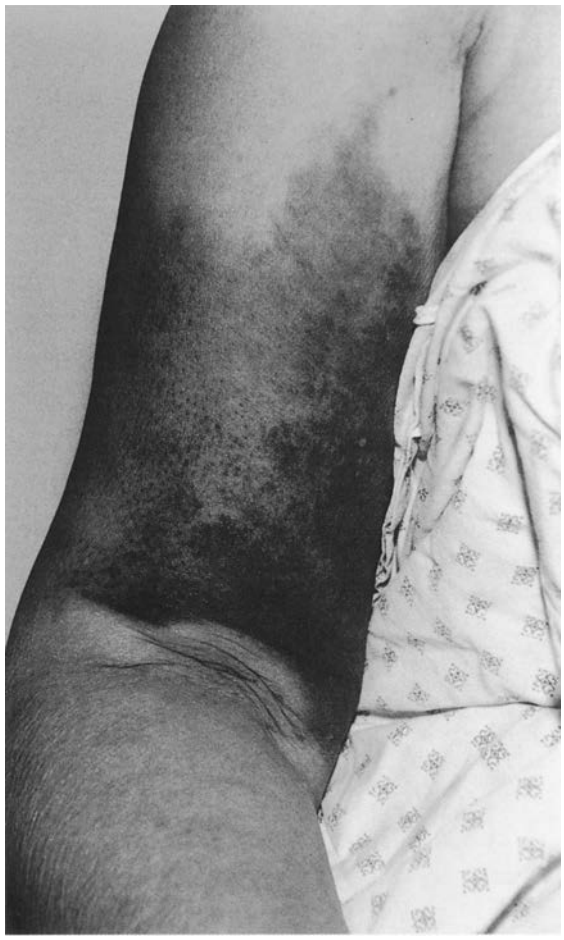


Figure 27-12 A 72-year-old woman, 2 days following closed reduction of a two-part greater tuberosity fracture–dislocation; the area of ecchymosis has extended to the elbow.

Assessment of fracture stability is an essential part of the examination. The humeral shaft should be gently rotated internally and externally as the proximal portion of the humerus is palpated. If the proximal and distal portions move as a unit, the fracture is stable; however, motion or crepitus is consistent with an unstable or less stable fracture pattern.

Essential to the clinical evaluation of the patient with a proximal humerus fracture is a complete neurovascular examination of the involved upper extremity. Associated axillary artery and brachial plexus injuries have been reported,[69,98,152,154,173] especially with fracture fragments displaced medial to the coracoid process. A 45% incidence of nerve injury has recently been reported.[46] Fracture–dislocations also increase the incidence of neurovascular injury.[123] The most commonly injured peripheral nerve is the axillary nerve.[13] Both sensory and motor testing should be performed to evaluate axillary nerve function. This may be difficult in the setting of acute injury. Therefore, serial examinations should be performed.

Radiographic Evaluation

One of the most important components of the evaluation of proximal humerus fractures is the determination of the position of the fracture fragments and the degree of displacement. Thus, an adequate radiographic evaluation is mandatory.

Trauma Series

The cornerstone of the radiographic evaluation of proximal humerus fractures is the trauma series (Fig. 27-13). The trauma series consists of AP and lateral views of the shoulder, obtained in the plane of the scapula, and an axillary view. Fracture classification and treatment decisions are generally based on these three radiographic views. Each view contributes information obtained from three different perpendicular planes.[2,13,35,66,141] The scapular AP view offers a general overview of the fracture and is usually evaluated first. This view should be made perpendicular to the scapular plane (as differentiated from the plane of the chest), which requires angling the x-ray beam approximately 40 degrees in a mediolateral direction. This compensates for the position of the scapula on the chest wall. It will demonstrate the glenoid in profile as well as the true glenohumeral joint space. In the undislocated shoulder, it shows the humeral head to be clearly separated from the glenoid.

At least one view obtained at 90 degrees to the scapular AP is required for assessment of proximal humerus fractures and fracture–dislocations.[130] This orthogonal view provides important information about angulation and displacement of the fracture fragments[13] and the presence of an associated dislocation.[17,20,104,172] Both the scapular lateral and axillary views are oriented orthogonally to the scapular AP and fulfill the criteria for a second projection. However, the trauma series generally includes all three views.

The scapular lateral, also known as the scapular "Y," can provide important information not evident on a scapular AP view.[49,104,134,172] This view is a true lateral of the scapula, with the x-ray beam passing parallel to the spine of the scapula. This view is taken with the patient standing. It can be obtained with the involved upper extremity immobilized in a sling and does not require any movement of the extremity and, as such, does not add to patient discomfort. The scapular lateral assists in delineating the position of the humeral head relative to the glenoid and is particularly useful in showing posteriorly displaced fragments as well as shaft displacement.

The axillary view also permits assessment of the glenohumeral relation. This is generally obtained with the patient supine. The arm must be positioned in at least 30 degrees of abduction. In the acute setting, positioning is often performed by the physician. The x-ray plate is placed above the shoulder, and the beam is directed to the plate from a caudad position. The axillary view can also be useful in identifying fractures of the glenoid rim, posterior

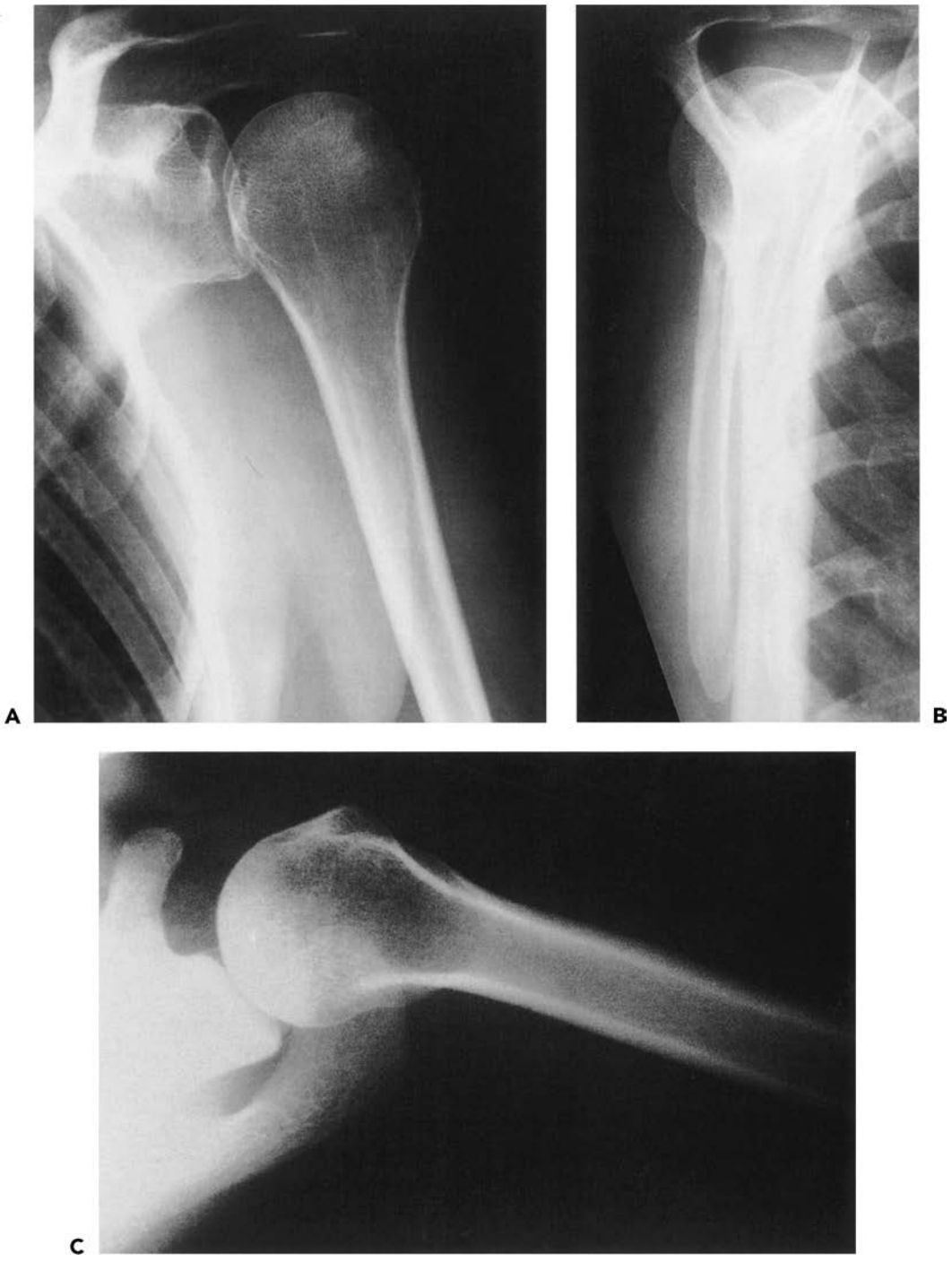


Figure 27-13 The trauma series of radiographs includes an **(A)** anteroposterior view and **(B)** lateral view in the plane of the scapula, and **(C)** an axillary view.

displacement of the greater tuberosity, medial displacement of the lesser tuberosity, and humeral head articular impression fractures.[42,66,119,130,167]

The relative efficacy of these different views has not been studied extensively. Silfverskiold et al.[148] reported in their prospective study that the scapular lateral view was more sensitive than the axillary view in detecting shoulder dislocations. They did not study fractures specifically. In 92% of their 75 cases, however, the scapular lateral and axillary views resulted in the same diagnosis.

We conducted a study in which the trauma series radiographs of 50 proximal humerus fractures were used to assess the relative contributions of the scapular lateral and axillary radiographs to fracture classification using the Neer system.[145] The radiographs were reviewed by five different orthopedic surgeons with varying levels of experience and

expertise. In the first viewing, radiographs were reviewed and classified in the following sequence:

1. After review of scapular AP view alone
2. After review of scapular AP and lateral views
3. After review of scapular AP, lateral, and axillary views

A second viewing of the same 50 cases was performed 6 months later in a changed sequence:

1. After review of scapular AP view alone
2. After review of scapular AP and axillary views
3. After review of scapular AP, axillary, and scapula lateral views

For the cumulative experience of these five observers, review of the scapular AP and axillary views achieved a final classification in 99% of cases. However, after review of the scapular AP and lateral, the final classification was achieved in only 79% of cases ($P < 0.05$). These results indicate that when combined with the scapular AP radiograph, the axillary view contributes significantly more information than the scapular lateral radiograph in determining fracture classification. Posterior displacement of 1 cm of the greater tuberosity is missed 25% of the time when only AP and lateral scapular views are used alone. Additionally, we also reviewed the accuracy of the axillary projection to determine fracture angulation in experimentally produced surgical neck fractures. We found that it was not reliable projection for assessing the degree of fracture angulation primarily as a result of the variability in arm position (abduction, flexion, or extension) when the radiograph was obtained.[150]

Additional Radiographic Views

If any of these three views of the trauma series is inadequate, it should be repeated. Usually, the fracture can be evaluated and treated based on this set of radiographs. Additional radiographic views may be helpful and have been advocated by others.

The apical oblique view[58] is obtained by directing the x-ray beam through the glenohumeral joint at an angle of 45 degrees to the plane of the chest wall and angled 45 degrees caudally. When compared with the scapular lateral view, it provides additional useful information in the evaluation of proximal humerus fractures and fracture–dislocations, specifically in demonstrating dislocations and posterolateral humeral head compression fractures.[84,128] However, it has not become as widely accepted as the scapular lateral view.

Modified axillary views, such as the Velpeau axillary lateral,[17] the Stripp axillary lateral,[76] and the trauma axillary lateral,[160] have been described. These views permit an axillary lateral to be obtained without removing the injured arm from the sling. The Velpeau axillary lateral is probably the most commonly used, taken with the patient leaning backward approximately 30 degrees over the x-ray table. The x-ray cassette is placed beneath the shoulder on the table, and the x-ray beam passes vertically from superior to inferior through the shoulder joint. Although this view has the benefit of avoiding the need to position the injured extremity, we prefer the standard axillary view because it offers less distortion and bony overlap and thereby provides more useful information.

Other Diagnostic Modalities

Computed tomography scans of proximal humeral fractures and fracture–dislocations may be indicated when the trauma series radiographs are indeterminate. CT scans have been recommended to evaluate the rotation of fragments, the degree of tuberosity displacement, as well as articular impression fractures, head-splitting fractures, and chronic fracture–dislocations.[13,14,31,81,82,92] Castagno et al. reported a small series of 17 patients in whom CT scans of acute proximal humeral fractures demonstrated important information not evident on plain radiographs.[31] Also, spiral CT with three-dimensional and multiplanar reconstructions was reported to provide additional information, compared with standard x-rays, in 10 patients with three- and four-part fractures. However, in this study, standard radiographs did not include an axillary view.[81]

As mentioned earlier in the classification and reliability section, CT does not seem to improve interobserver reliability and fracture classification.[135,151] In our experience, CT scans are most helpful in the evaluation of chronic fracture–dislocations, specifically to identify the size and location of humeral head impression defects and the degree of secondary glenoid changes[146] (Fig. 27-14).

Another imaging modality to consider is magnetic resonance imaging (MRI). MRI provides information about associated soft tissue injuries of the rotator cuff, biceps ten-

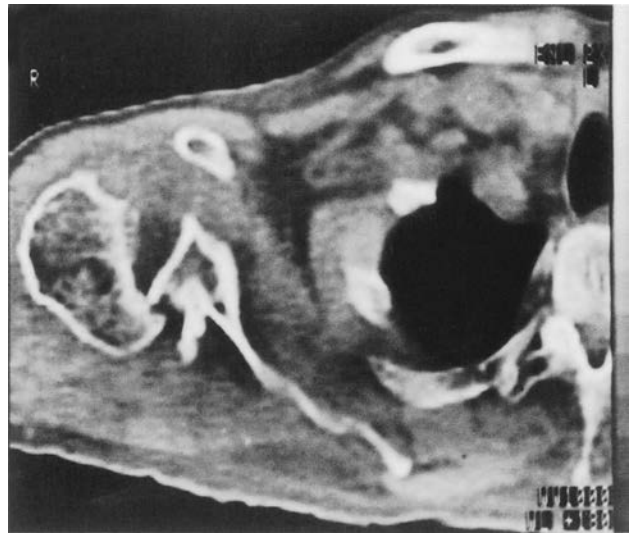


Figure 27-14 Computed tomography scan of the right shoulder in a 56-year-old man with a chronic posterior dislocation of the right shoulder. The image clearly shows the impression fracture of the anteromedial portion of the humeral head, as well as the amount of the articular surface involvement. The calcification about the posterior glenoid neck provides further evidence of the chronic nature of dislocation.

don, and glenoid labrum, which may be helpful in the management of these patients. However, cost benefit issues have to be considered carefully. Thus far, there have been no studies performed to support its routine use.

Vascular Injury and Indications for Arteriogram

The axillary artery lies just anterior and medial to the prox mal humerus.[118] Vascular injury following fracture–dislocation of the humerus is uncommon; it is rare in anterior dislocation without associated fracture; and it is extremely rare in closed humeral neck fractures, despite the fracture's proximity to the artery.[51,94,99,109,113,153,159] Morris et al. identified no vascular injuries associated with proximal humeral fractures in a series of 220 acute arterial injuries.[109] Neer reported on 117 displaced proximal humerus fractures over a 15-year period and found no vascular complications.[113] The literature documents only 16 cases of axillary artery injury from proximal humerus fractures.[94,99,102,103,109,153,159,173]

In pure anterior dislocations, the axillary artery can be damaged in its second part by avulsion of the thoracoacromial trunk or in its third part from avulsion of the subscapular and circumflex humeral vessels (with subsequent linear tears in the artery and intraluminal thrombosis). The mechanism of arterial injury in a pure dislocation is thought to be secondary to tethering of the axillary artery by the subscapular and circumflex arteries, thus accounting for the injuries primarily occurring in the third part.[107] The pectoralis minor muscle may act as a fulcrum that puts the artery on stretch and then pinches the artery with dislocation.[26] The artery may also be fixed to a scarred joint capsule from previous injury or surgery, or atherosclerotic vessels may be present. Both factors increase the risk of vascular injury. Excessive force during reduction may also cause vascular injury.[51]

Although rare, the possibility of axillary arterial injury should be considered in proximal humerus fractures with severe medial displacement of the shaft.[157] The mechanism of arterial injury may be through direct injury by sharp fracture fragments, thrombosis from vessel contusion, or damage from violent stretching or avulsion of the artery with shoulder hyperabduction, especially in fragile atheromatous vessels. Vascular spasm, although exceedingly rare, may occur from kinking.[105]

Although uncommon, when vascular complications do occur, they represent a true emergency.[137] Early recognition and treatment of vascular injuries play an important role in the eventual outcome: If an arterial repair is done in the "golden" period, within 12 hours of injury, a reasonable chance of success can be expected.[168] A careful neurologic and vascular examination is necessary at initial presentation. Axillary arterial injuries are obvious when clinical findings include an expanding hematoma, absent distal pulses in a cold, pale extremity, or the presence of a pulsatile painful mass with a bruit (in a pseudoaneurysm).[168] However, symptoms are often vague and nonspecific, particularly when ischemic changes are progressive. Because of the tremendous collateral circulation around the shoulder, peripheral pulses, initially weak, may return completely over time, despite axillary artery disruption. This may mask an injury and falsely lead the examiner to consider arterial spasm as the cause of the clinical findings. Distal pulses are reported to be palpable in 27% of patients with major arterial injuries about the shoulder.[42] Therefore, a careful assessment of peripheral pulses must be performed.[6] If a patient with a fracture or fracture–dislocation of the shoulder has no pulse, a pulse that comes and goes, or a pulse of lower volume, further investigation is necessary, even if the pulse returns to normal after manipulation or over time.[54] Any suspicion of a vascular injury requires a Doppler examination to determine both the magnitude and quality of the arterial signal. Arteriography should be performed immediately when the Doppler examination or clinical findings suggest arterial compromise.[6,173] Late signs of vascular complications, including false aneurysms and acute ischemia with gangrene, can appear weeks, months, or even years after the initial injury.[6,10,51,54,71,94,99,109,153,155] Thus, angiography is recommended when there is any clinical suspicion of a vascular injury (Fig. 27-15). When an arterial injury occurs in association with a proximal humeral fracture or fracture–dislocation, the fracture should be anatomically reduced and stabilized with internal fixation before vascular repair, to protect the vascular repair so that additional

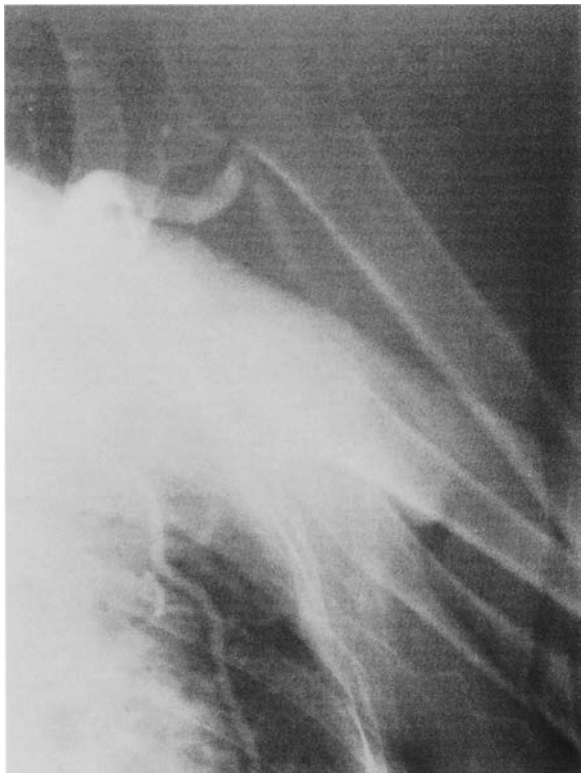


Figure 27-15 Angiogram showing the humeral circumflex vessels pinched in the fracture site, with subsequent tenting of the axillary artery and obstruction of flow after closed reduction of a two-part proximal humerus fracture.

fracture manipulation will not be necessary after repair.[173] However, it has also been suggested that the order of treatment (reperfusion vs. osteosynthesis) should be dictated by the degree of ischemia.[157]

Treatment of Physeal Fractures

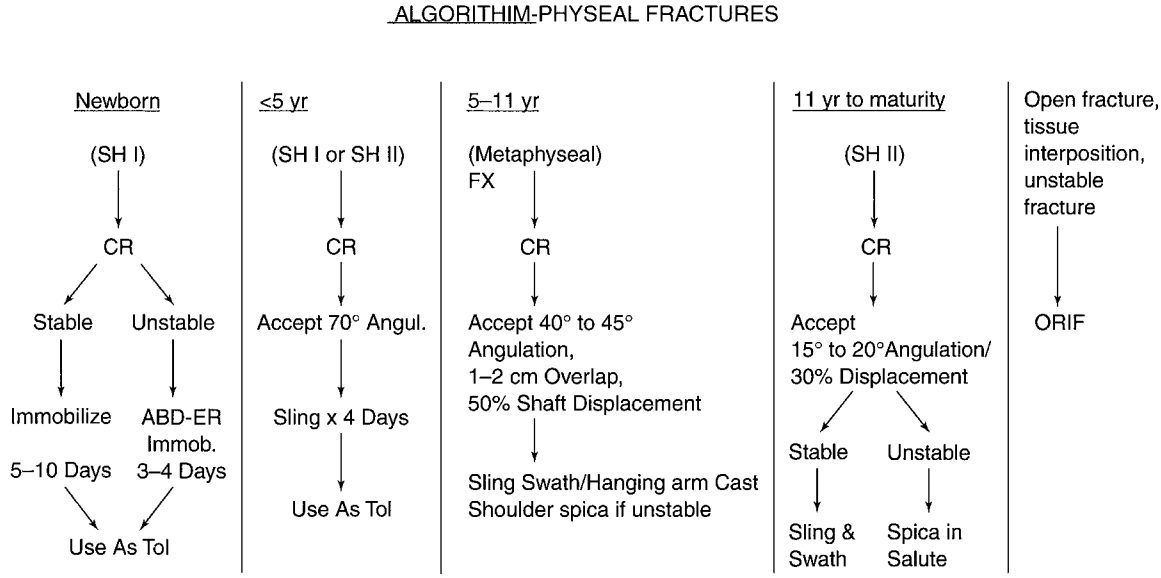
Most proximal humeral physeal fractures can be treated nonoperatively, even when there is significant displacement, because of the great remodeling potential[2,8] (Algorithm 27-1). As a general rule, the younger the patient is, the greater the potential is for remodeling and the greater the initial deformity is that can be accepted. Neer-Horowitz grade I and II fractures do exceptionally well with sling and swath treatment without reduction, because of the remodeling potential of the proximal humeral physis. Neer-Horowitz grade III and grade IV fractures usually require reduction, but may be left with up to 3 cm of shortening or residual angulation because this is often clinically insignificant.

In newborns, most fractures are Salter-Harris type I, for the physeal plate is less resistant to trauma than the bone, joint capsule, and ligaments. If there is marked displacement, a closed reduction can be performed by applying gentle longitudinal traction with the shoulder in 135 degrees of abduction, 30 degrees of forward flexion, and neutral rotation, and directing a posterior force on the shaft.[18] Ultrasound can be used to evaluate the reduction. The prognosis is excellent, even if an anatomic reduction is not fully achieved. If the fracture is stable, the arm can be immobilized against the chest for 5 to 10 days, then used as tolerated. Unstable fractures should be maintained in abduction and external rotation for 3 to 4 days until callous forms.

Salter-Harris type I fractures, and less frequently Salter-Harris type II fractures, occur in children younger than age 5. Closed reduction may be necessary, but up to 70 degrees of angulation with any displacement is acceptable because of the extensive remodeling that is possible in this young age.[112] The arm is held in a sling for 4 days followed by progressive activity.

In the 5- to 11-year age group, metaphyseal fractures are most common because the rapid remodeling in this area results in some degree of weakening. Closed reduction may be necessary, but bayonet apposition with 1 to 2 cm of overlap, 40 to 45 degrees of angulation, or displacement of one-half the width of the shaft are acceptable.[57] Stable fractures are immobilized in a sling and swath; a hanging arm cast is used occasionally in older children. Rarely, a spica cast is used for 2 to 3 weeks in unstable fractures to maintain the arm in the salute position. When callous is evident, the arm is slowly brought down to the side and immobilized in a sling and swath. After 3 to 4 weeks, the fracture should be healed and progressive range of motion exercises are begun. Strengthening of the rotator cuff, trapezius, and deltoid are added as range of motion progresses.[138]

From age 11 until skeletal maturity, there is less remodeling potential than in younger children, so less displacement and angulation is acceptable. The fractures encountered are more frequently Salter-Harris type II, with some Salter-Harris type I fractures. Treatment is by closed reduction with 15 to 20 degrees of angulation and less then 30% displacement considered acceptable. A stable fracture is treated with a sling and swath for 2 to 3 weeks, followed by progressive range-of-motion exercises; an unstable fracture



ALGORITHM 27-1

KEY: SH, Salter-Harris; CR, closed reduction; ABD, abduction; ER, external rotation; FX, fracture; ORIF, open reduction internal fixation; ROM, range of motion; AROM, active range of motion; AAROM, active assisted range of motion; PROM, passive range of motion; AVN, avascular necrosis; IR, rotator cuff tear; PP, percutaneous pinning; As Tol, as tolerated use.

may need immobilization in a shoulder spica cast with the arm in the salute position.[138]

The indications for operative treatment of physeal fractures of the proximal humerus are not well defined; many factors must be considered. Growth-remaining charts may be of benefit to determine if the patient has sufficient potential to remodel a significantly displaced fracture. The literature supports nonoperative treatment, even for markedly displaced fractures,[2] because the functional outcomes have been satisfactory even in the presence of limitation of motion and persistent deformity.[29] The literature also supports closed reduction and pinning or open reduction and internal fixation in older children with markedly displaced fractures and limited remodeling potential, in whom an acceptable reduction cannot be maintained.[156]

TREATMENT

In this chapter we will focus on the nonoperative management of proximal humerus fractures. For some fracture types it is the preferred treatment approach; for others it will only be acceptable under special circumstances. This will be discussed as each fracture type is reviewed.

The indication for operative versus nonoperative management of adult proximal humerus fractures is determined by numerous factors including the patient's physiologic age, arm dominance, associated injuries, fracture type, degree of fracture displacement, and bone quality. The surgeon's knowledge and skill will greatly affect the functional outcome. He or she must also have the ability to accurately diagnose the fracture, based on an understanding of the relevant anatomy and interpretation of complete radiographs. The patient's general medical condition and ability to undergo major surgery as well as to comply with an intensive rehabilitation program are of paramount importance in developing a management plan.[42]

One-Part Fractures: Adult

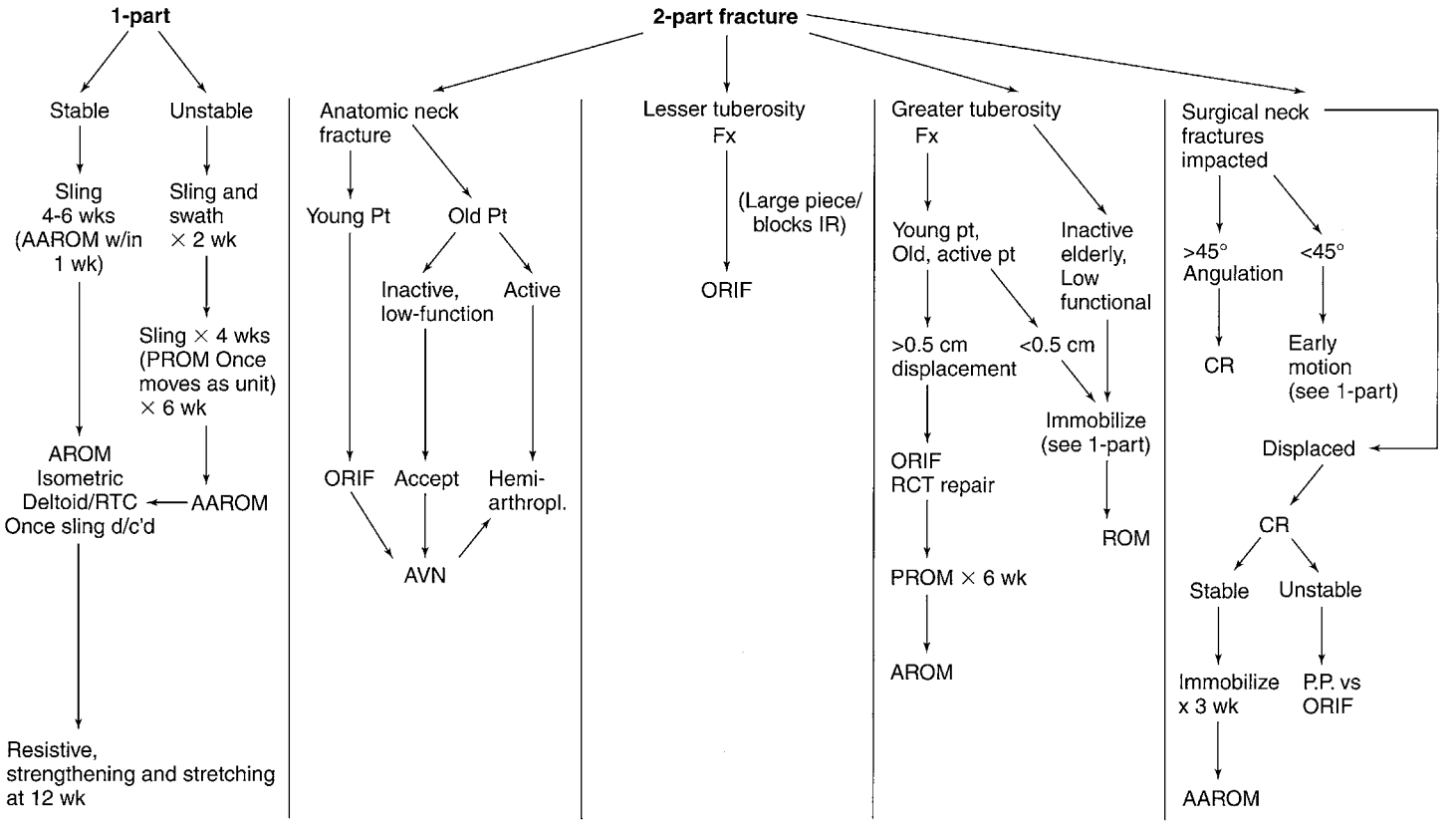
Minimally displaced fractures account for over 80% of all proximal humerus fractures.[114] They are often referred to as "one-part" fractures, based on Neer's four-segment classification. By definition, although there may be many fracture lines, there is no significant displacement of any segment (Algorithm 27-2). However, it is essential to confirm the stability of the fracture. Many of these fractures are impacted and, with rotation of the humerus, the proximal humerus and shaft move together as one unit. This is a stable fracture, and an early range-of-motion program is appropriate. However, some minimally displaced fractures are not impacted, and rotation of the humerus indicates that the proximal segments and shaft do not move as a unit. This fracture is not stable and requires a period of immobilization until sufficient healing has occurred. Once clinical stability is present, as evidenced by movement of the head and the shaft as a unit, range-of-motion exercises can be initiated.

For minimally displaced stable fractures, we prefer immobilization in a simple arm sling. Padding should be placed in the axilla and around the elbow in the sling to prevent skin maceration. Elderly patients must be watched carefully for skin problems, even with use of the simple sling. Swelling and ecchymosis add to the risk of skin problems. When the humerus does not move as a unit, sling and swath immobilization may be preferred. This consists of a standard arm sling with a 6-in. elastic (Ace) bandage used as a swath. The Ace bandage is secured to the sling with safety pins. With a sling and swath of this type, padding should be used in the axilla and around the elbow as described. The swath should be removed two to three times each day to check the skin about the elbow and to allow elbow, wrist, and hand range of motion. In addition, the arm can be gently abducted by gravity to check the skin and axilla.

With minimally displaced fractures that are stable, patients should be started on a range-of-motion program as soon as the initial discomfort subsides. Within 1 week after injury, range of motion therapy should begin, preferably under the supervision of an occupational or physical therapist. This consists of active elbow, wrist, and hand exercises in combination with assisted shoulder range-of-motion exercises. These are begun in the supine position for forward elevation, external rotation, and internal rotation to the chest. Therapy can be supervised in one or two weekly sessions and performed three to five times daily at home by the patient. The patient's arm is maintained in a sling for 4 to 6 weeks. The sling is discontinued when clinical and early radiographic evidence of union is confirmed. At that time, an active range-of-motion program is begun, starting in the supine position, with gradual progression to the sitting position. Isometric deltoid and rotator cuff strengthening exercises are initiated at the same time. When a reasonable range of active shoulder motion is achieved, isotonic resistive exercises for deltoid and rotator cuff muscles are added. Stretching exercises are also added. Approximately 12 weeks after fracture, a more vigorous stretching program is begun.

The results of treatment of minimally displaced proximal humerus fractures, using immobilization and early range of motion, have been generally reported as good to excellent.[33,87,90,100,106,114,116,171,172] In our prospective study of patients with one-part stable proximal humerus fractures treated with the aforementioned standardized protocol of short-term immobilization combined with an early range-of-motion program (mean follow-up of 22 months), recovery of range of motion was generally good. Patients regained 87% of forward elevation, 79% of external rotation, and 89% of internal rotation, compared with the opposite shoulder. However, recovery of motion did not necessarily correlate with functional outcomes. Unlike previous studies, we found a high incidence of residual shoulder pain (67%), with a significant number (27%) of patients describing

Adult proximal humerus fracture algorithm



ALGORITHM 27-2

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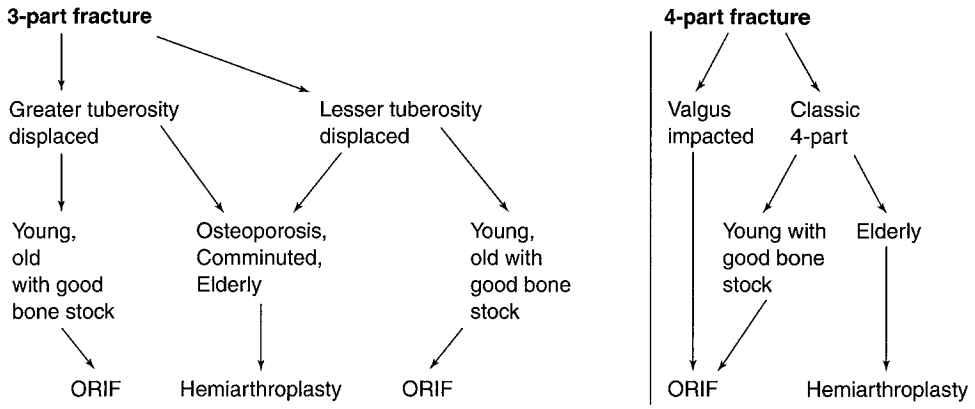


ALGORITHM 27-2 (*continued*)

the pain as moderate to severe. Moreover, almost 30% of patients required some form of medication for pain relief.

In this series, almost two-thirds (65%) of patients reported some limitation of function, with 42% reporting a significant restriction of overall function. This primarily affected activities of daily living or inability to sleep on the affected side (40%), inability to carry a package at the side (46%), and inability to use the hand over the head (51%).[85]

A similar rehabilitation program is utilized for minimally displaced fractures that do not move as a unit. The shoulder is immobilized until it moves as a unit (clinically stable), although active range of motion of the elbow, wrist, and hand can be initiated immediately. This is usually no longer than 2 to 3 weeks. Gentle assisted range-of-motion exercises of the shoulder are initiated at that time. Range of motion regained with these fractures may be slightly less than with stable, minimally displaced fractures. However, the vast majority of patients regain a good functional range of motion.[172] Overly aggressive early passive range of motion, or active range of motion before healing, increases the risk of healing complications.[42]

Displaced Fractures: Two-Part Fractures

Two-part fractures involve displacement of one of the four segments. Therefore, four different fracture patterns are possible: anatomic neck, greater tuberosity, lesser tuberosity, and surgical neck. Even though surgical neck fractures are the most common fracture pattern, the treatment approach for all four types will be discussed.

Anatomic Neck

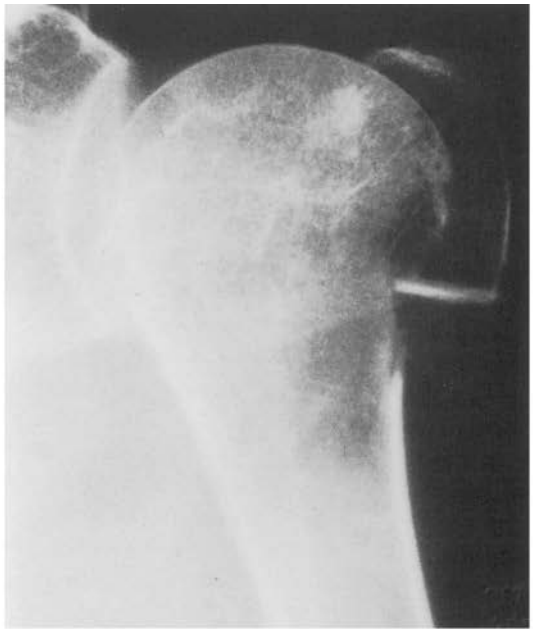
A displaced anatomic neck fracture in the absence of tuberosity displacement is quite rare. It may be difficult to recognize on standard radiographic views and a CT scan may be necessary. The problem with this fracture is the high risk of osteonecrosis because of disruption of the intra- and extraosseous blood supply to the humeral head. A true anatomic neck fracture occurs medial to the rotator cuff insertion and, therefore, is devoid of soft tissue or bony attachments. As a result, these fractures are difficult to treat by closed reduction. Open reduction and internal fixation (ORIF) is preferred, with the goal of avoiding prosthetic replacement in young patients. If a displaced anatomic neck fracture occurs in an elderly patient, a choice has to be made between open reduction and internal fixation (and the risk of nonunion, malunion, and osteonecrosis) and primary hemiarthroplasty. Malunion in the absence of osteonecrosis may be compatible with a good functional outcome. However, if osteonecrosis occurs with collapse and articular incongruity resulting in significant pain, then delayed hemiarthroplasty may be necessary. We prefer primary hemiarthroplasty for the active elderly patient whose functional requirements would be better served by early prosthetic replacement and initiation of a rehabilitation program to regain range of motion. If the articular surface is comminuted, then the fracture is similar to a head-splitting fracture, and primary hemiarthroplasty is preferred. Unfortunately, these fractures are so uncommon that the reported experience with either operative or nonoperative management is not sufficient to support broad conclusions.

Lesser Tuberosity

Two-part lesser tuberosity fractures in the absence of posterior dislocations are uncommon. When encountered, they may be found in association with nondisplaced surgical neck fractures. The fragment is displaced medially as a result of the pull of the subscapularis muscle. This invariably results in some separation of the anterior portion of the rotator cuff. However, these fractures are of minimal clinical significance unless the fragment is large and includes a significant portion of the articular surface.[111] In this situation, internal rotation may be blocked. It is often difficult to determine the size of the fragment from standard radiographs. A CT scan may be very helpful in this situation. If a large fragment is involved, open reduction and internal fixation is indicated.

Greater Tuberosity

Two-part greater tuberosity fractures are relatively common and can be the source of significant disability. The greater tuberosity fragment is usually displaced superiorly into the subacromial space if the supraspinatus is involved, or posteriorly if the infraspinatus or teres minor is the primary deforming force (Fig. 27-16). The degree and direction of displacement indicate that a longitudinal tear of the rotator cuff is also present. Displaced greater tuberosity fragments will often block abduction or external rotation by impinging on the underside of the acromion or posterior glenoid, respectively. Open reduction and internal fixation of the fragment and repair of the rotator cuff tear is usually the preferred treatment approach.[7,48,114] Although the criterion for displaced fractures is 1 cm, greater tuberosity fractures appear to be different from other proximal humerus fractures. Less than 1-cm displacement may be problematic, particularly when the displacement is superiorly into the subacromial space. Open reduction and internal fixation has been required in 20% of patients when the displacement was between 0.5 and 1 cm.[104] Concomitant subacromial spurs may exacerbate impingement, even when the tuberosity is displaced less than 1 cm. It is important to include posterior as well as superior displacement in the indication for open reduction, for posterior displacement



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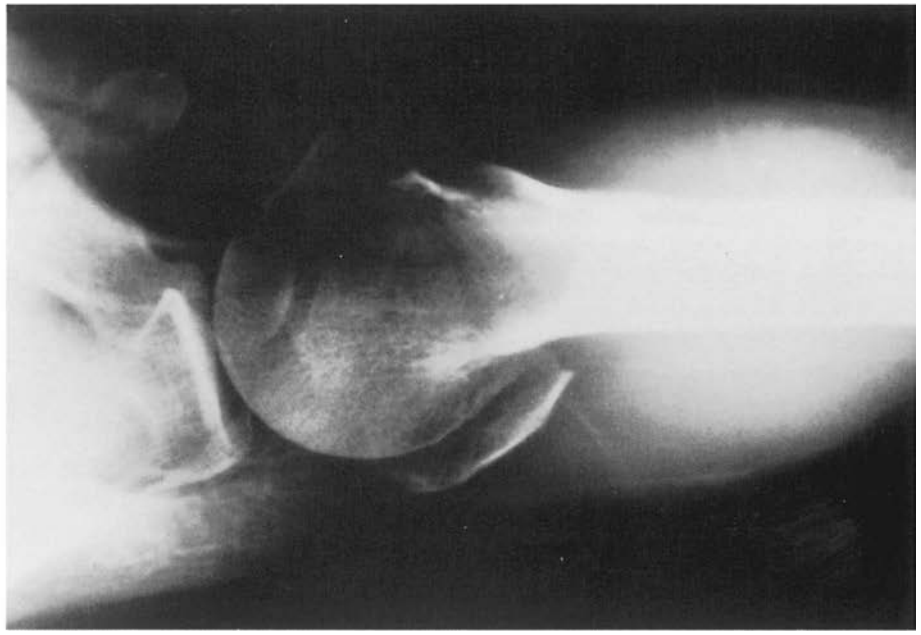

B

Figure 27-16 **(A)** Anteroposterior (AP) and **(B)** an axillary view of a greater tuberosity fracture. Superior displacement into the subacromial space is noted on the AP view, whereas a significant amount of posterior displacement onto the articular surface of the humeral head is best seen on the axillary view.

may also lead to functional deficits, especially in external rotation. Posterior greater tuberosity displacement is best assessed on the axillary radiograph.[56,104]

Surgical Neck

Two-part surgical neck fractures are quite common in the elderly. In these fractures, the shaft may be completely displaced and pulled medially by the deforming force of the pectoralis major, or it may be impacted and angulated at the surgical neck area. Each type of fracture pattern requires a different treatment approach. Treatment depends on fracture stability and the displacement pattern. Posterior angulation is more tolerable than varus or anterior angulation.[42] Impacted fractures with less than a 45-degree angulation may be treated nonoperatively with early motion. Impacted fractures with greater than a 45-degree anterior angulation may limit forward elevation. Therefore, disimpaction, followed by reimpaction to achieve better alignment, should be considered in active patients.

Closed Reduction

When there is complete medial displacement of the shaft, achieving an acceptable functional outcome will require reduction of the fracture. We prefer to perform a closed reduction under anesthesia (regional or general) to obtain better muscle relaxation. The maneuver to achieve closed reduction requires adduction and flexion of the arm across the chest to relax the pull of the pectoralis major. The shaft is then displaced proximally for impaction into the proximal humeral fragment. In thin patients, the proximal fragment can be stabilized manually to enhance impaction. We use fluoroscopy for this maneuver, not only for the initial evaluation of the reduction but, more importantly, to evaluate the stability of the reduction. If the reduction is stable, the arm can be placed in Velpeau-type immobilization (with appropriate padding) with the arm across the chest. Previously, immobilization was continued for approximately 3 weeks; however, more recently we have decreased our time for immobilization to 1 week. Similarly, Hodgson et al.[73] prospectively compared immediate physiotherapy (within 1 week) with delayed physiotherapy (after 3 weeks) in 86 patients with two-part fractures and noted less pain and better shoulder function in the immediate physiotherapy group at 16 weeks. However, by 52 weeks there was no statistical difference between the groups. Radiographs should be repeated during the first week to be certain displacement has not occurred. When there is radiographic evidence of healing or the fracture is stable clinically, an active assisted range-of-motion program can be initiated.

If the fracture can be reduced closed but remains unstable when assessed fluoroscopically, the fracture requires fixation to maintain reduction and allow healing and initiation of range of motion. This can be accomplished via percutaneous pinning or open reduction.

Displaced Fractures: Three-Part Fractures

Two types of three-part fracture patterns can occur. The first type is displacement of the greater tuberosity and shaft, with the lesser tuberosity remaining attached to the articular segment. This pattern occurs more commonly. The second, and less commonly, encountered type of three-part fracture is displacement of the lesser tuberosity and shaft segments, with the greater tuberosity remaining attached to the articular segment.

Treatment options for these fractures include closed reduction, open reduction and internal fixation, or prosthetic replacement. At best, an adequate closed reduction is difficult to achieve, and more difficult to maintain, because the deforming muscular forces cannot be adequately offset by the position of immobilization. The results of closed reduction for three-part fractures have been quite variable.[33,91,97,106,114,171] Neer reported no successful results in 20 three-part fractures treated by closed reduction.[114] However, Young and Wallace reported 76% acceptable results following the nonoperative management of displaced fractures.[171] Unfortunately, they did not specify the number of three-part fractures treated. Leyshon treated 34 patients nonoperatively with 76% satisfactory results.[97] Because of inherent difficulties in performing a prospective randomized study for these uncommon injuries, the orthopedic literature on three-part proximal humerus fractures comprises mainly small series and retrospective reviews.

One exception, however, is the study of Zyto et al.[174] They prospectively followed 37 three-part fractures randomized into treatment with open reduction and internal fixation with tension band versus sling and early motion. Using the Constant score, they concluded that the outcome of operative and nonoperative treatment of three-part fractures of the humerus in elderly patients is similar, with more complications found in the surgically treated group. Criteria to be in the study included injuries not caused by high-energy trauma, at least 30% contact between the humeral head and shaft, and the ability of the patients to cooperate. They did not specify the methods used to classify the fracture patterns or whether interobserver reliability issues were considered. The potential uncertainty of fracture classification among observers makes it difficult to know the specific fracture types being treated. This may explain the wide range of outcomes reported for nonoperative and operative management. Zyto et al. also retrospectively assessed the long-term outcome of nonoperative treatment of three- and four-part fractures in the elderly. They assessed 17 shoulders with a minimum of 10 years of follow-up and found the range of motion to be satisfactory with mean flexion and abduction greater than 90 degrees. They found only four of the patients reported pain, which was described as mild.[175]

Despite some of the favorable results supporting nonoperative treatment, currently operative management is

the treatment of choice for three-part fractures of the proximal humerus.[12,13,40,67,126,139]

Displaced Fractures: Four-Part Fractures

Valgus-impacted

In "classic" four-part fractures, all four segments are displaced and the articular surface is devoid of soft tissue attachments. This results in disruption of the blood supply to the humeral head and a high likelihood of osteonecrosis. There is great variation in the reported incidence of osteonecrosis after these injuries, ranging from 21% to 75%[79,95,97,101,113,158] Neer reported osteonecrosis in six of eight patients with four-part fractures treated with open reduction and internal fixation.[113] Leyson reported osteonecrosis in six of eight patients treated nonoperatively.[97] Lee and Hansen reported a 21% incidence.[95] The variation could be secondary to the inclusion of four-part valgus-impacted fractures, which have a lower rate of osteonecrosis. In this fracture pattern type, the articular segment is impacted and angulated, rather than displaced, and may thus retain its vascularity from the posterior medial vessels traveling within the periosteum.[24,142] Theoretically, this source of blood supply can be interrupted by either a fracture line passing along the edge of the articular surface or through destruction of these vessels by lateral displacement of the articular segment relative to the main distal fragment.[125] Brookes et al. simulated four-part fractures in cadavers and found that perfusion of the humeral head was prevented, unless the head fragment extended distally below the articular surface medially; this allowed some perfusion of the head to persist by the anastomoses of the posterior medial vessels with the arcuate artery.[24]

Reconstruction rather than replacement for the valgus-impacted humeral head fracture has been extensively discussed in the recent literature, because the rate of osteonecrosis following this fracture pattern has been much lower than with the classic four-part fracture.[41] Jakob et al. reported that only 5 out of 19 (26%) valgus-impacted fractures developed osteonecrosis after either treatment by closed (five patients) or open (14 patients) reduction and minimal internal fixation with either AO screws or K-wires. They believed that replacement of the humeral head was not indicated because 74% of these patients had good results after 4 years. However, they also did not advocate nonoperative treatment because of anticipated problems with joint incongruity, impingement from the malpositioned tuberosities, and shoulder stiffness.[79]

In contrast, Court-Brown et al. found nonoperative treatment to be successful in elderly patients with valgus-impacted fractures. In their series, good to excellent results were obtained in 80% of patients treated.[38]

Classic Four-Part Fractures

The results of nonoperative treatment for classic four-part fractures have been consistently unsatisfactory as reported in the literature. Studies that appear to show acceptable results with nonoperative treatment of displaced fractures either have included no true four-part fractures or the classification of the fractures was such that an analysis of four-part fractures was not possible. Compito et al. reviewed 97 fractures from five series of true four-part fractures treated nonoperatively and showed that this resulted in a satisfactory outcome in only 5%.[36]

Misra performed a systematic review of the literature to compare the clinical outcomes of the management of three- and four-part fractures of the proximal humerus by nonoperative methods, internal fixation, external fixation, and arthroplasty. Only 24 reports published between 1969 and 1999 met the eligibility criteria. He found that patients managed nonoperatively had more pain and poorer range of motion than those managed operatively. However, he concluded that the current published data were inadequate for evidence-based decision making for the treatment of complex proximal humeral fractures.[108]

FRACTURE–DISLOCATIONS

Fracture–dislocations of the glenohumeral joint are characterized by a fracture of the proximal humerus in the presence of a complete loss of contact between the articular surfaces of the humeral head and glenoid. These injuries are usually associated with significant disruption of the soft tissues. Such traumatized tissues are prone to develop periarticular scarring and heterotopic bone formation. Repeated forceful attempts at closed reduction will increase the risk and severity of these problems. Therefore, although the first goal of treatment is prompt definitive reduction of the dislocation, multiple manipulations are to be avoided. Excessive force should never be used in the manipulation of fracture–dislocations, because it adds to the amount of soft tissue injury, endangers the adjacent neurovascular structures, and risks displacing previously undisplaced fractures.

Fracture–dislocations are classified by the direction of the dislocation (anterior vs. posterior) and the type of proximal humerus fracture present. The most systematic method to categorize these injuries is the Neer classification of proximal humeral fractures, as described earlier. The category of intraarticular fractures of the humeral head includes not only "head-splitting" fractures, but also the much more common impression fractures frequently associated with dislocations. Posterolateral impression fractures occur in association with anterior dislocations, whereas anterolateral impression fractures occur in cases of posterior dislocation.

Clinical and Radiographic Evaluation

Clinical Evaluation

Fracture–dislocations of the shoulder may occur at any age and by either indirect or direct mechanisms. In general, fracture–dislocations represent higher-energy injuries than simple dislocations or fractures alone. The history may indicate as much. The most common indirect mechanism involves a simple fall onto the outstretched upper extremity. Seizures and electroconvulsive therapy are other potential indirect causes of fracture–dislocations. The direct mechanism of injury involves a direct blow to the proximal humerus, and usually is the result of a fall directly onto the shoulder. As with proximal humerus fractures, patients should be asked about the circumstances surrounding their injury. If a fall was involved, patients should be asked about any head trauma, possible loss of consciousness, other injuries to the ipsilateral upper extremity, any subjective numbness or tingling involving that extremity, or any other focal sites of pain and tenderness. Other important history facts include patient age, occupation, hand dominance, and length of time since the injury.

Common presenting complaints include pain and swelling about the shoulder, especially with attempted motion. On examination, patients will typically exhibit crepitus and severe pain with minimal active or passive range of motion. Ecchymosis generally does not occur until 24 hours or more after the injury, but can occur sooner, usually in elderly patients. Eventually, ecchymosis may involve the entire extremity, as well as the axilla and down along the chest wall.

As with simple dislocations, there will be a change of the normal contour of the shoulder. In anterior fracture–dislocations, there is an anterior prominence in the shoulder, the lateral acromion becomes more prominent, and the coracoid is more difficult to palpate. There is a flattening of the posterior aspect of the joint. Conversely, in cases of posterior fracture–dislocations, the posterior aspect of the shoulder will be full, the anterior aspect of the shoulder hollow, and the coracoid more prominent. These findings can be quite subtle in large, muscular individuals, especially if considerable swelling is present.

Most fracture–dislocations result in the affected arm being held in a very characteristic manner. If anteriorly dislocated, the arm will usually be held in a position of relative abduction and external rotation; there can be a firm, painful block to passive internal rotation as the posterior aspect of the humeral head abuts against the anterior glenoid. Conversely, in posterior dislocations, the arm may be in internal rotation, with a firm, painful block to passive external rotation as the anterior aspect of the humeral head impacts on the posterior rim of the glenoid. The primary exception to these findings occurs when a surgical neck fracture is present, in which case motion in any plane may be possible through the fracture site, rather than at the articulation.

A thorough neurovascular examination of the involved extremity is of paramount importance in cases of glenohumeral fracture–dislocation because of the increased risk of associated vascular or neurologic complications. Vascular injuries, although rare, are devastating when they occur and usually involve injury to atherosclerotic axillary vessels in the elderly. Suspicion of a vascular injury is worked up in a manner similar to that discussed with proximal humerus fractures.

Neurologic injuries are much more common sequelae of fracture–dislocations, with the incidence reported ranging from 2% to 30%.[47,52,63,96,163,170] The axillary nerve is most commonly involved, although the entire brachial plexus or any component thereof may be affected. These injuries almost always represent traction neurapraxias, and most resolve clinically within 5 months.[16,28] However, it is critical that a thorough neurologic examination of the entire extremity be documented, both before and after any attempted manipulations. In evaluation of axillary nerve function, electromyographic (EMG) studies have shown that the commonly used method of testing sensation over the lateral aspect of the shoulder is quite unreliable.[16] A much more reliable method is to test for isometric deltoid motor function, which can be performed even in the context of the acute injury.

Despite the numerous signs and symptoms described in the foregoing, glenohumeral fracture–dislocations may be difficult to diagnose and can be missed by the initial examiner, particularly those involving posterior dislocations.[15,133,169] It is estimated that up to 50% of these injuries are initially misdiagnosed.[32] To avoid this pitfall, it is important to maintain a high level of suspicion for these relatively uncommon injuries, and to obtain a thorough and accurate radiographic evaluation.

Radiographic Evaluation

The plane of the glenohumeral articulation lies approximately 40 degrees anterior to the sagittal plane of the body. This must be taken into account if precise radiographs of this joint are to be obtained. Misdiagnosis of shoulder injuries is often attributed to radiographs being obtained in the plane of the body, rather than in the plane of the scapula centered on the glenohumeral joint. This results in overlapping structures that can be confusing and preclude accurate assessment of the injuries.

The axillary view is essential for evaluating glenoid and humeral articular surfaces, to identify displaced tuberosities, and it is the most sensitive view for detecting glenohumeral dislocations and subluxations. This view is most easily obtained with the patient in the supine position and the affected arm gently abducted to at least 30 degrees. If

possible, the arm can be supported in this position by the patient holding onto an IV pole.[11] If the patient is unable to assist, the arm should be positioned by someone wearing a lead gown.

Although the axillary view is the most difficult of the three views to obtain and requires moving the patient's painful arm, if done carefully, the great majority of patients will be able to tolerate the procedure. For those few patients who are unable to do so, however, the Velpeau axillary view described earlier is a suitable substitute that does not require removing the arm from the sling.[17]

Computed tomography is another imaging modality that can be used in the more complex or comminuted cases. This test provides unparalleled resolution for bony detail and, therefore, provides the most accurate assessment of fragment displacement, amount of articular involvement with a head-splitting or impression fracture (see Fig. 27-14), glenoid rim fractures, and chronic fracture–dislocations.

Although it is not as precise in assessing bony detail as a CT scan, MRI plays a very important role in evaluating traumatic shoulder injuries. It is the test of choice for evaluating soft tissue injuries about the shoulder and so can be used in patients for whom associated glenoid labral or rotator cuff tears are suspected. It can also assess injuries involving the axillary vessels and brachial plexus, and it remains the most sensitive method to detect osteonecrosis, a potential sequela of fracture dislocations.

Associated Injuries

Rotator Cuff Tears

All fracture–dislocations in which either or both tuberosities are significantly displaced will, by necessity, have a longitudinal split in the rotator cuff—often along the rotator interval between the supraspinatus and the subscapularis. In these cases, rotator cuff repair should be performed at the time open reduction and internal fixation is performed. In patients in whom the tuberosities do not fulfill the criteria for "displacement," associated cuff tears are not uncommon as a result of the gradual deterioration that occurs with advancing age. Therefore, the elderly are particularly susceptible to this injury, because their already degenerated cuff tendons are less able to withstand the high-tensile forces generated during dislocation.

Neurologic Injuries

Injury to peripheral nerves and the brachial plexus accompany fracture–dislocation of the shoulder, with a reported incidence of between 2% and 30%. This variability partly reflects age differences of the groups studied, as well as the method of assessing neurologic deficits—physical examination versus EMG. The axillary nerve, which courses anteriorly along the anterior border of the subscapularis, is especially vulnerable to traction injury during an anterior dislocation. It is the most commonly injured nerve in shoulder fracture–dislocations,[47,52,63,96,163,170] and the likelihood of injury increases with the age of the patient, the duration of the dislocation, and the severity of the trauma producing the injury.[122,170] The vast majority of these injuries represent traction neurapraxias and usually resolve spontaneously within 5 months.[16,28] Other isolated nerve injuries to the musculocutaneous, radial, and ulnar nerves are much less common.

When a neurologic injury is suspected, the initial physical examination findings should be carefully documented. An EMG should be obtained 3 weeks following the injury for further documentation. This is a much more sensitive method of assessing a neurologic injury than the physical examination. The difficulties in assessing axillary neuropathy based on physical examinations are twofold. First, dermatomal sensory testing is very unreliable.[170] Second, the high incidence of rotator cuff tears associated with these shoulder injuries confuses the picture somewhat. Clinical findings of weak abduction can be due to rotator cuff tear, axillary neuropathy, or the acute pain associated with the injury.

Vascular Injuries

Fracture–dislocations of the shoulder can, on rare occasions, be associated with vascular injuries—usually involving the axillary artery, its branches, or the axillary vein. These injuries can occur either during the injury itself or during the reduction maneuver. Attempted reductions of chronic dislocations are particularly risky and should be discouraged. Invariably, too many attempts are made and too much force used. Vascular damage most frequently occurs in elderly patients with fragile, atherosclerotic vessels and in patients whose soft tissues (and vessels) may be adherent as a result of previous dislocations or other injuries.[4,43,70] When a vascular injury occurs, it is an emergency and must be addressed immediately. These injuries are associated with a mortality rate of up to 50%.

Different types of arterial damage can occur, including intimal damage followed by thrombosis, avulsion of a large arterial branch, and laceration of the artery itself. As discussed earlier, anatomic studies have shown the axillary artery to be relatively fixed at the lateral border of the pectoralis minor muscle.[27] The artery becomes taut with abduction and external rotation of the arm, and is vulnerable to injury when the humeral head dislocates anteriorly and the pectoralis minor acts as a fulcrum over which the artery is stressed. There is the further risk with fracture–dislocations that the artery can be impaled or lacerated by the fracture fragments. The clinical findings and management of axillary artery injury is as discussed previously.

Impression Fractures of the Humeral Head

All shoulder dislocations impact the humeral head against the glenoid to some extent. An anteriorly dislocated humeral head is compressed against the anterior glenoid by the concomitant muscular contraction, thereby causing an impression fracture of the posterolateral humeral head. This defect, referred to as a Hill-Sachs lesion, can be quite large, especially in cases of chronic dislocation. Posterior dislocations frequently result in impression fractures of the anteromedial portion of the humeral head, referred to as a "reverse Hill-Sachs lesion" (see Fig. 27-14). These impression fractures can be quite large, especially in chronic posterior dislocations. In general, the larger the size of the defect is, the more difficult it is to obtain a successful closed reduction. These impression fractures play a much more prominent role in the treatment of chronic dislocations.

Treatment

Two-Part Fracture–Dislocations: Surgical Neck

Two-part surgical neck fracture–dislocations are very uncommon, but when they occur, the dislocation is almost always anterior.

In general, the approach to these injuries is to first achieve a gentle, closed reduction of the dislocation and then to determine the appropriate treatment for the fracture, based on the evaluation of the postreduction radiographs. Although conscious sedation is usually sufficient, if closed reduction is unsuccessful, general anesthesia should be used to avoid multiple attempts. Closed reduction of the anteriorly dislocated humeral head can often be accomplished with gentle fingertip mobilization of the humeral head while longitudinal traction is applied to the arm. There are a few circumstances in which open reduction is indicated:

1. Vascular injury
2. Open fracture
3. Failed or difficult closed reduction
4. Anterior or posterior dislocation associated with a nondisplaced surgical neck fracture (closed manipulation risks fracture displacement)

Two-Part Fracture–Dislocations: Greater Tuberosity

Greater tuberosity fractures have been reported to occur in up to 33% of anterior shoulder dislocations.[132] This is the most common type of fracture–dislocation. The initial step in treatment of these injuries should always be gentle, closed reduction of the dislocation, followed by a complete set of postreduction radiographs. In many cases, the reduction restores the greater tuberosity fragment to near-anatomic position (Fig. 27-17). When this

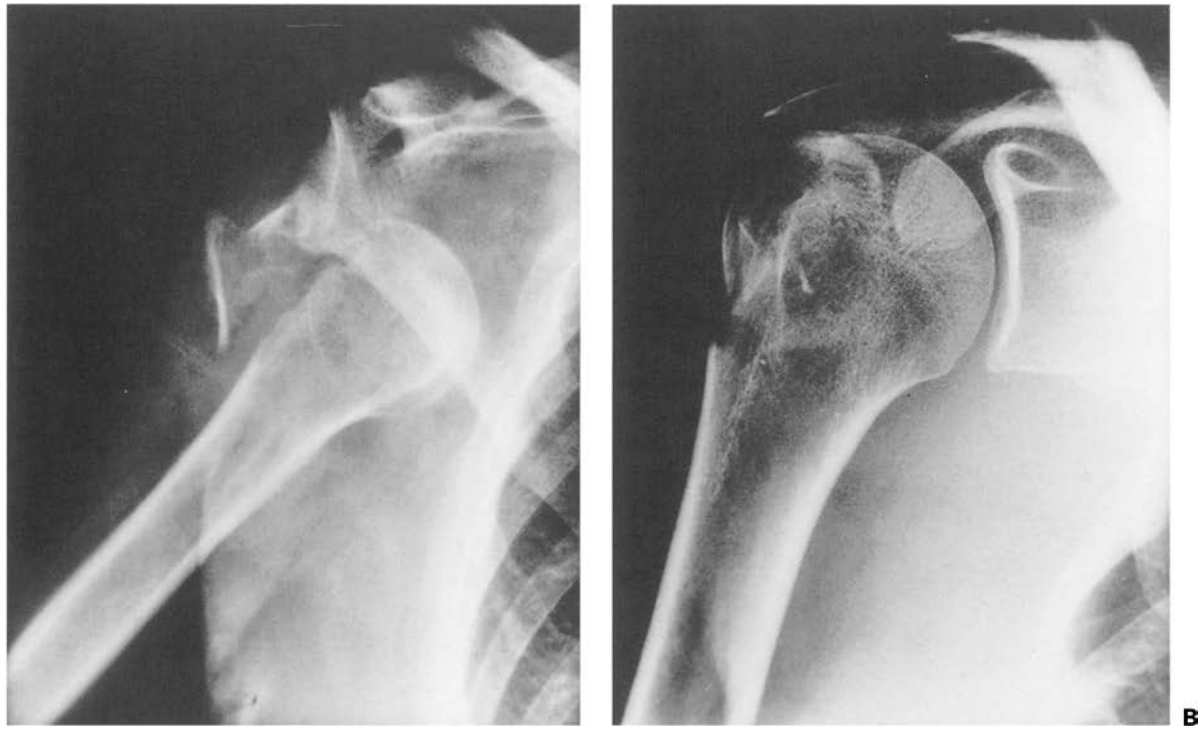

Figure 27-17 A 61-year-old woman who fell onto the outstretched right upper extremity sustaining **(A)** a two-part anterior greater tuberosity fracture dislocation. **(B)** Following closed reduction, there is near-anatomic reduction of the greater tuberosity fragment.

occurs, nonoperative management is preferred, consisting of sling immobilization for approximately 4 weeks. However, passive range-of-motion exercises should be started as soon as discomfort subsides. Frequent follow-up radiographs are essential in the postreduction period to ensure that the tuberosity has not redisplaced with the early motion program.

Management is as with greater tuberosity fractures, discussed earlier. In cases where the greater tuberosity remains displaced by more than 5 mm, operative management is indicated, unless the patient's medical condition or limited functional demands indicate otherwise.[162] Prognostically, dislocations associated with a greater tuberosity fracture rarely go on to become recurrent, because the anterior soft tissues are usually spared, and the joint stability is restored once fracture healing occurs.

Two-Part Fracture–Dislocations: Lesser Tuberosity

Posterior glenohumeral dislocations are sometimes accompanied by lesser tuberosity fractures. This represents an avulsion-type fracture as a result of the strong pull of the attached subscapularis tendon and anterior capsule. When these injuries are acute or recent (within 2 weeks), the first step in treatment should be attempted closed reduction of the dislocation. This can be accomplished with the patient supine, by application of longitudinal traction to the arm while it is held in 90 degrees of forward flexion. The arm should then be gradually adducted across the chest while maintaining traction, in an effort to unlock the head from behind the glenoid. The maneuver can sometimes be facilitated by application of digital pressure on the humeral head from behind or a laterally directed force on the proximal humerus.

Postreduction radiographs should then be obtained to document reduction of the glenohumeral joint, to assess the position of the lesser tuberosity fragment, and to ensure that no other fractures have been displaced by the reduction maneuver. If the reduction is stable and residual tuberosity displacement is less than 1 cm, then the patient should undergo 4 weeks of immobilization in a modified shoulder spica cast or orthosis that maintains the arm at the side in 10 to 15 degrees of external rotation, 10 to 15 of degrees extension, and 10 to 15 degrees of abduction. When residual instability is present or the displacement of a large lesser tuberosity fragment exceeds 1 cm, operative management is indicated. The goals of operative management are to restore stability and anatomically reduce and securely fix the lesser tuberosity fragment. However, the decision to proceed with operative management should be based on a careful assessment of all factors, not the least of which is the patient's own functional expectations.

Three-Part Fracture–Dislocations: Anterior

Three-part anterior fracture–dislocations consist of anterior dislocation of the humeral head with displaced fractures of the surgical neck and greater tuberosity. The lesser tuberosity remains attached to the humeral head and provides some blood supply to the articular surface through the subscapularis and anterior capsule attachments. However, the arcuate artery of Laing, which carries most of the direct blood supply to the head, is at risk with this injury because it penetrates the medial portion of the greater tuberosity, with the resulting risk of osteonecrosis.[60] Open reduction of the dislocation should be performed, for it can be done much more gently, thus reducing the risk of further soft tissue injury. Our preferred treatment for this injury consists of open reduction of the dislocation, followed by fixation of the displaced fragments or proximal humeral replacement.

Three-Part Fracture–Dislocations: Posterior

Three-part posterior fracture–dislocations are characterized by posterior dislocation of the humeral head, with the greater tuberosity attached, in combination with displaced fractures of the surgical neck and lesser tuberosity. Complete radiographs (trauma series) are essential to accurately assess this injury and to avoid overlooking the posterior dislocation.

As with three-part anterior dislocations, nonoperative treatment is not recommended for these injuries. They require open reduction and fracture treatment of the dislocation to be performed as soon as possible after the injury.

Four-Part Fracture–Dislocations

All four-part fracture–dislocations, whether anterior or posterior, are associated with significant disruption of the blood supply to the humeral head. Osteonecrosis of the articular segment is almost inevitable, and for this reason, proximal humeral replacement is the treatment of choice; open reduction and internal fixation is considered only in young patients with good bone quality. Nonoperative treatment is not recommended for this injury pattern.

Summary of Nonoperative Treatment

Most proximal humerus fractures are minimally displaced and therefore can be treated nonoperatively. Two-part surgical neck fractures with less than 45 degrees of angulation and minimally displaced tuberosity fractures that do not impede range of motion can also be treated nonoperatively. Three- and four-part fractures typically should be managed operatively; however, nonoperative treatment may be considered in elderly patients, particularly in those patients with the valgus-impacted pattern.

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28 Minimally Invasive Techniques for Proximal Humeral Fractures

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INTRODUCTION: DEFINITION OF MINIMALLY INVASIVE FIXATION

Minimally invasive fracture fixation is defined as a technique utilizing indirect percutaneous reduction and fixation under fluoroscopic visualization with hardware (usually pins and/or screws) placed without extensive exposure and soft tissue dissection. Most fractures of the proximal humerus can be treated successfully with nonoperative treatment and early mobilization. Less than 20% of the fractures are displaced and unstable requiring operative stabilization.[11,15,17] There have been numerous controversies over the treatment of these displaced and unstable fractures. Various methods of open reduction and internal fixation have been described and historically have been utilized for the treatment of unstable displaced humerus fractures. Open reduction and internal fixation can potentially increase the risk of avascular necrosis of the humeral head because of damage to the vascular supply to the humeral head secondary to extensive soft tissue stripping at the time of reduction and/or hardware application.[10,11,15,16,40] The primary blood supply to the humeral head is the anterior humeral circumflex artery, which runs along the lateral aspect of the bicipital groove. The blood supply may be compromised at the time of injury and may also be injured during operative treatment. Therefore, increased attention has been given to reducing soft tissue stripping during reduction and plate application. This, in turn, has led to

increased interest in minimally invasive techniques of fracture fixation. The goal is the same for any type of fixation—anatomic reduction and stable fixation.

Recently, there has been a surge of interest in minimally invasive fixation techniques in many different subspecialty areas of orthopedics. The recent trauma literature contains several reports of percutaneous fixation of the femur, tibia, and tibial pilon fractures.[18,27,33] The importance of preserving blood supply and minimizing soft tissue stripping are increasingly recognized. With regard to the treatment of proximal humerus fractures, several techniques have been introduced in the context of minimally invasive fixation. These include flexible intramedullary pins, external fixation, and percutaneous pinning techniques. Zifko et al.[43] introduced 2-mm pins into the humerus from the distal medullary canal about 3 cm proximal to the olecranon fossa in a retrograde fashion. They reported excellent or good clinical results in 58% of patients and a 98% bone union rate. Kristiansen[22] and Kyle and Conner[23] studied outcomes of closed reduction and external fixation of proximal humerus fractures. The former reported excellent or satisfactory results in 68% of his cases and the latter reported satisfactory results in 80% of their cases. External fixation is not indicated in the majority of fractures of the proximal humerus, but it can be a logical alternative in the management of a few selected fractures.[23]

There have been several reports of successful closed reduction and percutaneous pinning during the past two decades.[8,12,14,17,35,36,39] The technique of closed reduction and percutaneous pinning of the proximal humerus fractures was originally described by Böhler, who used the technique for the treatment of the proximal humeral epiphyseal injury in children.[4] The technique has been modified over years and applied to treat the proximal humerus fractures more commonly seen in the older population. The technique was initially used for isolated fractures of the greater tuberosity or surgical neck, and was gradually extended to the management of more complex fracture types (Table 28-1). In selected fractures, closed reduction and percutaneous pinning allows preservation of the intact soft tissue sleeve and periosteal blood supply while obtaining and maintaining a stable reduction. Other potential advantages include a smaller incision, less dissection, and less scarring. A minimally invasive approach minimizes trauma to the rotator cuff and deltoid, and with experience can decrease operative time. While still a difficult and technically demanding procedure, percutaneous pinning of selected proximal humerus fractures shows considerable potential.

TABLE 28-1
FRACTURES AMENABLE TO PERCUTANEOUS PINNING

Two-part	Surgical neck Greater tuberosity *Lesser tuberosities
Three-part	Surgical neck/greater tuberosity *Surgical neck/lesser tuberosity
Four-part	Valgus-impacted

*Without associated posterior dislocation.

INDICATIONS FOR MINIMALLY INVASIVE FIXATION

The technique of closed reduction and percutaneous fixation is difficult and demanding, and careful attention must be paid to appropriate indications and technical considerations. A successful outcome after operative treatment of unstable and displaced proximal humeral fractures, regardless of approach or choice of hardware, depends on a few critical factors: (a) anatomic reduction, (b) stable fixation, (c) careful management of soft tissues, and (d) appropriate rehabilitation. Percutaneous pinning offers an excellent alternative to the open approach in selected fractures (Table 28-2).

Fracture Type

The same principles of anatomic open reduction and stable fixation apply to minimally invasive fracture treatment. Closed reduction of the fracture must be attainable and the reduction must be stable under fluoroscopy after pinning. The fracture must be an acute injury generally less than 14 days old. An intact medial calcar (medial humeral metaphysis just under the articular segment) region is important for stability after reduction and fixation.

Two-Part Fracture of the Surgical Neck

A displaced two-part fracture of the surgical neck is an ideal indication for percutaneous pinning, provided the fracture can be reduced by closed manipulation under anesthesia. In this type of fracture, both tuberosities are attached to the head fragment so that the head stays in a neutral position. Most surgical neck fractures are treated

TABLE 28-2
CONDITIONS FOR SUCCESSFUL PINNING

- Good bone stock
- Intact medial calcar
- Substantial greater tuberosity fragment
- Stable reduction under fluoroscopy after pinning
- Reliable, cooperative patient

nonoperatively. Displacement of 50% or more in an active, healthy individual is an indication for operative treatment. Surgical neck fractures are often easily reduced by the closed method. If closed reduction is not possible, soft tissue such as the long head of the biceps tendon, capsule, or muscle may be interposed between the fragments.[2] In this situation, a limited open reduction is recommended. Even in these cases percutaneous pinning for fracture fixation with minimum hardware can be effective. Comminution of the fracture site, especially the medial calcar (metaphysis) region, is a relative contraindication to closed treatment. A large medial calcar may preclude a stable reduction and the risk of varus displacement is high. An impacted surgical neck fracture is an excellent indication for closed reduction and percutaneous pinning. The impacted anterior fragment should be disimpacted from the shaft to restore the normal neck–shaft angle.

Two-Part Fractures Involving the Greater and Lesser Tuberosities

Traditionally, two-part fractures of the tuberosities have been managed by open reduction and internal fixation.[9,15,29] There have been few attempts to manage these fractures by a closed method.

An isolated greater tuberosity fracture occurs with or without anterior glenohumeral dislocation. Greater tuberosity fractures are usually retracted posteriorly and superiorly, and closed reduction is difficult. However, if this fracture is associated with an anterior dislocation, a closed reduction of the glenohumeral dislocation may successfully reduce the greater tuberosity fracture.[2] Tuberosity fractures with 5 mm[6] or more of displacement are generally considered operative cases. Unlike surgical neck fractures, where displacement is easily tolerated, even minimal displacement of a tuberosity can be problematic. Percutaneous screw fixation of displaced greater tuberosity fractures can be considered if the fragment is substantial enough to allow hardware fixation. If the greater tuberosity fragment is irreducible by closed means or comminuted, open reduction and internal fixation with transosseous and cuff-to-bone suture fixation is required.

Isolated lesser tuberosity fractures are usually associated with posterior dislocations and may also be treated by closed reduction if the articular involvement is minimal. Percutaneous screw fixation can be employed for fixation of the reduced fragment.

Three-Part Fracture

Three-part fractures are often severely displaced and unstable because of the strong deforming forces by attached muscles. They are difficult to treat by closed reduction and minimal fixation methods. However, several authors reported satisfactory results with closed reduction and percutaneous fixation.[1,8,12,14,35,36,39] Closed reduction and percutaneous fixation is a good option for treatment in selected cases, where there is good bone stock without significant comminution. The long head of the biceps tendon may interpose between the fracture fragments, making closed reduction difficult. Sometimes the tendon can be released manually through the small reduction portal. If not, repeated attempts at reduction may cause further comminution and injure the interposed tendon, and a more formal open reduction and internal fixation should be performed.

Four-Part Fracture

Four-part proximal humerus fractures as classified by Neer[1,31,32] are particularly problematic. Historically, they have a very high rate of avascular necrosis following fixation. Because of this, Neer recommended hemiarthroplasty for the treatment of these fractures. However, a subgroup of the four-part proximal humerus fractures, "four-part valgus-impacted fractures," is readily amenable to reduction and fixation. Neer did not specify this fracture in his initial classification system. Similar fractures have been described by several authors[26,28,40] using different names. More recently, the AO/ASIF organization introduced a new classification system, which was essentially an expansion of Neer's original work; in their review, 14% of all documented cases are found to be valgus-impacted injuries and are classified as C2.1 or C2.2 type fractures.[15,26,28] The first authors to deal specifically with this type of fracture were Jakob et al.[17] in 1991.

Valgus-impacted four-part proximal humerus fractures have a unique configuration and a far lower risk of osteonecrosis if treated appropriately. In four-part valgus-impacted fractures, the head is impacted onto the shaft at the surgical neck, such that the articular surface is 90 degrees to the long axis of the shaft. The articular segment faces superiorly, toward the acromion, rather than medially, toward the glenoid. The tuberosities are displaced laterally.[13,17,34,36] (Fig. 28-1). The main source of vascularization for the humeral head, the ascending anterolateral branch of the anterior humeral circumflex artery,[5,10] is interrupted at its point of entry into the humeral head in the area of the intertubercular groove. Other sources of vascularization via both tuberosities and metaphyseal arterial anastomoses are deprived as well. However, perfusion of the humeral head via the intraosseous arcuate artery may continue if the head fragment includes part of the medial aspect of the upper part of the neck. In true valgus-impacted fractures, there is little or no displacement of the medial aspect of the humeral articular surface with respect to the medial aspect of the shaft (see Fig. 28-1). In such cases, the posteromedial arteries from the posterior humeral circumflex artery are still attached to the medial aspect of the articular fragment via the medial soft tissue

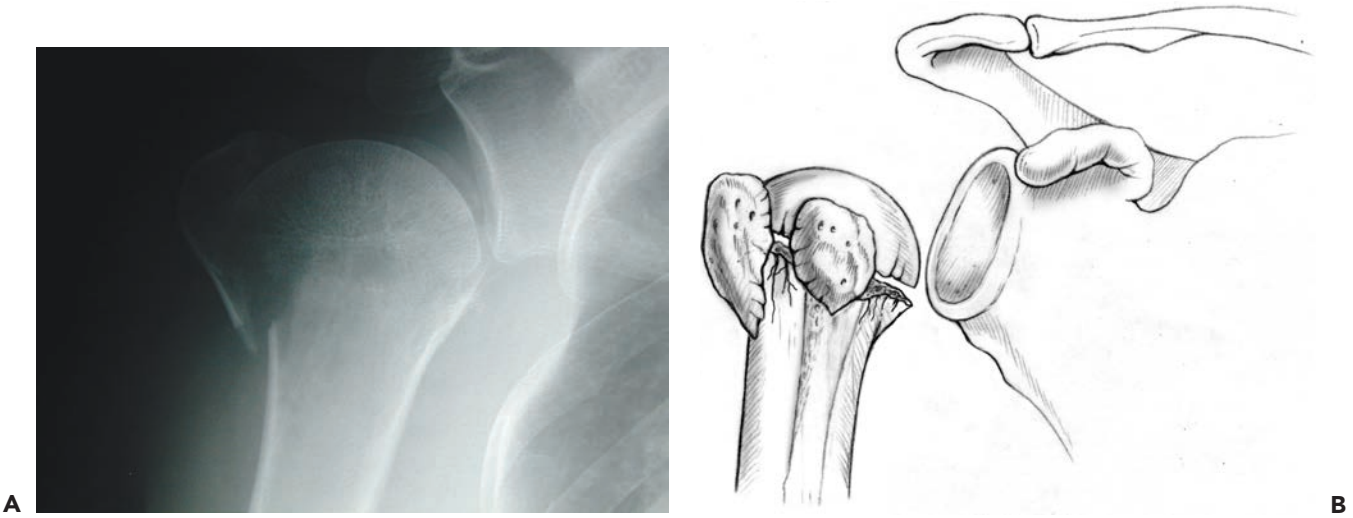


Figure 28-1 **(A)** Valgus-impacted four-part fracture of proximal humerus. The head is impacted onto the shaft at the surgical neck such that the articular segment faces superiorly, toward the acromion, rather than medially, toward the glenoid. The tuberosities are displaced laterally. **(B)** A schematic drawing of valgus-impacted four-part fracture. (Adapted from Resch H, Beck E, Bayley I. Reconstruction of the valgus-impacted humeral head fracture. *J Shoulder Elbow Surg* 1995;4(2):75.)

hinge and thus can supply the articular fragment through the anastomoses with the arcuate artery[5] (Fig. 28-2). Therefore, the prevalence of necrosis of the humeral head (8% to 26%) is much lower than that associated with standard four-part fractures (46% to 75%).[17,25,32,34-36]

In four-part valgus-impacted fractures, a continuous envelope of soft tissue bridging the shaft, tuberosities, glenohumeral joint capsule, and rotator cuff is maintained, even after the high-energy impaction that caused the fracture. This soft tissue envelope substantially contributes to stability after reduction and facilitates anatomic or nearly anatomic reduction of the tuberosities once the head is reduced.[13] However, several anatomic aspects of these unique fractures should be considered before

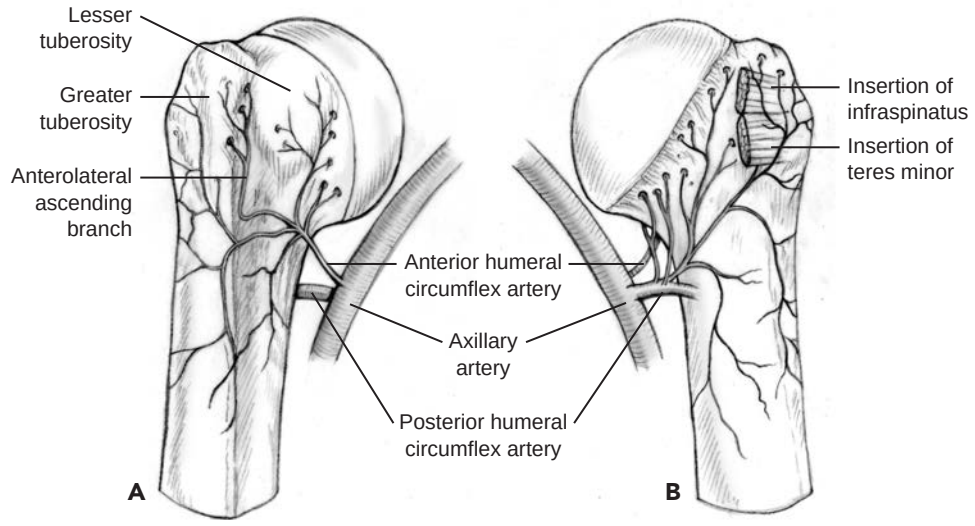


Figure 28-2 Normal blood supply to the humeral head. **(A)** The anterior aspect of the humeral head. The anterior circumflex artery enters the humeral head at the proximal end of the transition from the greater tuberosity to the intertubercular groove. After entering the bone, within the epiphysis, the anterolateral branch gives off the arcuate artery to form numerous intraosseous anastomoses. The arcuate artery vascularizes essentially the entire humeral head. **(B)** The posterior aspect of the humeral head. The posterior circumflex artery has abundant medial branches, which enter the head and anastomose with the arcuate artery from the anterior circumflex artery. (Adapted from Gerber C, Schneeberger AG, Vinh TS. The arterial vascularization of the humeral head. An anatomical study. *J Bone Joint Surg Am* 1990;72(10):1489,1491.)

proceeding to closed treatment. The medial periosteal vessels on the anatomic neck contribute enough blood supply to the humeral head that the incidence of avascular necrosis is low. If the medial periosteal vessels on the anatomic neck are to survive, the fracture line must be located distally to the point at which the vessel enters into the bone. The posteromedial vessels enter the head at the point just distal to the articular cartilage and form an anastomosis with the arcuate artery within the head. If the fracture line is located proximal to the entry point or is combined with comminution on the medial calcar region, the probability of survival of the articular fragment becomes minimal. Another point to consider is that the lateral displacement of the articular segment relative to the shaft compromises the medial periosteal vessel and results in a higher incidence of avascular necrosis. When pronounced lateral displacement of the articular fragment is present in an older patient, primary hemiarthroplasty is inevitable.[34] In young patients, however, closed reduction and percutaneous fixation or open reduction and internal fixation should be attempted first, to avoid hemiarthroplasty in this age group.

Other standard displaced four-part fractures in older patients should be treated with primary hemiarthroplasty because of its high rate of avascular necrosis and instability.[11,32] Resch et al.[36] tried closed reduction and percutaneous fixation in five cases of laterally displaced, nonimpacted four-part fractures. Two of the five required revision to prosthesis; one of the two presented with avascular necrosis. The other three had good results.

Age, Bone, and Soft Tissue

Typical closed reduction and percutaneous fixation employs 2.5-mm pins and/or 4.5-mm cannulated screws. Biomechanically, percutaneous fixation has inherently weaker fixation strength than open reduction and internal fixation with plates or locked intramuscular fixation.[1,21,30,41] Stable fixation with pins and screws requires good bone quality and compliance with a more conservative rehabilitation program. Significant osteopenia or associated conditions risk the postreduction stability and complicate any possible revisions. There are no definite guidelines about age. Chronologic age seems less important compared to physiologic age. Therefore, the patient must be physiologically young with compelling reasons to retain the natural humeral head. The patient must be motivated to undergo this operative procedure and comply with the rehabilitation protocol. The patient should have normal cognitive ability.

Closed reduction and percutaneous fixation is contraindicated in (a) patients with poor bone stock including pathologic fractures; (b) fractures where there is a comminuted proximal shaft fragment, especially in the medial calcar region; (c) displaced four-part fractures (other than the valgus-impacted configuration) in elderly people requiring hemiarthroplasty; (d) some head-split fractures; (e) nonacute fractures (older than 14 days); (f) noncompliant patients or patients unable or unwilling to comply with strict follow-up and rehabilitation limitations; (g) fractures with displaced greater tuberosity fractures that are too comminuted or small for hardware fixation; (h) open fractures which require open irrigation; (i) dirty contaminated deep skin wound over the fracture site; and (j) any medical illness that precludes a surgical intervention.

SURGICAL TECHNIQUE

Preoperative Planning

Patient evaluation begins with a thorough history and physical examination. The mechanism of injury should be noted and all associated injuries completely evaluated. Most proximal humeral fractures are the result of low-energy falls in elderly patients. In the younger population, high-energy injuries are commonly responsible for the fractures. A thorough neurovascular examination should be performed before percutaneous fixation is attempted. The patient's social situation should be assessed to discern whether the patient is likely to comply with rehabilitation and close follow-up. In the case of a dirty contaminated skin wound over the fracture site, the operation must be deferred until the wound heals or other treatment measures are required.

Radiographic evaluation consists of four standard views: a regular anteroposterior (AP) view of the shoulder, a true anteroposterior view (or scapula plane AP) of the shoulder, an axillary view, and a scapular Y-view. The true anteroposterior view of the shoulder with the arm in the neutral rotation is useful to assess the degree of head impaction and tuberosity displacement. The axillary view is helpful in assessing anteroposterior angulation, displacement of tuberosities, presence of articular surface defects, and presence of dislocation. If the patient cannot be positioned for an axillary view, the Velpeau axillary can be used.[3] Clear appreciation of the plane and location of all fracture lines is essential in determining the likelihood of successful percutaneous pinning. If evaluation is incomplete with plain x-rays alone, a computed tomography (CT) scan may be helpful.

The usefulness of CT scans has been a subject of debate. An axial CT scan with 2-mm sections adds detail to evaluation of complex fractures. Additional CT scan images and three-dimensional reconstructions were shown to improve the accuracy of evaluation of proximal humerus fractures.[7] On the other hand, Bernstein et al.[1] reported that the addition of CT scans were associated with a slight increase in intraobserver reliability, but no increase in interobserver

reproducibility. Magnetic resonance imaging (MRI) is not helpful in determining head vascularity after an acute injury and is not indicated.

Closed reduction and percutaneous fixation techniques are based on the assumption that closed reduction and fixation is attainable. Importantly, patients should be prepared beforehand for a situation in which closed reduction and fixation is not possible. The patient should be consented for alternative treatment methods before surgery. The surgeon should be prepared for open reduction and internal fixation or, if necessary, hemiarthroplasty.

Positioning

The patient is positioned to allow unencumbered access to the shoulder, both for easy visualization under fluoroscopy and for easy pin placement (Fig. 28-3). The patient is placed on a radiolucent operating table with the head in a head holder. The procedure can be performed with the patient in the supine position; however, raising the head of the bed 15 to 20 degrees is often helpful for orientation and instrumentation. The thorax is shifted as far lateral as possible to the side of the table to increase radiographic visualization, and stabilized to prevent the patient from falling off the table. The neck should be in a neutral position, and the hips and knees should be flexed. The arm is draped free. Adequate visualization of the shoulder under fluoroscopy should be confirmed before prepping and draping. Alternatively, a complete beach-chair position may also be utilized, although the fluoroscopy must be swung over the top to obtain an axillary view. The x-ray unit is closer to the surgeon's head, however, increasing radiation exposure to the surgeon.[42] A mechanical arm holder is used for positioning the arm during the procedure. The holder can be useful for placing traction on the arm when necessary.

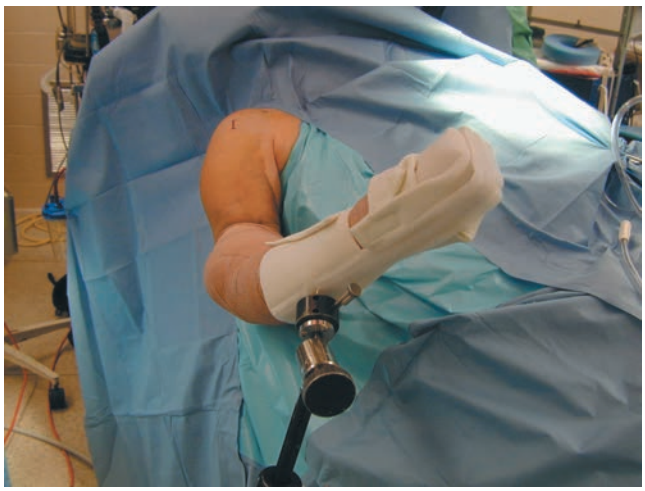


Figure 28-3 Operative setup for closed reduction and percutaneous fixation. The patient is placed in the supine position on the radiolucent operating table. The thorax is shifted to the lateral side of the table and stabilized. The mechanical arm holder is useful for maintaining position of the arm and placing traction on the arm.

The C-arm fluoroscopy is positioned at the head of the bed, parallel to the patient, leaving the area lateral to the shoulder open for access and pin placement. Alternatively, the C-arm can be angled perpendicular to the patient; however, it is much more difficult to get an axillary view with the C-arm in this position. The monitor is placed on the opposite side of the table. We recommend not using an adhesive, plastic drape directly on the skin of the operative site, because it can inadvertently become adherent to the pins during insertion and may be introduced into the wound. The shoulder should be draped to accommodate conversion to an open procedure, should it be necessary.

Fracture Reduction

Bony landmarks are outlined on the skin, especially the acromion. The reduction maneuver may be performed before the sterile prepping and draping (if it can be done entirely by closed means) to assess the feasibility of the closed technique.

Two-Part Fracture of the Surgical Neck

The humeral shaft is typically displaced medially, angulated with the apex anterior and slightly internally rotated because of the pull of the pectoralis major muscle. The head fragment usually remains in a neutral or slightly varus position. Closed reduction is performed by flexing, adducting, and slightly internally rotating the humerus to relax the pectoralis major muscle. Traction is then applied along the long axis of the humerus with a posteriorly directed force applied on the proximal shaft. If the reduction is satisfactory under fluoroscopy, an assistant takes over the position of the reduction and maintains the posteriorly directed force on the shaft. The arm is in slight abduction (20 degrees) in the scapular plane and in neutral rotation.[42]

The fracture fragments can be manipulated through a small "reduction portal." A 1- to 2-cm incision is made off the anterolateral corner of the acromion at the level of the surgical neck. The deltoid is gently and bluntly spread. A blunt instrument can be introduced through this portal, into the fracture, and used to manipulate and/or lever the fragments. Overly aggressive manipulation should be avoided to preserve and protect the osteoporotic bone that often characterizes proximal humerus fractures. This "reduction portal" can be utilized in many of the various fracture configurations.

Two-Part Fracture of the Greater or Lesser Tuberosity

Tuberosity fractures will be discussed in the following section, Three-Part Fracture.

Three-Part Fracture

When a three-part fracture involves the greater tuberosity, the humeral head is internally rotated due to the unopposed pull of the subscapularis. The shaft is displaced medially, internally rotated, and anteriorly angulated by the pectoralis major in the same way as the two-part surgical neck fracture (Fig. 28-4). The closed reduction starts with bringing the arm in flexion, adduction, and internal rotation to relax the pectoralis major. An assistant then applies traction along the line of the humerus with a posteriorly directed force on the proximal shaft. Once satisfactory reduction of the surgical neck is achieved under the C-arm fluoroscopy, traction is released and the arm is brought down to the side and placed in slight abduction. The posteriorly directed force should be maintained until the fracture is fixed with pins. If the rotation of the head fragment is so pronounced that the above measure is not helpful, a Steinmann pin may be drilled into the humeral head for derotation. The reduction is secured by means of two or three terminally threaded 2.5-mm pins drilled from the anterolateral aspect of the shaft to the head in a retrograde fashion. After fixation of the head–shaft interface, the arm is then returned carefully to the neutral rotation.

The greater tuberosity fragment is reduced percutaneously using a hook through the previously described "reduction portal." The hook is placed into the rotator cuff tissue near its insertion to the tuberosity fragment and is used to pull the fragment in an anterior, inferior direction. Alternatively, a puncture incision may be made over the greater tuberosity lateral to the acromion so that a

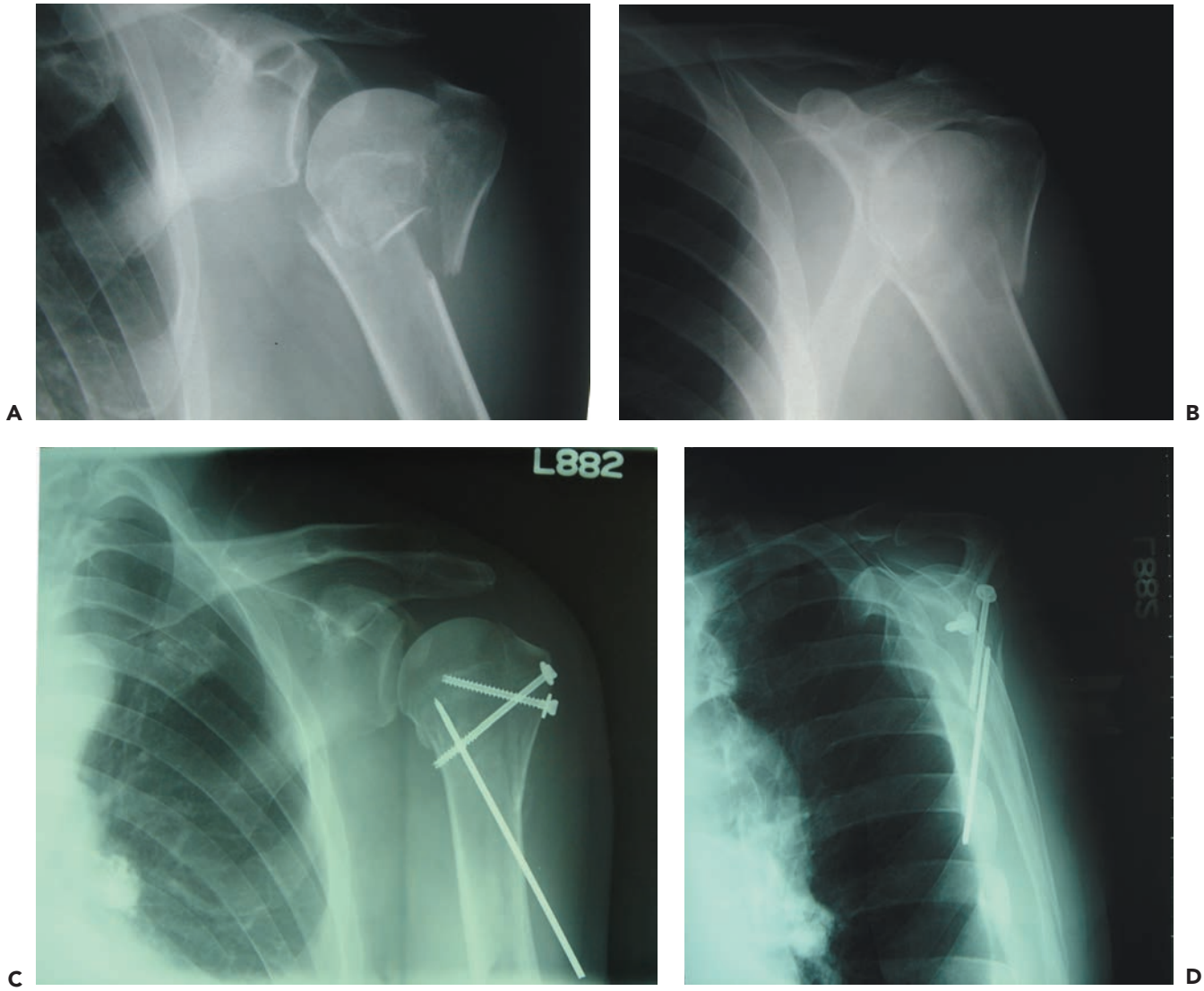


Figure 28-4 Three-part fracture of the proximal humerus involving the greater tuberosity. The humeral head is internally rotated due to the unopposed pull of the subscapularis. The shaft is displaced medially, internally rotated, and anteriorly angulated by the pectoralis major: **(A)** a true anteroposterior view; **(B)** a regular anteroposterior view; **(C,D)** postoperative radiographs.

reduction tool is introduced to pull the greater tuberosity fragment anteroinferiorly. Once reduced, the greater tuberosity should be held temporarily with K-wires or guide pins for 4.5-mm cannulated screws. When the guide pins for 4.5-mm cannulated screws are used, the drilling points will be the final screw insertion sites. Thus, the drilling points should be selected carefully not to cause further fragmentation of the tuberosity. The accuracy of reduction should be verified by at least biplanar fluoroscopic examination.

When the lesser tuberosity is fractured in a three-part fracture, the head fragment is externally rotated, abducted, and anteriorly angulated by the posterosuperior rotator cuff muscles. Thus, the reduction maneuver is composed of external rotation and flexion of the arm and a posteromedially directed force on the proximal shaft with longitudinal traction. Anteroposterior and axillary C-arm fluoroscopy should verify the reduction. A Steinmann pin may also be employed to facilitate the derotation of the head fragment. The reduction should be held in position using multiple terminally threaded 2.5-mm pins as described previously. Then the arm is brought to the side and internally rotated to seat the medially displaced lesser tuberosity on the head–shaft composite. The C-arm is now set for the axillary view. A "reduction portal" distal to the anterolateral corner of the acromion may be created. A hook can be advanced toward the lesser tuberosity, which then can be pulled laterally to its normal position. An axillary fluoroscopic view shows the accuracy of the reduction. Temporary fixation is provided with K-wires or guide pins for 4.5-mm cannulated screws.

A three-part fracture with an impacted head fragment requires a small incision over the fracture site for disimpaction of the head. The detailed technique of reduction is discussed in the following section, Four-Part Valgus-Impacted Fracture.

Four-Part Valgus-Impacted Fracture

The arm is held at the side in neutral rotation. A small 1- to 2-cm incision is made 2 to 3 cm distal to the anterolateral corner of the acromion. Formation of this "reduction portal" facilitates reduction of the fracture percutaneously prior to pin fixation (Fig. 28-5). The reduction portal is positioned distal to the anterolateral corner of the acromion at the level of the surgical neck of the humerus, posterior and lateral to the biceps tendon. The fracture between the greater and lesser tuberosities lies approximately 0.5 to 1 cm posterior to the bicipital groove. Locating the reduction portal over the split between the tuberosities enables elevation of the head fragment by placing the instrument through the fracture line.

After the incision is made, the deltoid is gently and bluntly spread to avoid possible injury to the anterior branch of the axillary nerve in this location. A blunt-tipped elevator or a small bone tamp is placed through the reduc-

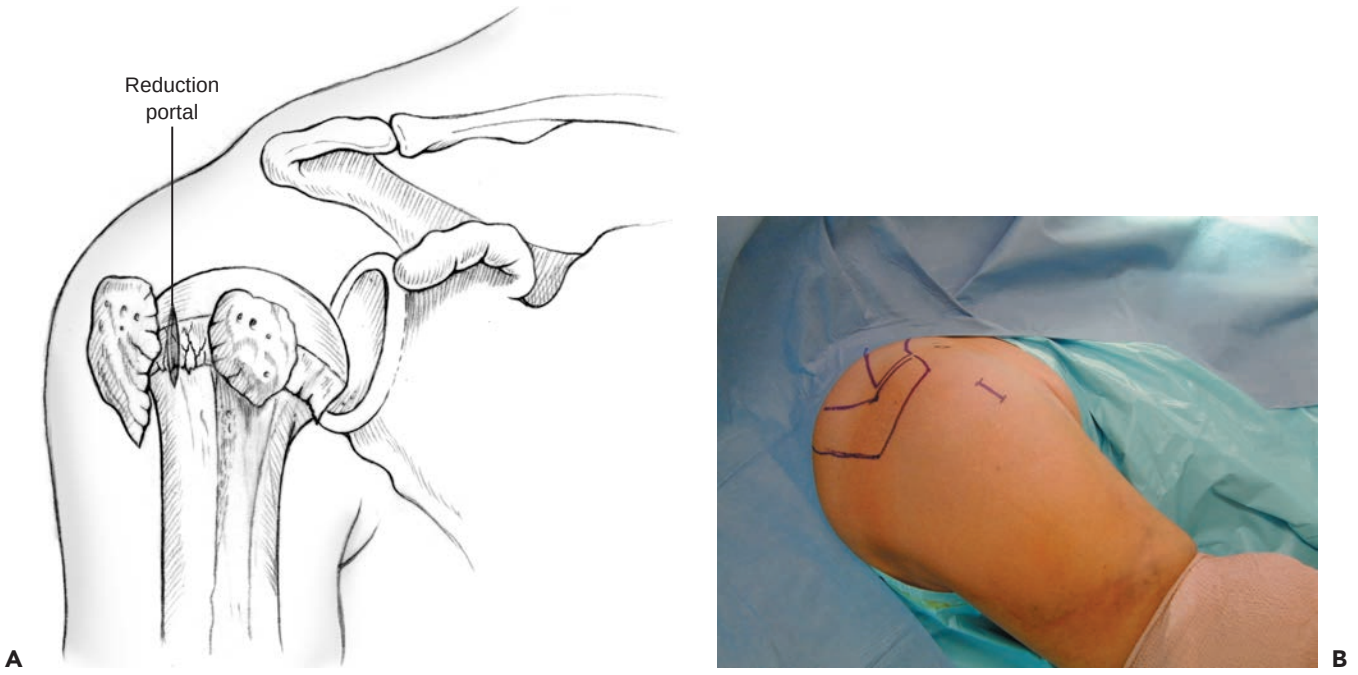
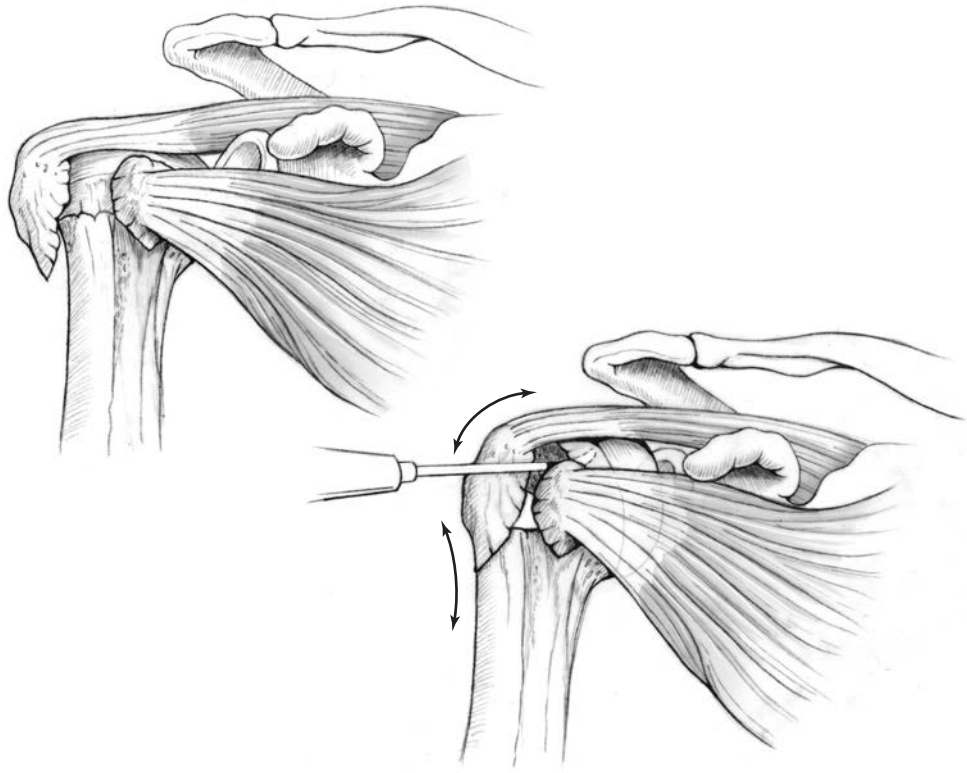


Figure 28-5 **(A)** The reduction portal. A small 1- to 2-cm incision is made 2 to 3 cm distal to the anterolateral corner of the acromion. The reduction portal is positioned at the level of the surgical neck of the humerus, posterior and lateral to the biceps tendon. Locating the reduction portal over the split between the tuberosities enables elevation of the head fragment by placing the instrument through the fracture line. **(B)** An intraoperative photograph showing the location of the reduction portal.

tion portal to lie deep to the lateral margin of the head fragment (Fig. 28-6). The position of the instrument is confirmed with the C-arm fluoroscopy. The elevator or bone tamp is gently tapped with a mallet, elevating the head out of valgus into the reduced position, restoring the normal neck–shaft angle. Characteristically, in a valgus-impacted proximal humerus fracture, once the head fragment is reduced, the intact periosteal sleeve between the fracture fragments causes the tuberosities to fall spontaneously into the reduced position. Occasionally, the lesser tuberosity may still be displaced medially and can require lateral traction via a small hook in the subdeltoid space to



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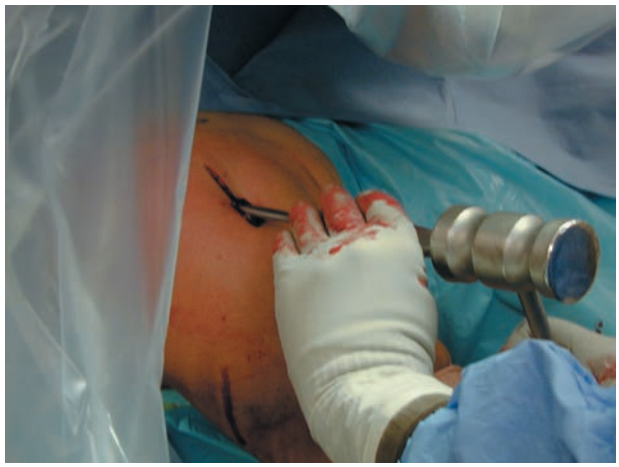

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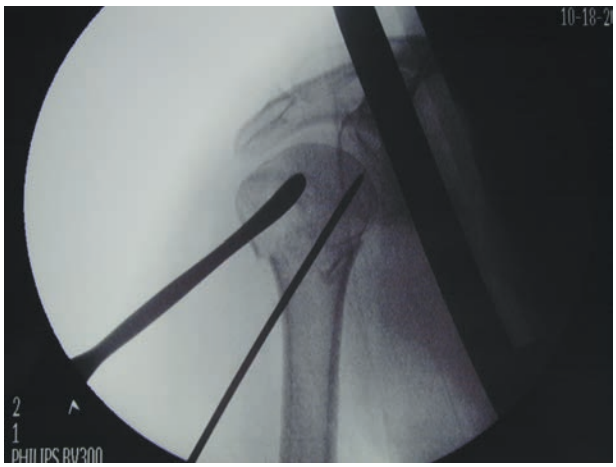


C

Figure 28-6 **(A)** A blunt-tipped elevator or a small bone tamp is placed through the reduction portal to lie deep to the lateral margin of the head fragment. The position of the instrument is confirmed with the C-arm fluoroscopy. **(B)** The elevator or bone tamp is gently tapped with a mallet, elevating the head out of valgus into the reduced position, restoring the normal neck–shaft angle. **(C)** When the head is raised, the periosteum on the medial side acts like a hinge, and the greater tuberosity returns to its anatomic position as a result of the inferior pull of the periosteum and the superior pull of the rotator cuff. (Adapted from Resch H, Povacz P, Frohlich R, Wambacher M. Percutaneous fixation of three- and four-part fractures of the proximal humerus. *J Bone Joint Surg Br* 1997;79(2):297.)

bring it into the anatomic position. Final reduction is confirmed with the fluoroscopic images. The pins are then placed under fluoroscopic guidance.

Overzealous impaction with the mallet is a potential pitfall, leading to loss of cancellous bone in the head fragment and potential fracture. Valgus-impacted fractures can be reduced by this closed technique only before healing has taken place. Ideally, surgery is recommended within 2 weeks of injury. Beyond that time point, more aggressive manipulation is required to mobilize the head fragment.

Fixation Technique

For the fixation of the head–shaft fracture plane, we use 2.5- or 2.7-mm terminally threaded pins. Terminally-threaded K-wires or, alternatively, the guidewires from the Synthes (Synthes, Paoli, PA) 7.3-mm cannulated screw set can be used. Fully threaded pins are not used in order to protect the soft tissues. Terminal threads are desirable to minimize the risk of pin migration. Pins are inserted through very small incisions. A scalpel is used to incise only the skin. A curved hemostat or similar instrument is used to dissect down to the periosteum and to mobilize soft tissue to avoid neurovascular injury. A protective sheath such as a drill guide should be used to prevent soft tissue from being caught inadvertently, and it helps guide the pin insertion. Two to three retrograde pins are placed from the shaft into the head fragment. The pins should enter the skin distal to the site where the pins actually enter the bone to obtain the correct angle so that the pins do not cut out posteriorly before gaining fixation in the head fragment. The entry points of the pins should be distal enough to the fracture line not only for adequate purchase of the intact humeral shaft cortex, but also to avoid injury to the anterior branch of the axillary nerve. However, the entry points should also be proximal to the deltoid insertion to avoid injury to the radial nerve. The direction of the pins is generally anterolateral to posteromedial (about 30 degrees) because of the anatomic retroversion of the humeral head. The pins should not be placed directly in the coronal plane, or they will cut out anteriorly. The starting points of the pins should not be too close to one another to avoid a stress riser in the lateral cortex. The pins should be widely spaced across the fracture site and within the head whenever possible. Furthermore, the pins should be multidirectional or multiplanar to maximize the stability of the construct. Two to three pins parallel to one another will act as a single axis of fixation and thus allow rotation. An additional retrograde anterior pin can be employed for further stability. When the anterior pin is placed, it should be kept in mind that the long head of the biceps tendon and the cephalic vein are just a few millimeters away from the pinning point. The pins are stopped within 0.5 to 1 cm of the subchondral bone.

The tuberosities are then secured. Pins or cannulated screws can be selected. We prefer fixation with cannulated screws because the proximal ends of the pins protrude through the deltoid and can cause irreparable damage to the muscle. Pins, if used, must be removed before starting early range-of-motion exercises for this reason. We prefer 4.5-mm cannulated screws to secure the tuberosities. The 4.5-mm screws have substantial guide pins and come in adequate lengths. The guide pins are placed under fluoroscopic guidance through the greater tuberosity, from superolateral to inferomedial, approximately 1 cm distal to the rotator cuff insertion, engaging the medial cortex of the shaft fragment (Fig. 28-7). At this point, care should be taken not to advance the pins past the medial cortex because of the vicinity of the axillary nerve and the posterior humeral circumflex artery. A screw with a washer is used, but one must be careful not to overtighten the screw as this can cause a fracture of the greater tuberosity. Two screws are ideal. The second screw can be a cancellous screw directed into the articular fragment.

Fixation of the lesser tuberosity is debatable in four-part valgus-impacted fractures. Once the humeral head and greater tuberosity are reduced and fixed, the lesser tuberosity is nearly always in the anatomic position. We generally prefer to leave the lesser tuberosity in the reduced position without additional fixation. This has not been found to result in any functional disability postoperatively. If the reduction is incomplete or unstable, a percutaneous cannulated screw (4.5 mm) can be placed from the anterior to posterior direction. Stability of fixation is assessed by gently rotating the arm under fluoroscopy. If there is any concern regarding stability, open reduction and internal fixation should be performed.

After percutaneous fixation, the pins are cut below the skin. The pins should be cut short enough to be buried entirely even after the initial swelling subsides and long enough for easy removal. Cutting the pin below the skin reduces the chance of superficial pin tract infection. All of these small incisions are closed using interrupted nylon suture (Fig. 28-8).

POSTOPERATIVE CONSIDERATIONS

Following the procedure, the affected extremity is immobilized in a sling for approximately 3 weeks. Active range-of-motion exercises of the wrist, elbow, and hand are encouraged. Radiographs are taken 1, 3, and 6 weeks postoperatively to ensure that no pin has migrated and no loss of reduction has occurred. The wound should also be inspected for any wound disruption or pin protrusion through the skin. Usually the pins become more prominent and occasionally protrude through the skin after initial swelling subsides after surgery. The pins should be cut short enough to avoid protrusion through the skin as this occurs. If the fracture reduction is felt to be stable and

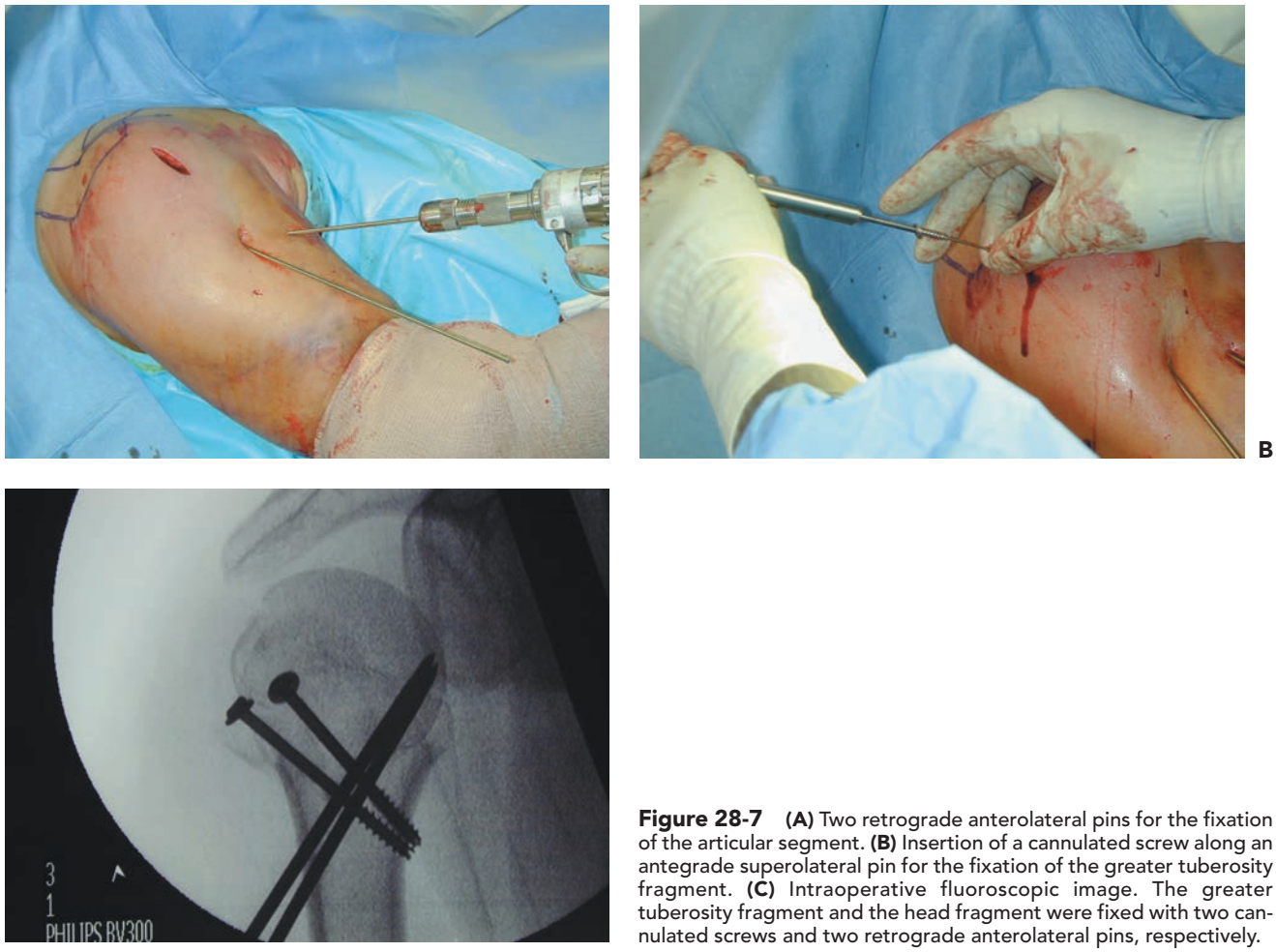


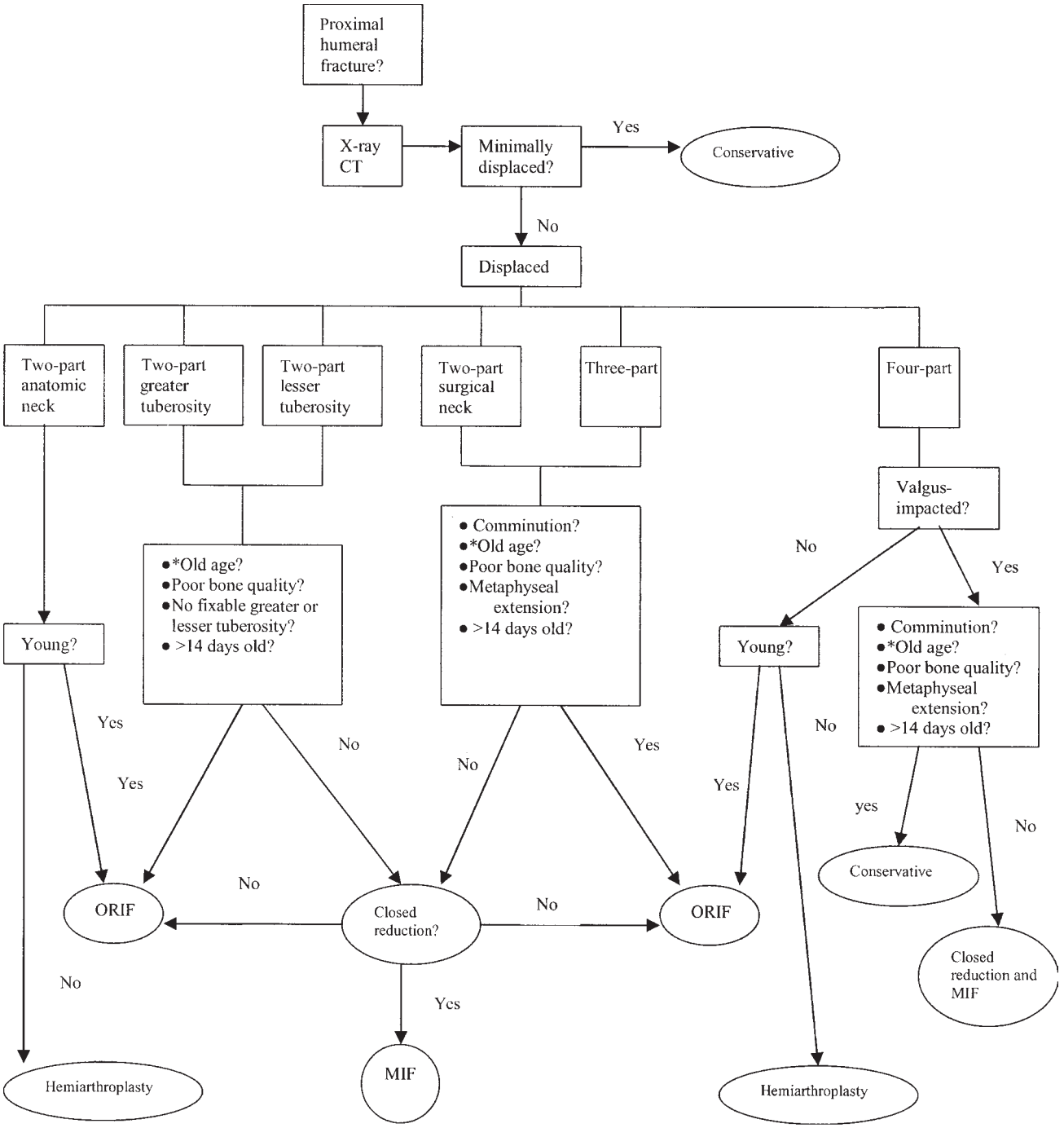
Figure 28-7 **(A)** Two retrograde anterolateral pins for the fixation of the articular segment. **(B)** Insertion of a cannulated screw along an antegrade superolateral pin for the fixation of the greater tuberosity fragment. **(C)** Intraoperative fluoroscopic image. The greater tuberosity fragment and the head fragment were fixed with two cannulated screws and two retrograde anterolateral pins, respectively.

screws have been used for fixing the tuberosities, pendulum exercises may be instituted 2 to 3 weeks postoperatively. If the fracture reduction was not solid at the time of the operation or pins have been used for fixing the tuberosities, exercises should not be started until 3 weeks after the surgery, at which time the pins of the tuberosities are removed. At 3 weeks, the passive forward flexion in the scapular plane and external rotation are started. Active assisted and active range-of-motion exercises are initiated at 6 weeks if there are signs of fracture healing. Progression to light strengthening exercises may start at that point. The pins are removed at 4 to 6 weeks. In a very unstable fracture configuration, it is best to leave the pins in for 6 weeks; however, loosening and migration of pins may necessitate earlier removal. The pins are removed either as an office procedure or in the operating room under local anesthesia, depending on patient and surgeon preference.

RESULTS

In the early report of Kocialkowski and Wallace[19], 22 cases of proximal humerus fractures were treated by closed reduction and percutaneous fixation using smooth K-wires. In their series, two cases were four-part fractures. A poor result by Neer's criteria[31] was seen in 69% of the older age group (older than 50 years), but all patients under 50 years had excellent results. The two four-part fractures developed avascular necrosis. The main complication was pin migration. In the cases studied, only smooth wires were used and they migrated in nine cases (41%). Despite this discouraging early report, many authors have tried to improve fixation methods and treatment guidelines. Several biomechanical studies on the strength of different fixation methods provided better understanding of patient selection and percutaneous fixation technique.[21,30,41]

Jakob et al.[17] presented their results of the treatment of 19 valgus-impacted four-part proximal humerus fractures. Five of these were treated closed. Although they did not specify the results of the five cases, they reported that they obtained satisfactory or excellent results in 74% of the patients with closed or limited open reduction and minimal osteosynthesis; these results are superior to those of conservative treatment. They reported an avascular necrosis rate of 26%, which is a lower incidence than that reported by Neer[32] for four-part fractures.



* Physiologically old (mentally, physically).

Figure 28-8 Treatment algorithm. CT = computed tomography; MIF = minimall invasive fixation; ORIF = open reduction and internal fixation.

Jaberg et al.[14] reported the results of 48 fractures fixed with percutaneous stabilization of unstable fractures of the proximal humerus fractures. In this series, closed reduction was performed and the fractures were stabilized with terminally threaded 2.5-mm AO pins placed in both antegrade and retrograde fashion. This series had 29 fractures of the surgical neck, three of the anatomic neck, eight of three-part fractures, five of four-part fractures, and three fracture–dislocations. Overall, 70% had good or excellent results, 20% fair, and 8% poor. Complete avascular necrosis

with collapse of the humeral head developed in only two patients (one from a two-part anatomic neck fracture and the other from a four-part fracture). However, eight had localized avascular necrosis that resolved over 1 or 2 years. These were thought to represent subtotal avascular necrosis. The fractures united by 6 to 8 weeks postoperatively except in the two cases of avascular necrosis.

Resch et al.[36] published their results of closed reduction and percutaneous fixation of nine three-part fractures and 18 four-part fractures using screws and K-wires. None of the three-part fractures went on to avascular necrosis, and all had good or very good results. There was an 11% incidence of avascular necrosis in the four-part fractures. Among 18 four-part fractures, five had significant lateral displacement of the humeral head. One of these five required revision 1 week after the surgery, and one went on to late avascular necrosis.

Closed reduction and percutaneous fixation of 19 cases of two- and three-part fractures were reported by Chen et al.[8] They used cannulated screws for all the cases except young patients with open physes. Twelve patients had two-part fractures, and six had three-part fractures. By Neer's criteria, overall, 84% had good or excellent results, 10% had fair results, and 5% (one patient) had a poor result. Eleven two-part fractures (84%) and five three-part fractures (83%) had excellent or good results. No significant correlation of fracture type to functional result was noted in their series.

Soete et al.[39] recommended not using the closed reduction and percutaneous fixation technique for four-part fractures because the both of two poor results in their series came from the two four-part fractures. However, these were not the valgus-impacted type.

Herscovici et al.[12] reported their results of closed reduction and percutaneous fixation for 41 proximal humerus fractures. They compared the failure rates of three different percutaneous fixation instruments. There was no failure with using Schanz pins (2.5-mm terminally threaded). There was one (20%) failure with 2.5-mm terminally threaded Dynamic Hip Screw guide pins and 100% failure rate with 2-mm K-wires. There were 21 two-part fractures, 16 three-part fractures, and four four-part fractures. The four-part fractures were not the valgus-impacted type. All patients with four-part fractures did not respond to the fixation and three of them had avascular necrosis develop, irrespective of the type of pin used. In the remaining 33 patients with two- and three-part fractures, a union rate of 94% was observed. One three-part fracture developed avascular necrosis. They recommended against the percutaneous fixation technique for the treatment of four-part fractures, although none of the four-part fractures in their study was the valgus-impacted type.

As previously discussed, the closed reduction and percutaneous fixation technique has been advocated for the treatment of relatively young patients with good bone stock. Recently, Zingg et al.[44] achieved good results in elderly patients (average age 72) with the closed reduction and percutaneous fixation technique. They evaluated the functional and subjective results of 31 patients using the Constant score and Oxford Shoulder Score. Injured shoulders achieved an average function of 82.6%, compared to the uninjured side. The subjective results were very good in 58% of the patients, good in 22%, satisfying in 9%, and poor in 9%. One fracture developed avascular necrosis. They thought the technique was a valuable method for the fixation of the proximal humerus fractures in elderly patients.

COMPLICATIONS

Nerve Injury

The most worrisome potential complication of percutaneous fixation is nerve injury. There is no published incidence of nerve injury after percutaneous fixation, yet most authors commonly expressed their concerns about potential nerve injury. Nerves at risk are primarily the axillary, the musculocutaneous, and to a lesser extent the radial nerve. The axillary nerve courses posteriorly through the quadrangular space and then is divided into the anterior and posterior branches just beneath the inferior capsule of the glenohumeral joint. The anterior branch courses anteriorly on the undersurface of the deltoid and is located approximately 3 to 6 cm distal to the lateral border of the acromion. When making the anterolateral reduction portal, the deltoid should be gently and bluntly spread to avoid any nerve traction. This incision is usually located superior to the zone where the nerve runs; however, one should be cautious during this part of the procedure. The anterior branch of the axillary nerve is also at risk when placing screws through the greater tuberosity, and thus a drill guide can be inserted more superiorly and gently advanced distally to keep the nerve from the path of the drill.

An anatomic study by Rowles and McGrory[38] investigated the spatial relationship between percutaneously inserted pins and neurovascular structures. According to their study, the proximal lateral retrograde pins were located a mean distance of 3 mm from the anterior branch of the axillary nerve. They recommended making the starting point for all lateral pins distal to a point along the lateral aspect of the shaft equal to twice the distance from the top of the humeral head to a line perpendicular to the shaft at the inferiormost margin of the articular cartilage of the humeral head (Fig. 28-9). A safe zone for lateral pins was found between this point and deltoid tuberosity distally. The proximal antegrade greater tuberosity pins were found to have their tips at a mean distance of 7 mm from the posterior humeral circumflex artery and 6 mm from the axillary nerve with the arm in a neutral rotation. These distances decreased with internal rotation and increased with external rotation. The anterior pin was located adjacent to the long head of the biceps tendon, 11 mm from the cephalic vein, and could potentially be near the musculocutaneous nerve. These findings emphasize the importance of

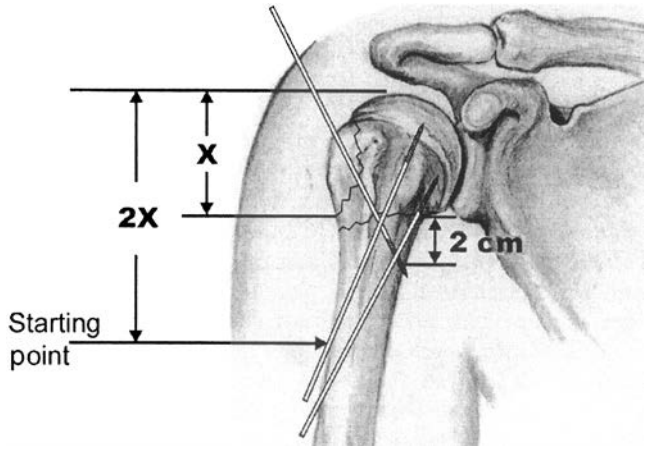


Figure 28-9 Illustration of the proposed starting point for placement of the lateral pins and the end point for the greater tuberosity pins. The starting point for the proximal lateral pin should be at or distal to a point twice the distance from the superior aspect of the humeral head to the inferiormost margin of the humeral head. The greater tuberosity pins should engage the cortex of the humeral neck greater than or equal to 20 mm from the inferiormost aspect of the humeral head. (Reprinted with permission from Rowles DJ, McGrory JE. Percutaneous pinning of the proximal part of the humerus. An anatomic study. *J Bone Joint Surg Am* 2001;83-A(11):1697.)

using a protective sleeve at the time of drilling. The radial nerve will not be injured as long as the retrograde pins are inserted proximal to the deltoid insertion.

Pin Migration

The most common complication is pin migration. Most commonly, the pins back out and become prominent under the skin. Proximal migration into the joint is possible. Percutaneous pinning requires very close follow-up and strict patient compliance. Serious complications of pin migration can be prevented by following patients with radiographs at regular intervals. Sudden increase of pain in either the shoulder or arm may indicate a migrating pin. If the pin is loose, protrudes though the skin, and hinders the rehabilitation, it should be removed. If a pin migrates medially and penetrates the articular cartilage of the head, it should be removed immediately. It is strongly recommended that terminally threaded pins rather than smooth pins be used whenever possible. Moreover, one should be careful not to penetrate the head and back the pin out. This may diminish the mechanical advantage of the terminal threads of the pin and allow loosening.

Loss of Fixation

Loss of fixation may occur with any type of fracture fixation. In some situations, this can be treated with repeat percutaneous fixation. However, if it is felt that the fracture is unstable and further loss of fixation may occur, open reduction and internal fixation is recommended. Loss of fixation has been found to be associated with several factors: the method used for fixation, the severity of the fracture comminution, the bone quality, and patient compliance. According to the study of Wheeler and Colville,[41] the percutaneous pin fixation is unable to maintain rotation loads much greater than rotational loads seen during physiologic loading. They thought percutaneous pin fixation would be unlikely to withstand sudden impact loading and had the potential to fail under activities of daily living. This study suggests a need for more conservative rehabilitation after percutaneous fixation than the traditional open reduction and internal fixation. Patients are encouraged to wear slings for 3 weeks and the activities of daily living are usually delayed until there are signs of fracture healing and the pins are removed. Naidu et al.[30] reported a biomechanical comparison of several different percutaneous fixation constructs. Their study showed that for torsional stability, multiplanar fixation is more important than the number of cortices engaged. On the other hand, bending stiffness increases with the number of cortices purchased. They suggested that a multiplanar pin construct is needed to optimize torsional stiffness, and additional tuberosity pins are needed to augment bending stiffness. A wide pin spread within the fracture can increase stability.

Pin Tract Infection

Superficial infections of the pins have been reported with an incidence of from 0% to 23%.[8,12,14,19,36,39] Jaberg et al.[14] reported four (9%) superficial pin tract infections out of 48 patients, which all resolved with the removal of the pins and local wound care. There was one deep infection in a diabetic patient. In their series, the pins were left through the skin. Because of this risk, we prefer to cut the pins deep to the skin.

Avascular Necrosis of the Humeral Head

The incidence of avascular necrosis after closed reduction and percutaneous fixation of the proximal humerus fracture has been reported to be from 0% to 16%.[8,12,14,39] According to Jaberg et al.,[14] there are two forms of avascular necrosis of the humeral head: complete, with collapse of the humeral head; and subtotal, localized with transient cyst formation and sclerosis of the humeral head. The patients with complete necrosis went on to hemiarthroplasties. The localized necrosis group had moderate pain in the early period. The pain and radiologic changes gradually resolved, showing a slower functional recovery, and all had good or excellent results. This gradual improvement in function and radiographic appearance is thought to result from creeping substitution phenomenon of the humerus head. This localized avascular necrosis of the humeral head was also observed by other authors.[20,24]

Malunion and Nonunion

Residual displacement of the humeral head at the surgical neck is usually well tolerated as long as the tuberosities are well reduced in relation to the humeral head. However, malunited tuberosity fractures are often symptomatic. If the displacement of the malunited greater tuberosity is severe enough to cause impingement in the subacromial space, open surgical intervention is required. Nonunion has almost never been reported.

Rotator Cuff Tear

Recently, Robinson and Page[37] increased concern about rotator cuff tears after four-part valgus-impacted fractures of the proximal humerus. In their study of 29 open reductions and internal fixations augmented with special bone substitute for valgus-impacted fractures, 11 patients had a substantial tear (2.5 cm in length) in the rotator cuff. The tear propagated through the rotator interval longitudinally. They recommended repair of this torn rotator cuff interval. We have not experienced any case complicated by neglected rotator cuff tear and do not know if this repair is necessary.

CONCLUSION

Minimally invasive fracture fixation has considerable potential in the proximal humerus. With the advantages of soft tissue and bone preservation, early results suggest that in selected fracture types, percutaneous pinning is a desirable option. Healing rates are excellent and complications are low. This procedure requires significant technical expertise; however, with an understanding of normal and fracture anatomy, it can be mastered and used to benefit many patients.

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29 Hemiarthroplasty for Proximal Humerus Fractures

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Charles A. Rockwood, Jr.

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INTRODUCTION AND INDICATIONS

The use of a prosthesis in managing fractures of the proximal humerus is ordinarily indicated in patients over 50 with four-part fracture–dislocations, simple four-part fractures, and many three-part fractures when there is osteopenia, comminution, and general debilitation of the patient.[34] In the patient physiologically and/or chronologically less than 50 years of age we would use closed pinning or perform open reduction and internal fixation rather than using a prosthesis. If rigid fixation is not achieved such that we could begin early postoperative range of motion and/or articular surfaces are damaged, then we would use a prosthesis in these cases as well. The less frequently encountered head-splitting fractures and displaced anatomic neck fractures should also be considered for treatment with arthroplasty.

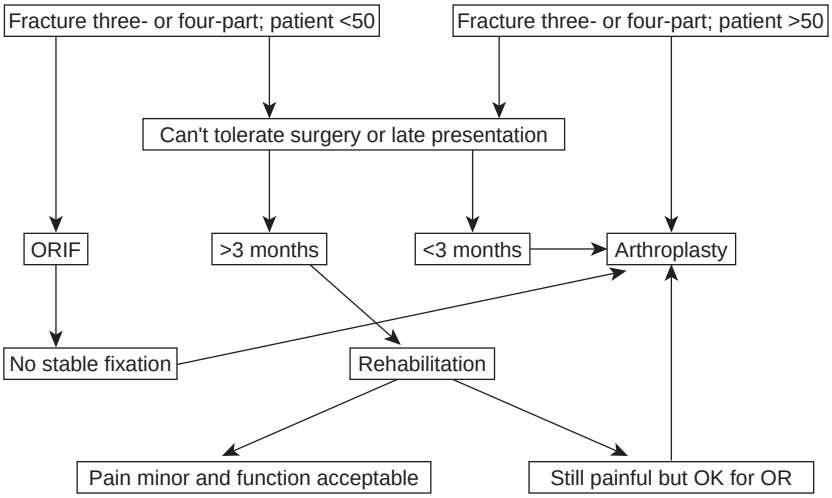
The timing of the reconstruction is important to the outcome. These fractures are best treated in the acute phase (i.e., during the first 7 to 14 days following the fracture). As time passes, the complexity of the intervention increases and the predictability of the outcome decreases.[2] The inferior outcomes when performing late arthroplasty for these injuries are largely related to fixed soft tissue contractures and inability to restore the tuberosities to their anatomic locations.[25] The treatment algorithm illustrates the basic principles of treatment decision making.

Shoulder arthroplasty for the treatment of complex proximal humerus fractures represents a formidable challenge. The surgeon should be honest with him- or herself regarding his or her ability and volume of experience.[17,20,24] The management of a displaced three- and four-part fracture of the proximal humerus is one of the most difficult shoulder problems faced by orthopedic surgeons.

SURGICAL TECHNIQUES

Prosthetic Design Considerations

Since 1991 we have used the DePuy Global Fx modular prosthesis (DePuy [Johnson and Johnson], Warsaw, IN) for



Algorithm 29-I A treatment decision algorithm. ORIF = open reduction and internal fixation.

treatment of complex fractures or fracture–dislocations of the proximal humerus. The use of a modular design has the advantage of allowing the surgeon greater latitude for soft tissue balancing by using various humeral head sizes. The Global Fx prosthesis is different from the Global Advantage prosthesis, which is used to manage patients with arthritis of the shoulder. The key difference is that the proximal body and fins are much smaller and allow for the relocation of the greater and lesser tuberosities around the prosthesis and fixation of the tuberosities to themselves and to the upper shaft of the humerus. The smaller size of the upper body also allows for room for autogenous or bone grafting into any defects between the tuberosities and the shaft of the humerus. The Fx is also available with proximal porous coating of the body for tuberosity healing.

The Global Fx prosthesis utilizes a jig, which allows the surgeon to place, adjust, and then hold the height and retroversion of the prosthesis while the surgeon places the shoulder through a trial range of motion. The various steps of how to use the fracture jig are described in the surgical technique.

Regardless of which system the operating surgeon uses, some additional universal considerations should be noted. First, the relationship of the fins to the version of the prosthesis must be known. Restoring the proper retroversion of the head will affect the anterior fin and lateral fin position, and this reference should be understood for appropriate reduction of the tuberosities. Second, the presence of a medial hole is desirable to allow horizontal stem-to-tuberosity fixation. The cerclage technique and other means of horizontal fixation have been demonstrated to provide increased interfragmentary stability.[1312] The geometry of the proximal body may also play a role in fixation stability as an irregular shape improves the effects of a cerclage compared with a smooth circular design.[35]

Preoperative Planning

Surgeons treating these fractures have only one chance for an optimal outcome because the results of revision surgery are less predictable and generally inferior to primary cases.[2] We emphasize that, if possible, displaced three- and four-part fractures should be managed by an orthopedist who has surgical experience with these injuries.[20] There are contraindications to surgical treatment that include active infection, previous neurologic defects, inability to medically tolerate surgery, and an uncooperative patient. It is not uncommon to have an associated nerve injury with a proximal humerus fracture, and treatment of these patients presents a dilemma. As most of the nerve injuries resolve, we have elected to treat these patients acutely and explore the nerve injury at the time of fracture repair.

It is important to have a lengthy discussion with the patient about the goals of the surgery. Unlike patients with arthritis, these patients generally exhibit near-normal shoulder function prior to fracture. They must understand that surgery will not provide a normal shoulder. With replacement surgery the desired outcome is good pain relief and functional use of the shoulder. One should aim to accomplish four key objectives at the time of surgery: anatomic sizing of the prosthesis, restoration of humeral length, prosthetic retroversion of 20 to 25 degrees, and secure and anatomic tuberosity fixation.

Patient Positioning

The patient is placed in a semi-Fowler or beach-chair position on the operating table (Fig. 29-1A). The standard head rest portion of the table should be removed and replaced with a versatile headrest such as a McConnell (McConnell Orthopedic Mfg. Co., Greenville, TX). This allows free

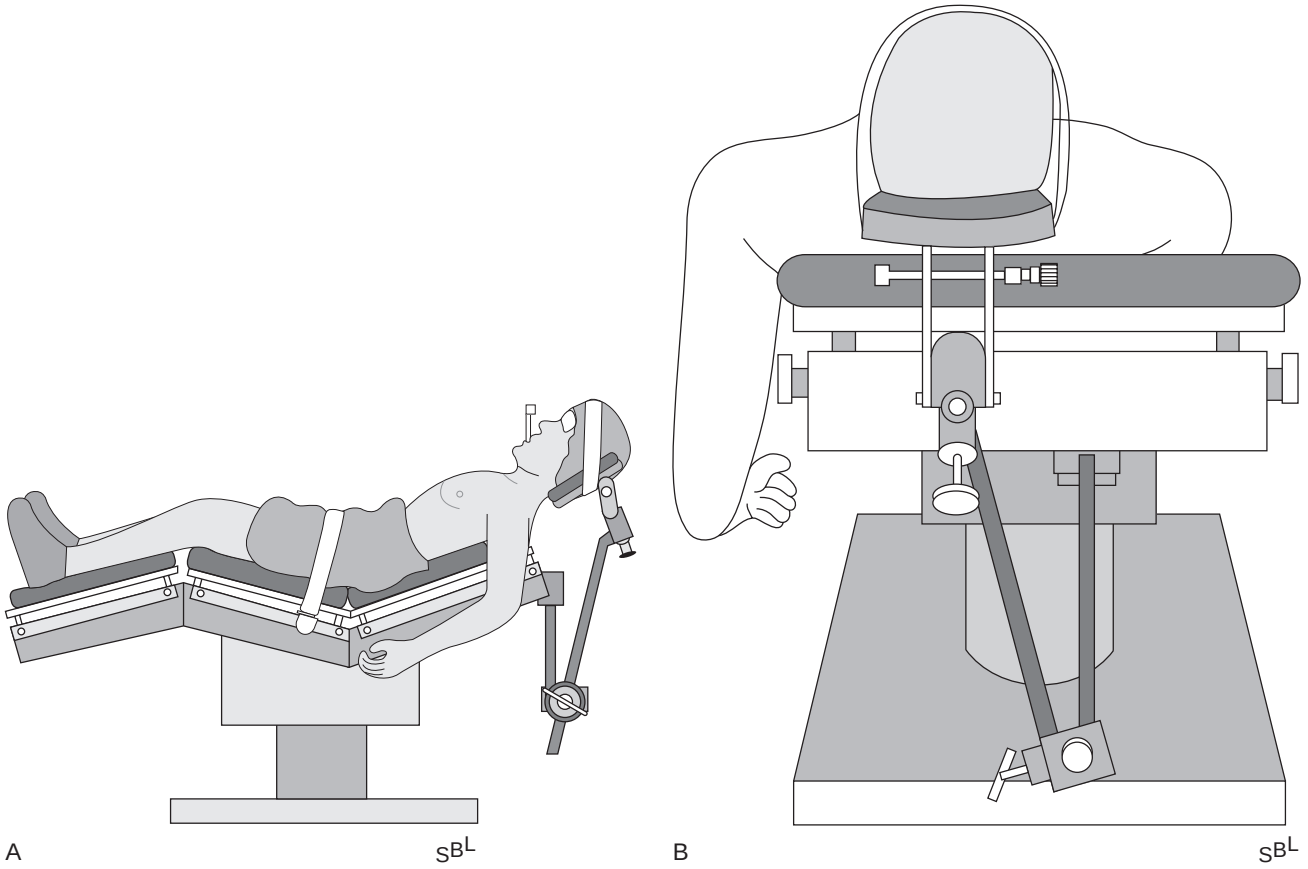


Figure 29-1 **(A)** Patient positioning (side view). **(B)** The involved shoulder should extend over the edge of the table.

access to the superior aspect of the shoulder region. The patient is positioned so that the involved shoulder extends over to the top corner and edge of the table (Fig. 29-1B). The patient's head is secured to the head rest with tape and the anesthesia equipment is draped to isolate it from the sterile field. Caution should be used to ensure that the brachial plexus is not under undue tension when securing the head to the head rest. A kidney post may be helpful to keep the patient's thorax secure on the operating table.

Procedure

An incision running from the clavicle, over the top of the coracoid, and down to the anterior aspect of the arm is utilized (Fig. 29-2). Once the incision has been made, the cephalic vein is located on the deltoid muscle near the deltopectoral interval. The cephalic vein is usually firmly imbedded in the deltoid, and there are many venules from the deltoid into the cephalic vein (Fig. 29-3). For this reason, we recommend that the vein not be sacrificed, but reflected laterally with the deltoid muscle. The deep surface of the deltoid is freed from the underlying tissues, from its origin on the clavicle down to its insertion in the humeral shaft. When the anterior margin of the deltoid has been

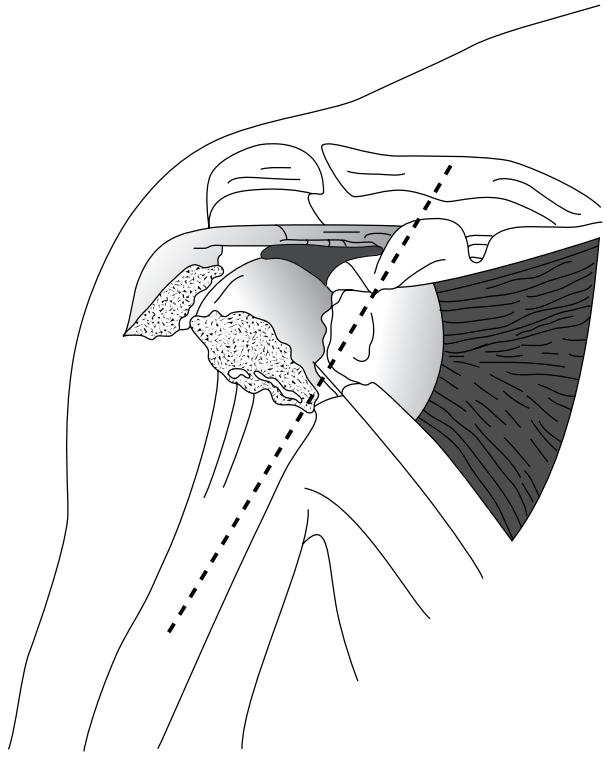


Figure 29-2 Deltopectoral incision.

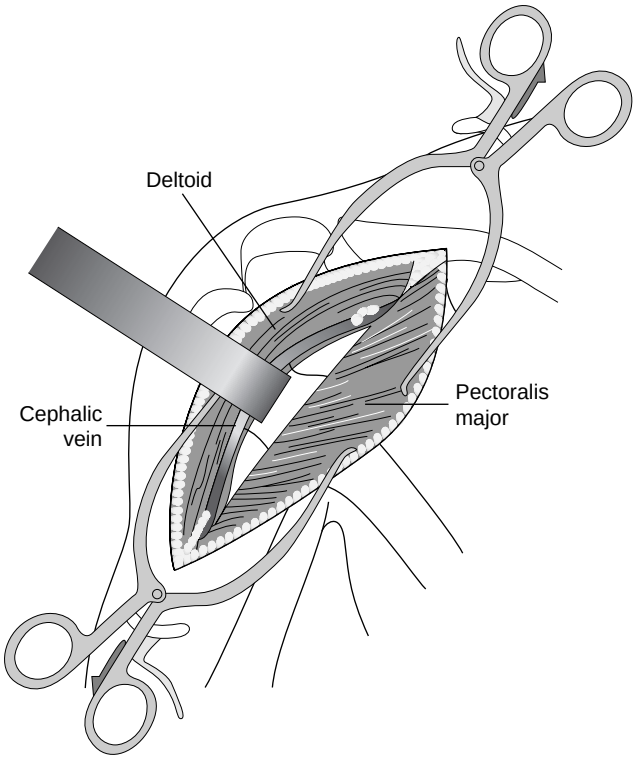


Figure 29-3 Retract the cephalic vein laterally to preserve the venous drainage of the deltoid muscle.

completely freed from its origin to its insertion, especially along its deep surface, the arm is abducted and externally rotated, which allows the deltoid to be gently retracted laterally with one or two Richardson retractors. The deltoid muscle is protected with a moist lap sponge, and the conjoined tendon is retracted medially with a large Richardson retractor.

Release of the insertion of the upper portion of the pectoralis major tendon from the humerus with an electrocautery cutting blade improves exposure of the inferior aspect of the joint. Ordinarily in a complex fracture of the proximal humerus, the circumflex vessels may be disrupted. However, when these vessels are visualized, ligation or cauterization will help control bleeding and improve exposure.

In fracture cases it is especially important to identify and protect the musculocutaneous and axillary nerves. The musculocutaneous nerve is palpated as it comes from the plexus into the posteromedial aspect of the conjoined tendon (Fig. 29-4). Usually, the nerve penetrates the muscle 1 to $1^1/_2$ in. inferior to the tip of the coracoid, but in some instances the nerve has a higher penetration into the conjoined muscle–tendon unit. It is important to remember the location of this nerve when retracting the conjoined tendon. The all-important axillary nerve is located by passing the volar surface of the index finger sown along the anterior surface of the subscapularis muscle. The index finger is hooked anteriorly to identify the axillary nerve (Fig. 29-5). Gentle traction on the lesser tuberosity fragment makes it

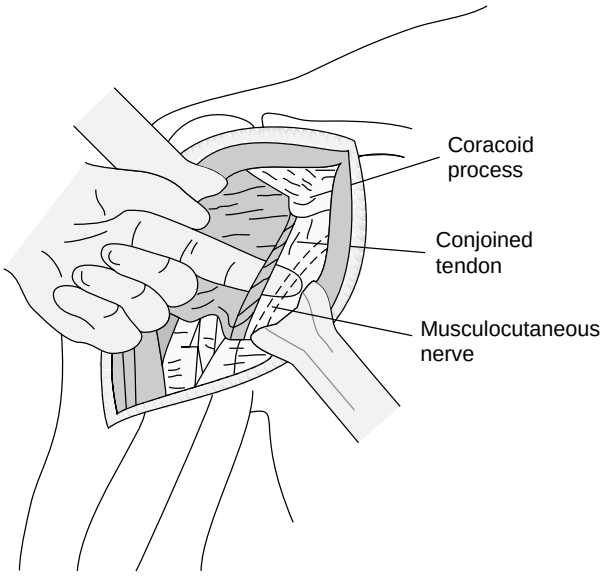


Figure 29-4 Palpate the musculocutaneous nerve beneath the conjoined tendon.

easier to identify the axillary nerve. Occasionally, as with a chronic fracture of the proximal humerus, the nerve will be adherent to the anterior surface of the subscapularis and difficult to locate. When this occurs, an elevator can be passed along the anterior surface of the muscle to create an interval between the muscle and nerve. The axillary nerve is always identified and carefully retracted (when freeing up the lesser tuberosity) to protect it from injury.

The biceps tendon is an excellent landmark to identify the interval between the lesser tuberosity and the greater tuberosity (Fig. 29-6A,B). First a pair of scissors is placed

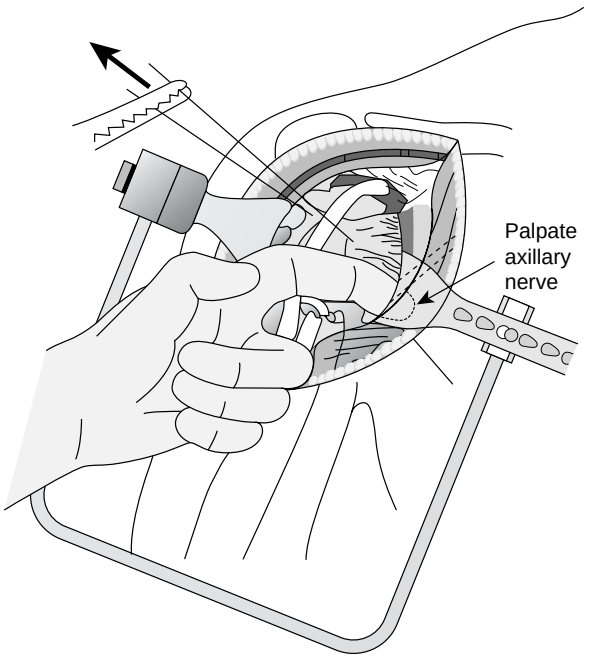


Figure 29-5 Palpate the axillary nerve.

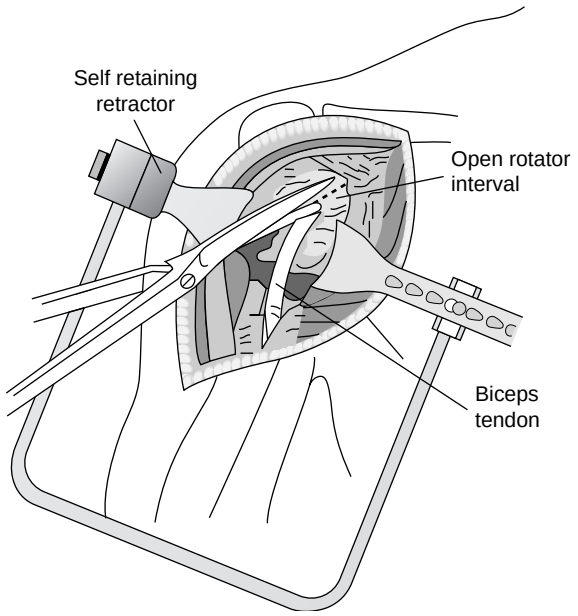


Figure 29-6 Identify the long head of the biceps and release the transverse humeral ligament and rotator interval.

into the sheath of the biceps tendon and used to divide the transverse humeral ligament. Continuing proximally with the scissors, the interval between the subscapularis and the supraspinatus tendons is opened all the way up to the insertion of the biceps tendon into the supraglenoid tubercle. If the biceps tendon has been ruptured, the scissors are placed in the bicipital groove and used to open the interval between the subscapularis and the supraspinatus tendon.

The lesser tuberosity is freed from the underlying humeral head and soft tissues. In similar fashion, the greater tuberosity is carefully identified and mobilized with its attached muscles (Fig. 7A,B). Occasionally the greater tuberosity will be in several fragments. When this occurs, we secure the major fragments together with heavy nonabsorbable sutures so that the majority of the supraspinatus and infraspinatus tendons can be repaired with the fragments at the time of closure. The greater and lesser tuberosity fragments are sufficiently mobilized so that they can be easily repaired around the prosthesis and to each other at the time of closure.

With the tuberosities out of the way, a bone hook or clamp is used to retrieve the humeral head. Once the humeral head has been excised, its dimensions are measured with a template (Fig. 29-8). The humeral head should be transferred to the back table, where an assistant can remove all of the cancellous bone from beneath the articular surface (Fig. 29-9). The autogenous cancellous bone will be used at the time the tuberosities are repaired to the shaft.

At this point, the arm should be extended down off the side of the table, which will deliver the shaft up and out of the wound. Fragments of bone and blood clot are removed from the canal of the humeral shaft. Next the tuberosities are further mobilized so that they will be ready for approximation around the prosthesis, to one another, and to the humeral shaft. The operating surgeon may choose to place the sutures for tuberosity fixation before or after setting the position of the stem. If exposure is limited, it is helpful to place the sutures before the prosthesis.

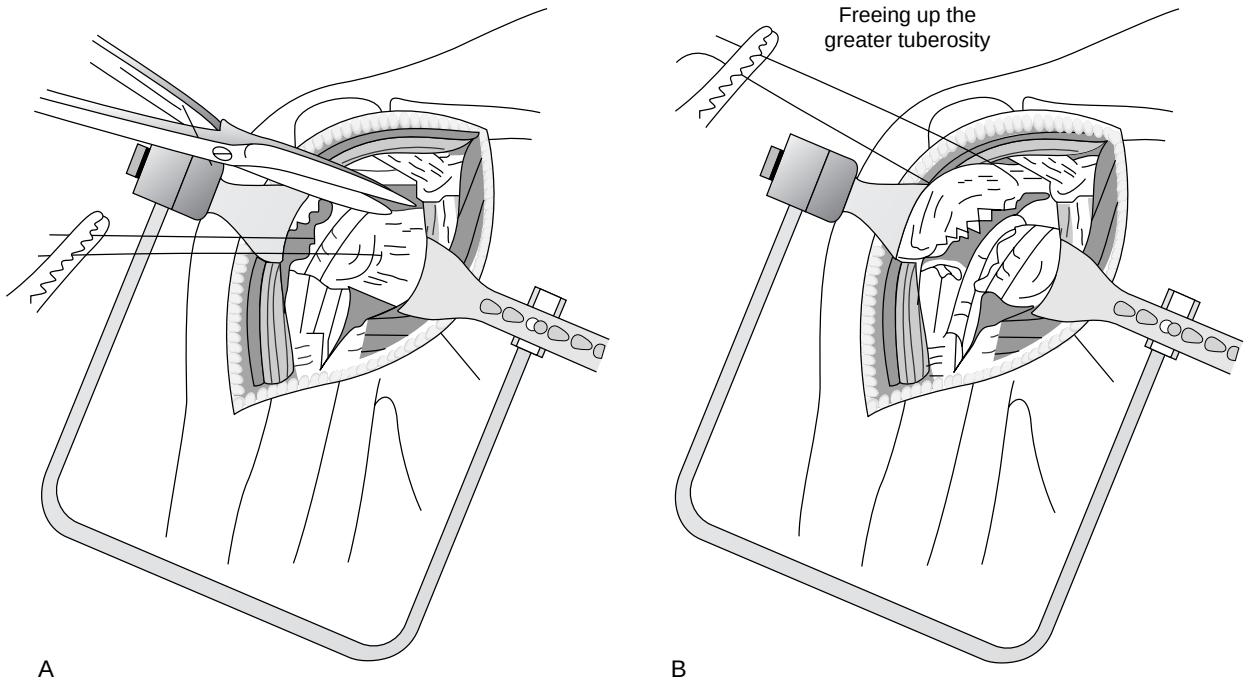


Figure 29-7 **(A)** Freeing the lesser tuberosity. **(B)** Freeing the greater tuberosity.

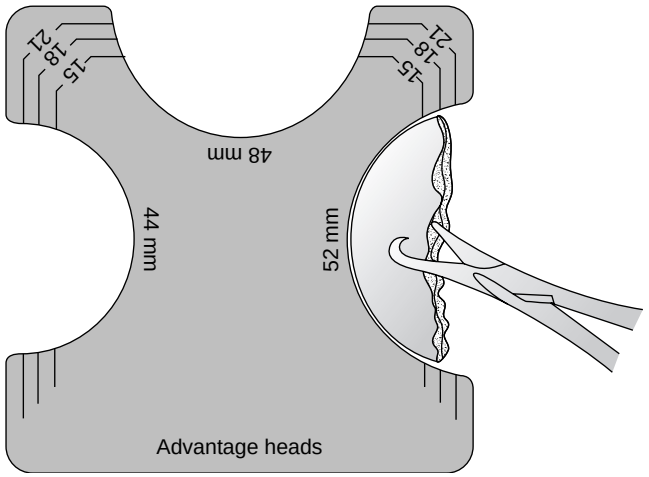


Figure 29-8 Measure the resected humeral head height and diameter with the template to select the head component.

The tuberosity-to-tuberosity fixation is accomplished with heavy nonabsorbable suture placed in a simple or inverted mattress fashion at the insertion of the rotator cuff on the bony tuberosity. The suture fixation at the bone–soft tissue interface is superior to the cancellous bone alone.

Technique without Using the Fx Jig

The proper size humeral head trial is attached to a small humeral broach body and placed into the intramedullary canal. The size of the broach should be small enough to allow the prosthesis to sit down on the upper shaft. In some situations, when trying to determine the correct size trial body, it is important to put in a stem that will fill the shaft of the humerus. If a stem is used that is too big, it will hold the prosthesis too proud. Similarly, a very small stem will allow the prosthesis to sit all the way down to where the collar is sitting on the shaft of the humerus. Usually, by experimenting with different modular trial bodies, it will be noted that the flare of the upper portion of the prosthesis will hold the prosthesis at the near-perfect height. The assistant applies gentle traction to the arm with the forearm held in 0 degrees of rotation. While the assistant maintains traction on the arm, the surgeon uses a tool to lift the collar of the prosthesis so that the head of the prosthesis is at the level of the glenoid fossa (Fig. 29-10). The distance between the top of the shaft of the humerus and the collar of the prosthesis is carefully measured, as it will correspond to the height that the final prosthesis must be cemented to in the proximal humerus. The length of the back surface of the humeral head to the humeral shaft should equal that of the greater tuberosity fragment. Both the trial and final prosthesis have horizontal 5-mm laser marks that can be used to determine the height of the prosthesis up and out of the shaft.

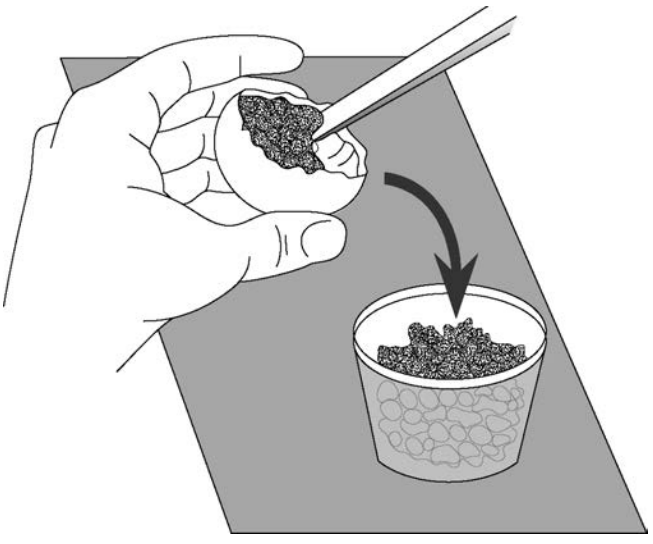


Figure 29-9 Obtain cancellous bone from the resected humeral head for later use as a bone graft.

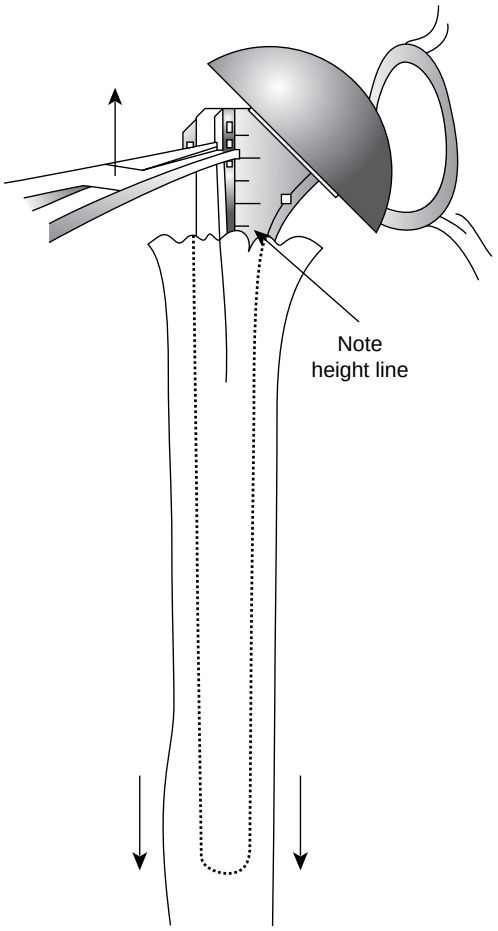


Figure 29-10 Set the height of the component.

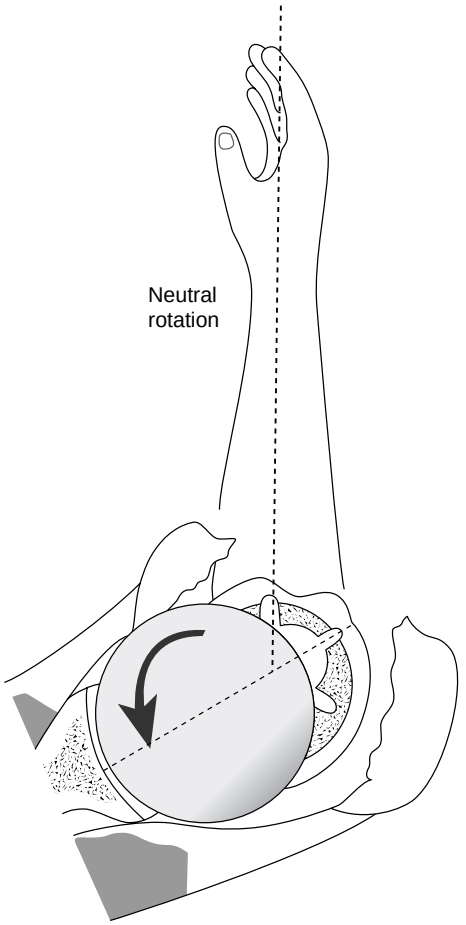


Figure 29-11 Rotate the prosthesis posteriorly to face the glenoid.

The next step will be to determine the proper amount of retrotorsion of the prosthesis. While traction is still being applied to the arm in neutral rotation, the prosthesis is rotated posteriorly until the head of the prosthesis is facing directly into the glenoid fossa. This will correspond to the 20 to 25 degrees of retrotorsion of the head and neck as seen in a normal humerus. A rongeur is used to make a notch in the anterior shaft of the humerus in direct line with the anterior fin of the prosthesis (Fig. 29-11). This will ensure correct retrotorsion of the humeral component when it is cemented into the humeral canal.

Technique When Using the Fx Jig

The Global Fx jig is used to hold the prosthesis at the selected height and retroversion while trialing reduction and range of motion. The surgeon attaches the humeral clamp to the upper shaft of the humerus and then aligns the vertical height gauge to the anterior fin of the trial prosthesis. The fin clamp is attached to the middle hole in the anterior fin (Fig. 29-12). The prosthesis is placed to allow anatomic or near-anatomic reduction of the tuberosities

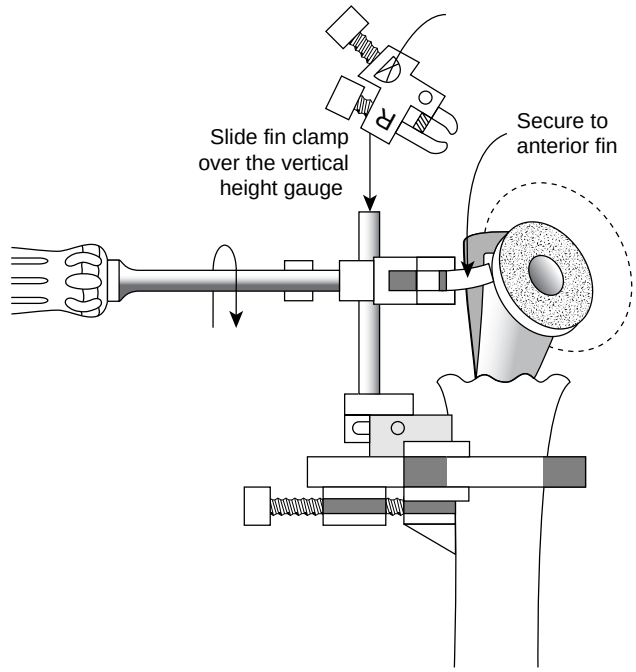


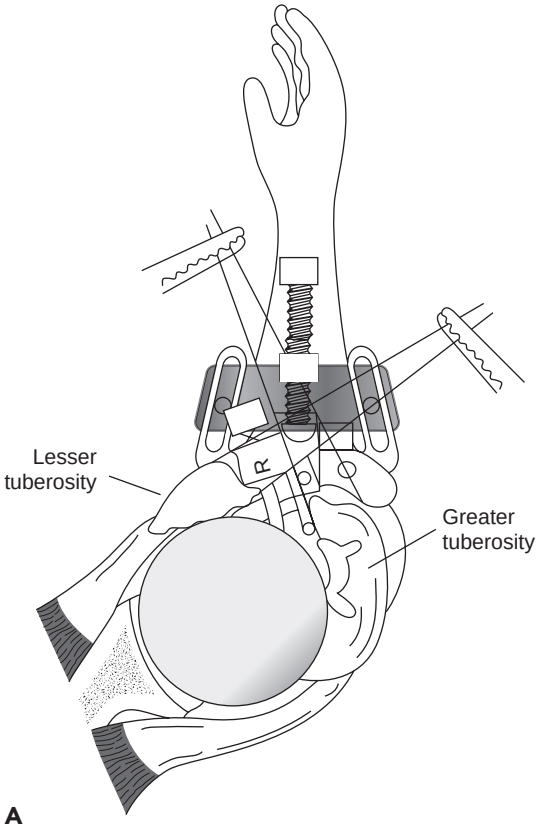
Figure 29-12 Slide the fin clamp over the vertical height gauge.

(Fig. 29-13A). The height is adjusted and noted on the vertical height gauge. It is sometimes helpful to use fluoroscopy to evaluate the height of the prosthesis in relation to the reduced tuberosities and glenoid (Fig. 29-13B). If the jig is aligned perpendicular to the epicondylar axis, this will automatically retrovert the prosthesis 20 to 25 degrees. If more or less retroversion is appropriate, this is accommodated by rotation of the jig on the humeral shaft. The shoulder range of motion can now be tested and minor adjustments made as desired (Fig. 29-14A,B). Proper height of the prosthetic is determined by fitting the greater tuberosity in the interval between the back side of the humeral head to the fractured shaft.

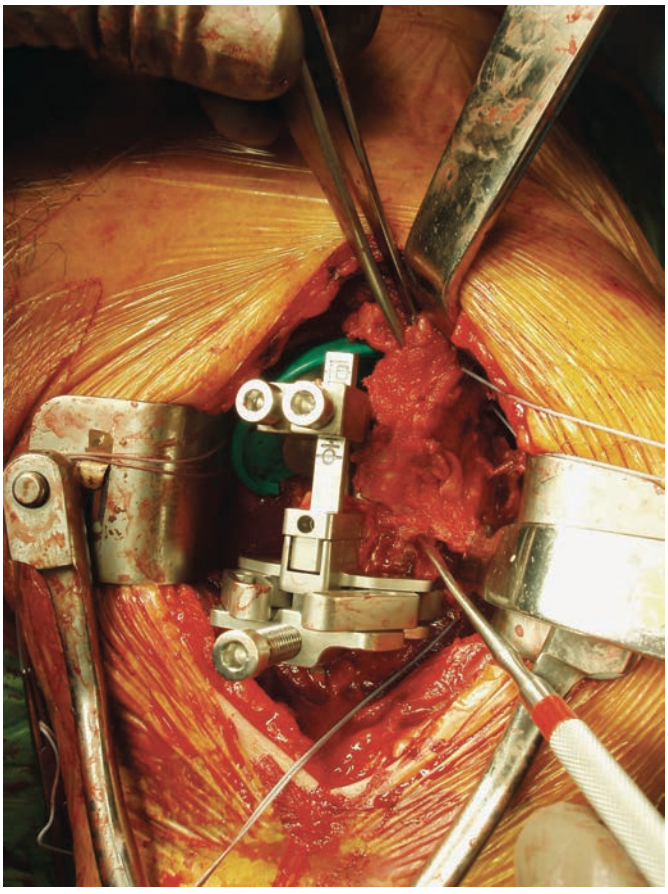
When the proper height and torsion of the trial prosthesis has been determined, two or more drill holes are made in the proximal anterior–medial and proximal anterior–lateral humeral shaft. Heavy nonabsorbable sutures are passed through these holes to secure the tuberosities back to the shaft of the humerus (Fig. 29-15).

Rather than cementing the body of the prosthesis into the humerus and then later attaching the humeral head, it is best to firmly secure the head to the prosthesis prior to implantation. The humeral body is manually supported in the impact stand on the head, then attached to the body with five of six blows from a 1 or 2-lb mallet (Fig. 29-16).

The medullary canal is thoroughly irrigated to remove blood and other debris. A vent tube is inserted down the medullary canal and bone cement is pushed down into the upper humerus with finger pressure (Fig. 29-17). Alternatively, a cement gun can be used but it is not pressurized. A bioabsorbable collagen cement retainer is used. The proper



A



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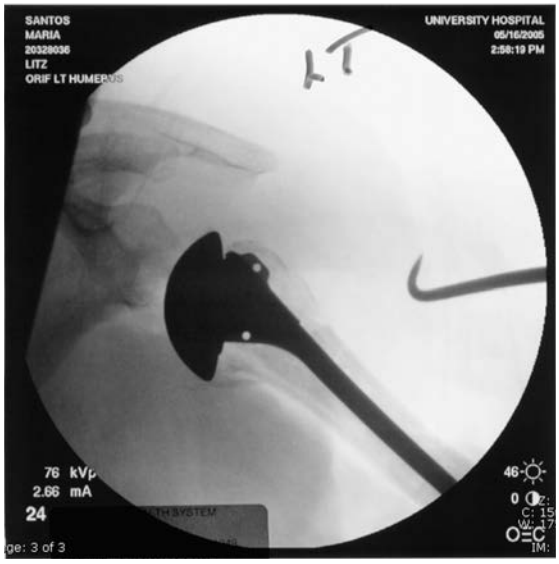


C

Figure 29-13 **(A)** Reduce the tuberosities around the positioning jig. **(B)** Intraoperative photograph demonstrating the use of the fracture jig with a trial stem and head. The greater tuberosity is used to help determine the proper height of the prosthetic by using it as an internal "ruler" to determine the proper distance between the back side of the prosthetic head and the proximal fracture of the humeral shaft. The height of the prosthesis is adjusted on the jig until the prosthetic height allows for perfect reduction of the greater tuberosity fragment. **(C)** Use fluoroscopy to confirm tuberosity reduction.

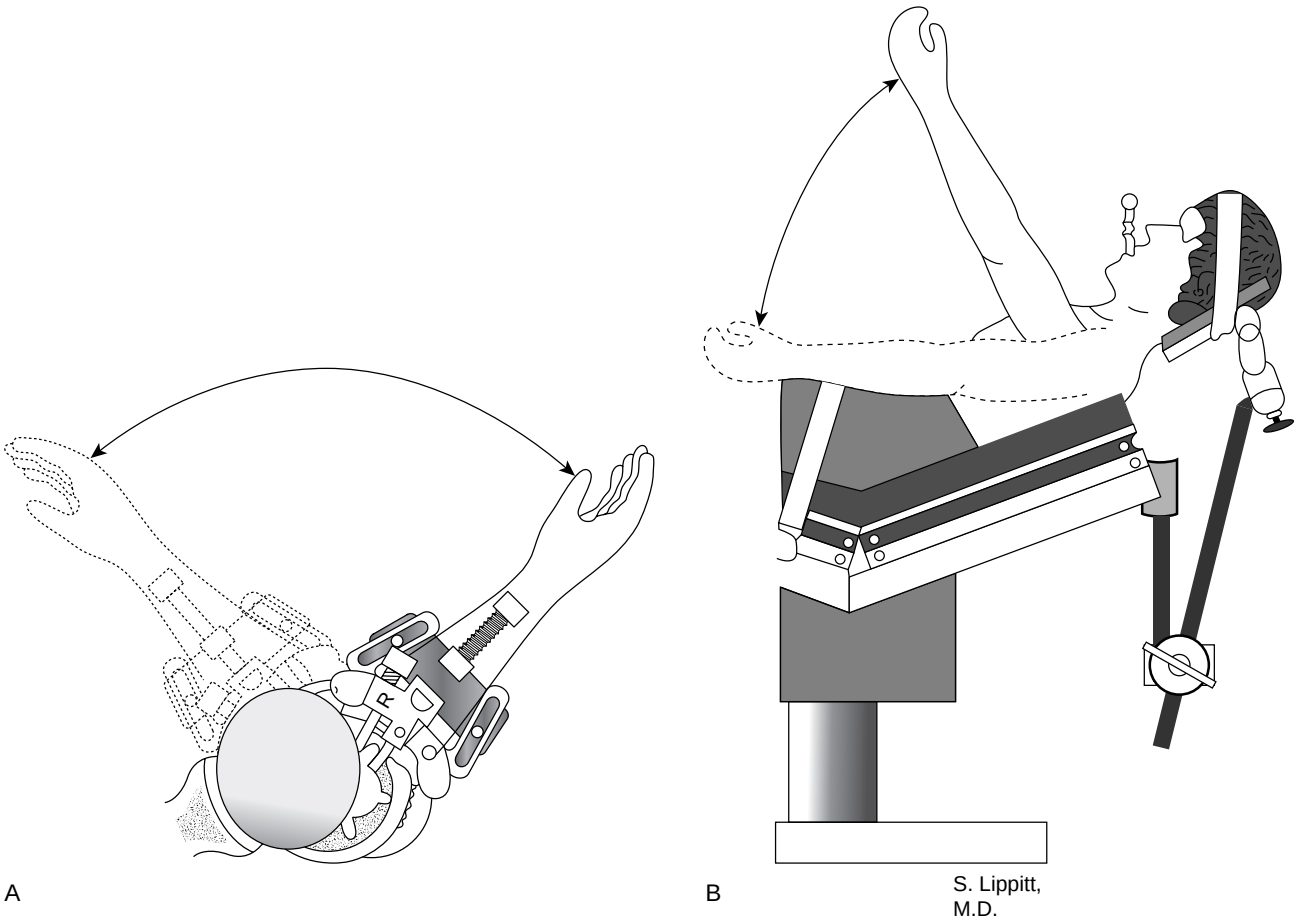


Figure 29-14 **(A)** The positioning jig allows testing of motion and stability in internal and external rotation. **(B)** The positioning jig allows testing of motion in forward elevation.

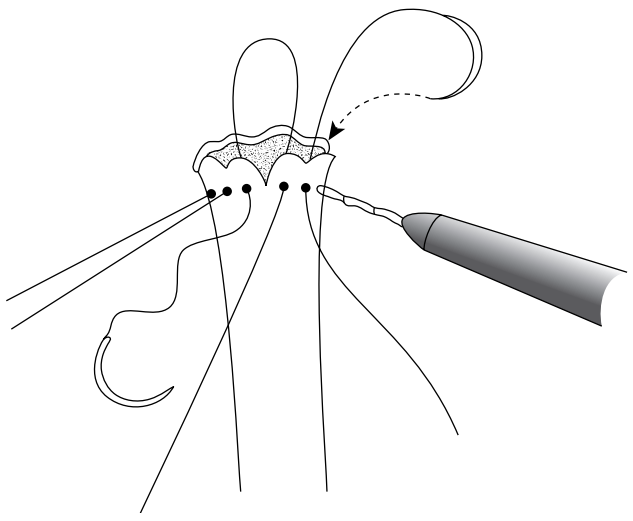


Figure 29-15 Drill holes for suture that will secure the tuberosities to the shaft.

size humeral prosthesis is inserted down into the medullary canal, with the surgeon being sure to maintain the proper retrotorsion by lining up the anterior fin with the previous notch in the anterior shaft of the humerus and by maintaining the proper degree of height out of the shaft or the humerus. Alternatively, the fracture jig can be left in position and used to position and hold the stem while the cement cures (Fig. 29-18).

When the cement has set, the prosthesis is reduced into the glenoid fossa. The cancellous bone graft from the humeral head is placed in the interval between the shaft of the humerus and the collar or neck of the prosthesis (Fig. 29-19). This additional bone graft will help to secure the healing of the tuberosities to the shaft and to each other. The tuberosities are then repaired back to the shaft of the humerus and to each other (Fig. 29-20). The heavy nonabsorbable tapes that were previously placed through the shaft of the humerus are passed through the tuberosities. If necessary, drill holes can be made in the tuberosities for secure fixation. If the tuberosities are fragmented, the sutures can be passed around the fragments and through the tendons, where they will provide firm fixation. If not

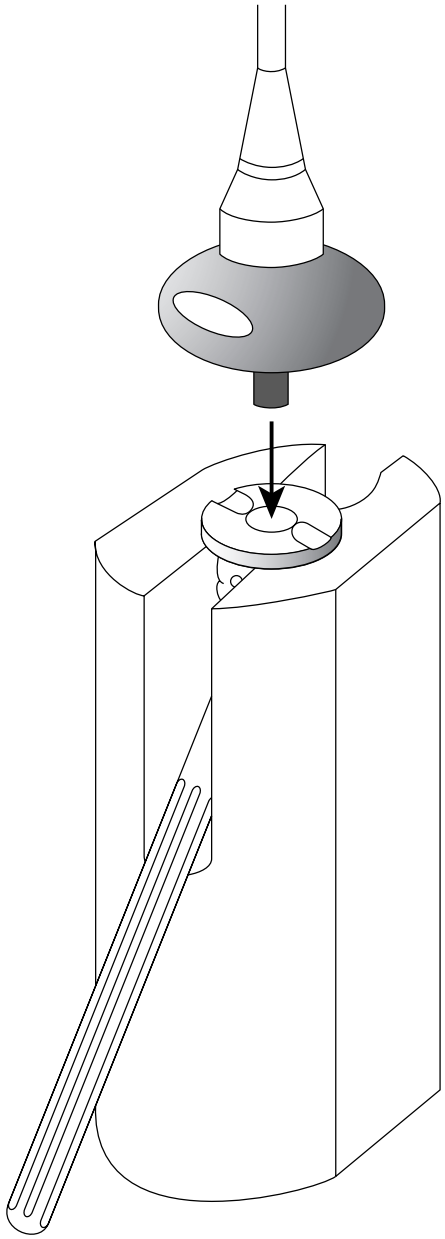


Figure 29-16 The head is secured to the body prior to implantation.

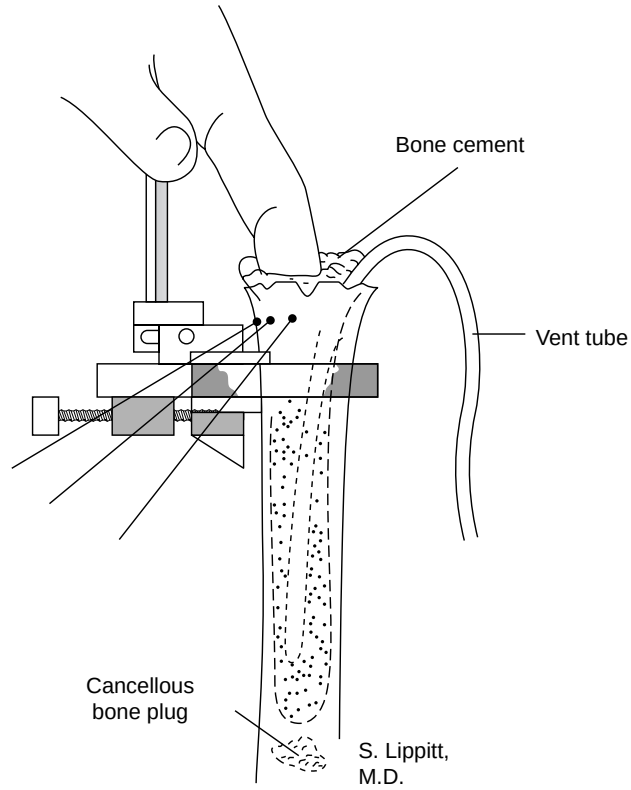


Figure 29-17 Cement is finger pressurized.

already done, nonabsorbable tapes are passed through the greater and lesser tuberosity fragments to secure them to each other. For additional stability, the sutures between the tuberosities can be passed through the holes in the anterior fin of the prosthesis (Fig. 29-21). To aid in repairing the tuberosities to each other, a towel clip is used to hold the tuberosities together while the sutures are tied. The split in the rotator interval is repaired with nonabsorbable sutures. The long head of the biceps that was released can be tenodesed in the interval between the tuberosities.

The wound is thoroughly irrigated with an antibiotic solution and a portable wound evacuation drain unit is placed to prevent the formation of a postoperative hematoma. The soft tissues are infiltrated with 0.25% Marcaine solution, which reduces the immediate postoperative pain and facilitates early passive motion of the shoulder on the same day as surgery. The wound is closed using 2.0 Vicryl in the deep subcutaneous layer and a running subcuticular nylon for the skin. We then obtain portable anteroposterior and axillary views of the shoulder to confirm component positioning before waking the patient (Fig. 29-22A–D).

Postoperative Management

Phase I

We agree with Neer that postoperative management is as important as the surgery itself and believe that early passive flexion and external rotation is the key to achieving satisfactory functional range of motion.[30]

Day 1: On the afternoon of the day of surgery, the sling is removed and the patient has free hand, wrist, and elbow motion. In bed, the patient begins passive forward flexion with the aid of an overhead pulley and passive external rotation with the aid of a stick. At the point the patient feels a good stretch, he or she holds this position for a five count. Each stretch is repeated five times. It is a great psychological benefit for the patient to see his or her arm

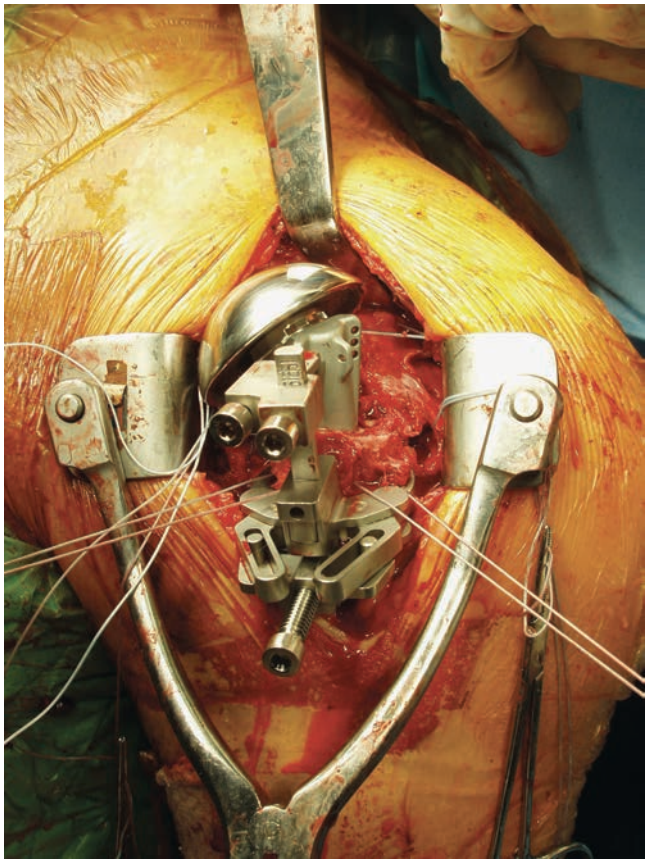


Figure 29-18 Intraoperative photograph of the use of the fracture jig to place the final prosthetic in the same place as the trial prosthetic. Once the cement is cured the jig is removed.

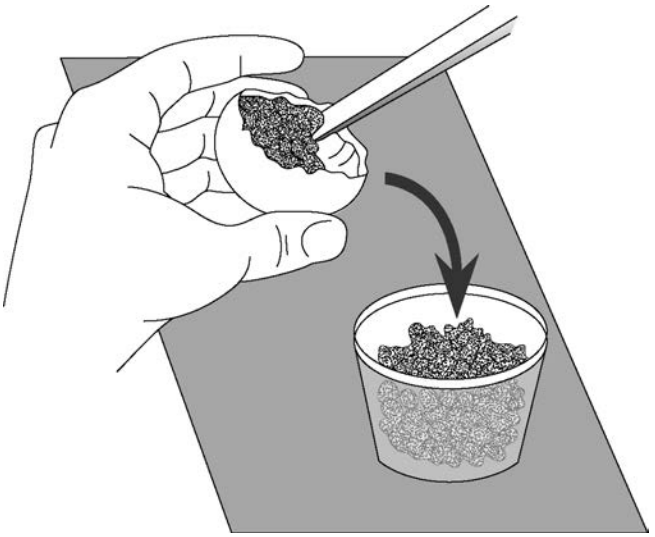
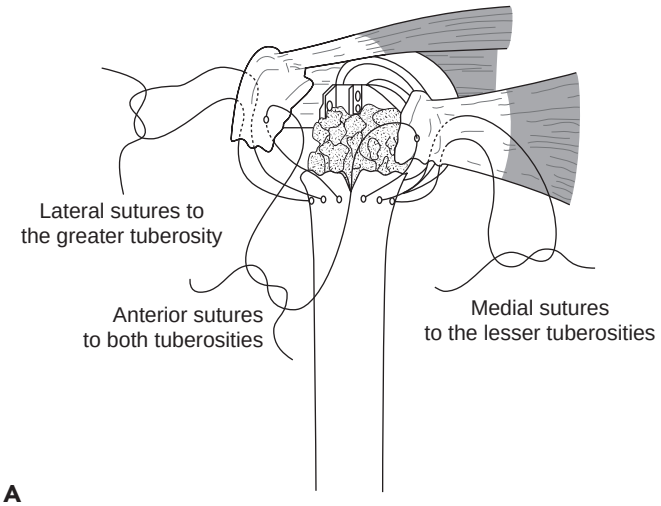
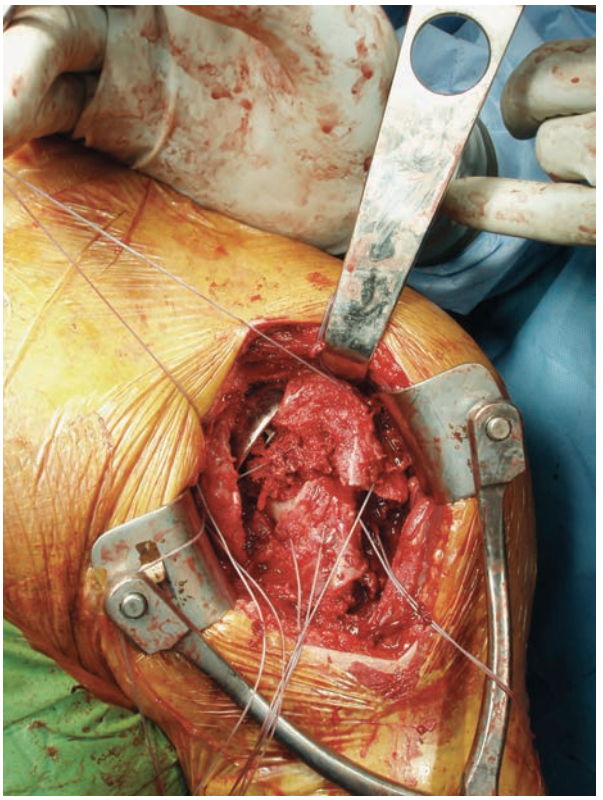


Figure 29-19 Place the cancellous graft around the prosthetic collar and the humeral shaft.



A



B

Figure 29-20 **(A)** Pass the sutures between the shaft and tuberosities. **(B)** Intraoperative photograph demonstrating the bone graft in place and the tuberosity to be fixed with heavy nonabsorbable sutures.

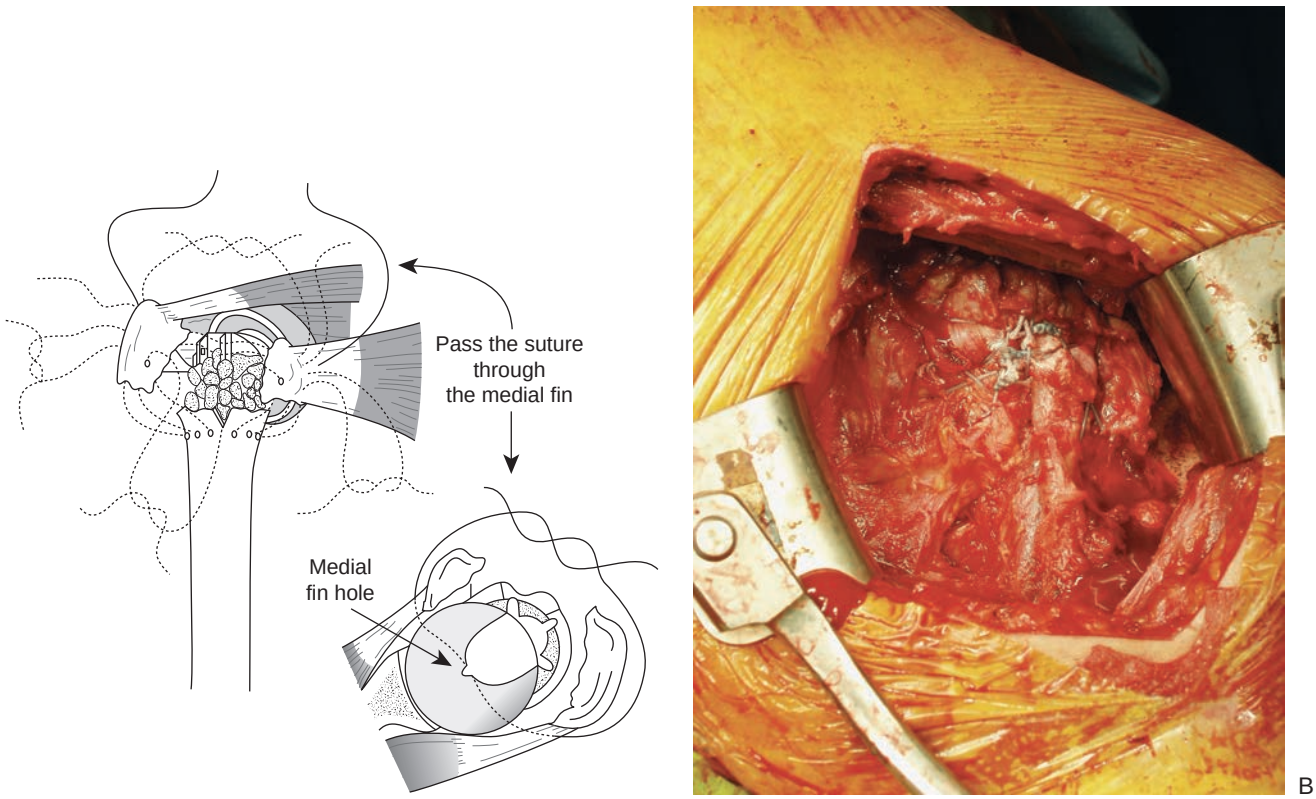


Figure 29-21 **(A)** Pass the sutures between the greater and lesser tuberosities and then cerclage the tuberosities with a suture passed through the medial fin. **(B)** Intraoperative photograph of the final fixation of the tuberosities.

moving before much postoperative pain and swelling set in. Patients are better able to relax physically and mentally as they perform further rehabilitation.

Day 2: On the first postoperative day pendulum exercises are added to the stick and pulley movements to be performed four to six times a day. The patient is helped out of bed and taught not to push off on the operated arm. Independent ambulation and incentive spirometry are encouraged. The patient also begins gentle everyday living activities such as brushing teeth, combing hair, eating, and drinking.

Day 3 to week 6: On postoperative day 2 the patient is ready for discharge home with a pulley kit and stick that will allow him or her to continue the passive motion rehabilitation. By 2 weeks the subcuticular suture is removed and the sling that had been worn in public is discontinued.

Phase II

Usually by 6 to 8 weeks the patient has achieved nice easy passive overhead elevation and is ready to begin resistive exercises. The exercises are designed to strengthen the three parts of the deltoid, rotator cuff, and scapular stabilizer muscles. With the arm at the side, external rotation, internal rotation, abduction, forward flexion, and extension motions are started in a controlled 45-degree arc. Each motion is performed for a five count against resistance provided by a graduated set of Therabands. The resistance is increased when the exercises are done easily without pain, usually at 2- or 3-week intervals.

RESULTS

We treated 28 patients with the above techniques, and at 3.5 years of follow-up (range 1 to 7 years) the overall visual analog scores for shoulder pain, function, the ability to use the arm at work and play, and overall quality of life were 16, 21, 22, and 18, respectively (0 = best and 100 = worst). The patients responded yes to a mean of 66% of Simple Shoulder Test activities. The mean active range of motion was 118 degrees of forward elevation (range 30 to 160 degrees), 28 degrees of external rotation (range 10 to 50 degrees), and internal rotation to T11 (range buttock to T4). The mean preoperative Shoulder Security Index SSI of 35 improved to a mean postoperative value of 80. Those younger than 60 and patients that had not previously been operated on demonstrated a comparatively superior improvement in outcome ($P < 0.05$). Reports in the orthopedic literature demonstrate that when operated on acutely, more than 80%

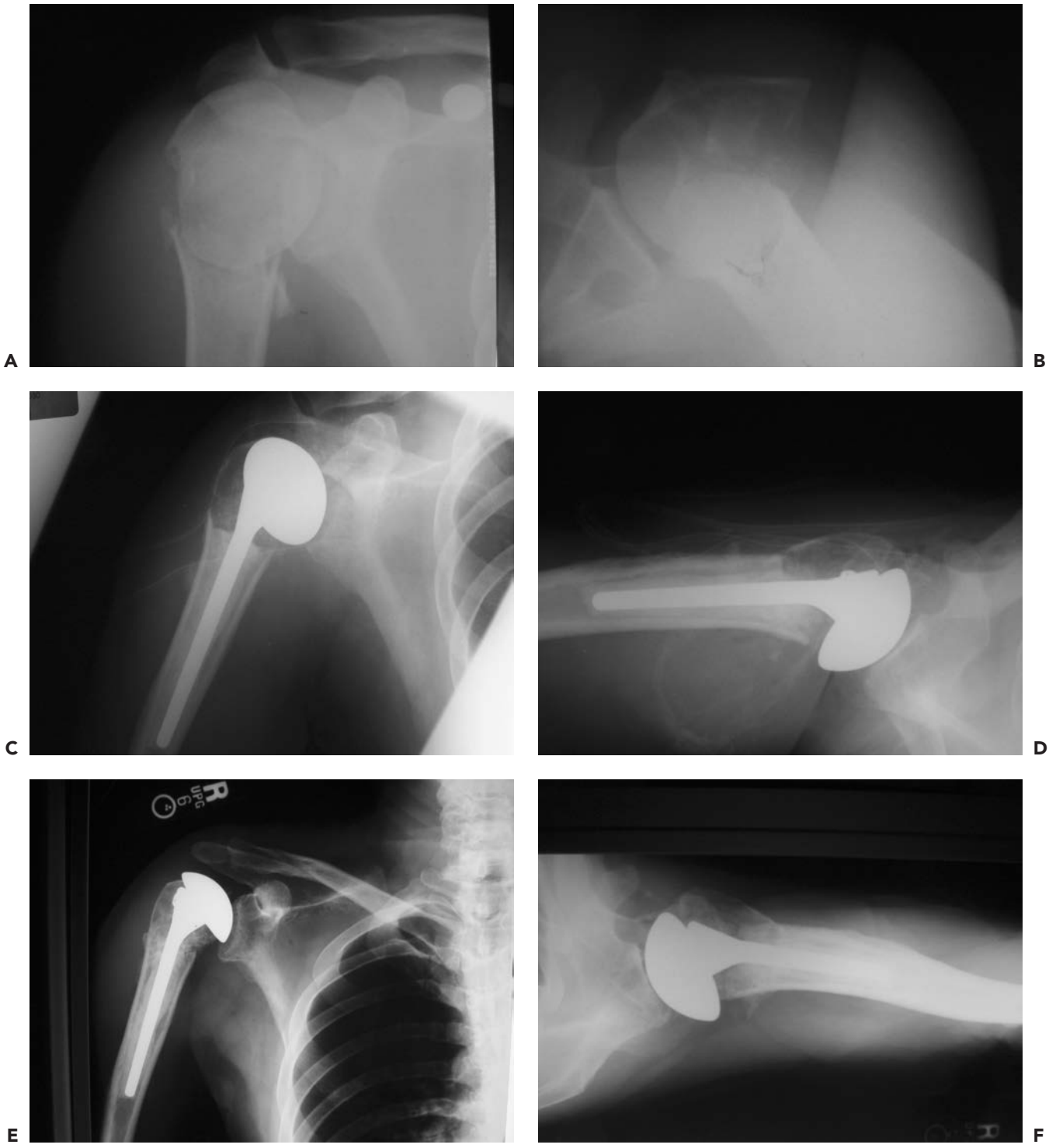


Figure 29-22 **(A)** Preoperative anteroposterior (AP) radiograph. **(B)** Preoperative axillary lateral radiograph. **(C)** Immediate postoperative AP radiograph. **(D)** Immediate postoperative axillary lateral radiograph. **(E)** Six-month postoperative AP radiograph. **(F)** Six-month postoperative axillary lateral radiograph.

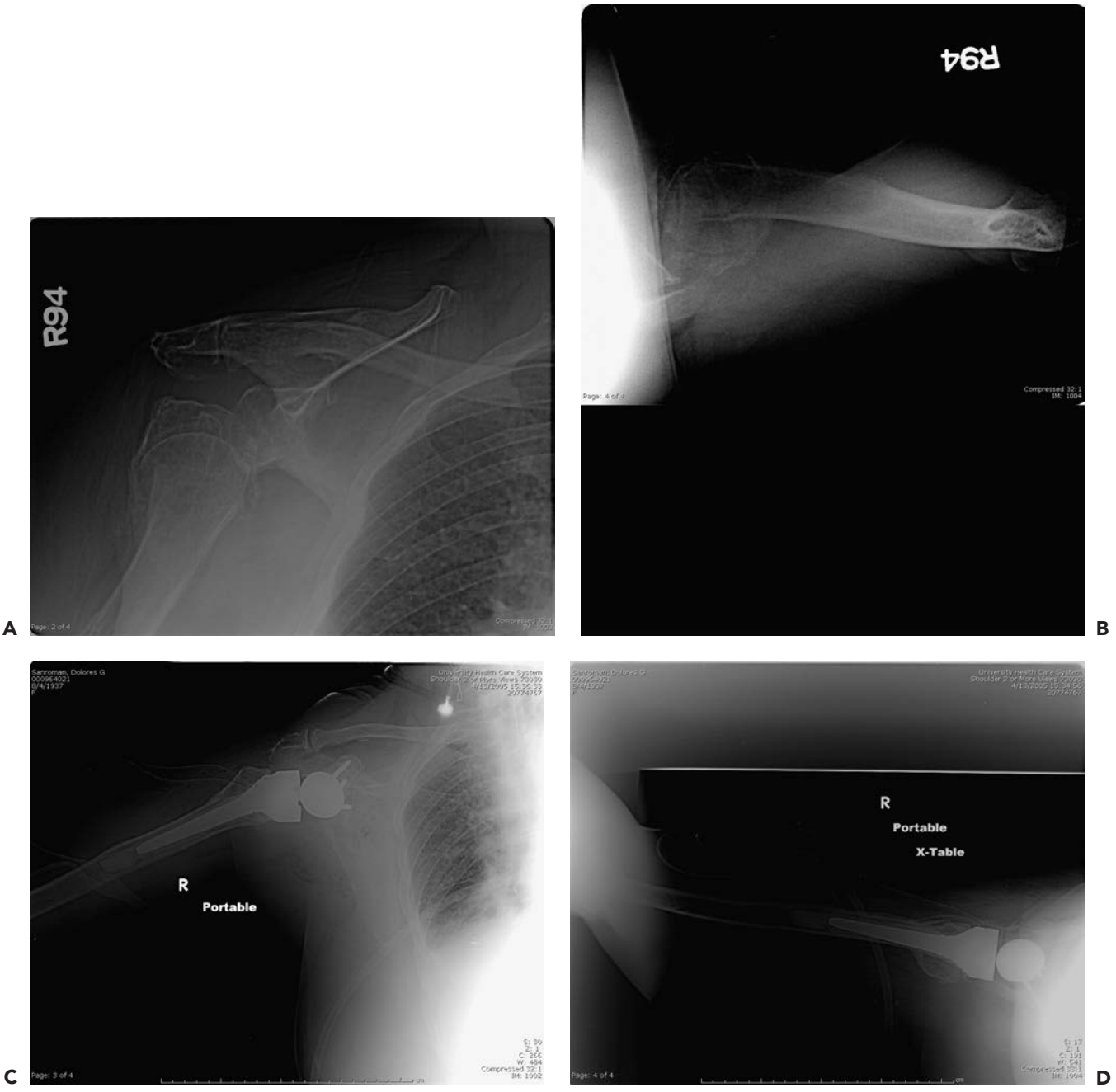


Figure 29-23 **(A)** Preoperative anteroposterior (AP) radiograph. **(B)** Preoperative axillary lateral radiograph. **(C)** Postoperative AP radiograph. **(D)** Postoperative axillary lateral radiograph.

of patients achieve reliable pain relief and satisfaction[18] (Table 29-1). However, postoperative motion is less predictable. The best results are achieved if technical errors and complications are avoided.[25,36]

Complications

Complications will arise despite meticulous technique and intraoperative caution. Stableforth reported a 6.1% incidence of brachial plexus injuries after fractures of the proximal humerus.[37] This emphasizes the importance of a careful preoperative examination to avoid confusion if a palsy is present in the postoperative period. If nerve injury is suspected either pre- or postoperatively, it should be explained to the patient and carefully followed. Electromyography and nerve conduction studies can follow the progress of the injury. If a palsy is present preoperatively, the continuity of the plexus can be confirmed at surgery. If the palsy appears postoperatively and there is no improvement by 3 months, early exploration may be indicated.

TABLE 29-1
OUTCOMES FOR SHOULDER HEMIARTHROPLASTY

Reference	Number of Shoulders	Patient Satisfaction	Pain Relief	Forward Elevation (degrees)
Gobel et al.[15]	20	14	16	75
Hawkins and Switlyk[19]	20	16	18	72
Tanner and Cofield[38]	16	14	16	101
Moeckel et al.[28]	22	20	20	119
Neer[30]	43	39	Nr	Nr
Wretenberg and Ekelund[40]	18	Nr	18	76
Zyto et al.[41]	27	19	Nr	70
Stableforth[37]	16	14	11	11–90
Neumann et al.[31]	22	18	22	120
Boileau et al.[2]	71	59	Nr	Nr
Kraulis and Hunter[23]	11	3	2	Nr
Dimakopaulos et al.[11]	38	34	32	130
Willems and Lim[39]	10	4	9	70–120
Skutek et al.[36]	13	11	12	Nr
Bosch et al.[5]	25	20	24	74
Mighell et al.[27]	80	66	66	128
Marotte et al.[26]	12	9	12	60–90
Boileau et al.[4]	78	62	Nr	114
Christoforakis et al.[6]	26	Nr	Nr	150
Kralinger et al.[22]	167	116	132	Nr
Demihran et al.[10]	32	24	31	Nr
Frich et al.[14]	42	29	Nr	113
Morici et al.[29]	25	18	Nr	Nr

Nr = not rewarded

Postoperative infections probably occur less than 1% of the time in shoulder arthroplasty. If the infection is acute and quickly recognized, irrigation, débridement, head exchange, and intravenous antibiotics may arrest the process. If more chronic in nature, infection will necessitate resection of the components and placement of antibiotic-impregnated cement spacer. Based on the bone stock, soft tissue qualities, and risk factors remaining after resolution of the infection, the reconstructive options can be weighed.

Interoperative vascular injury and postoperative venous thrombosis are both very rare with shoulder arthroplasty. The patient's hand and distal pulses should be checked before he or she leaves the operating room, and all bleeding should be identified and controlled. The fracture itself may put the anterior humeral circumflex artery at risk, and the surgeon should confirm that it is not damaged at surgery. With the raw fracture surfaces and hyperemic injury response, postoperative hematoma is not entirely uncommon. If suction drainage is not sufficient to keep the wound decompressed, one should try to aspirate the hematoma and apply a compressive dressing. If aspiration is not successful and tension remains across the surgical wound, we return to the operating theater, evacuate the hematoma, and identify the source of bleeding.

Ectopic bone formation is usually an incidental radiographic finding with no effect on the arthroplasty. It is more common in cases of fracture–dislocation. In some instances the new bone may bridge the glenohumeral joint, resulting in impingement and lost motion. In this unusual case, surgical resection may be indicated to restore a functional range of motion. Traditionally such surgery has been done after radioisotope imaging reveals less activity. However, one may take into consideration early intervention, as the patient will only get stiffer with time.

Clinical success/failure has been correlated with restoration of humeral length, proper version, secured tuberosity fixation, younger age, and early postoperative rehabilitation. Except for patient age, these are factors that are to some degree under the control of the surgeon and patient. Stiffness is one of the most common complications. Contributing factors are initial injury severity, prolonged immobilization, noncompliance with rehabilitation, and nonanatomic prosthetic reconstruction. A stiff shoulder is a painful shoulder and will result in an unsatisfied patient. To avoid any loss of motion due to soft tissue imbalance, the surgeon should as best as possible restore the patient's native geometry. To allow the necessary early rehabilitation, it is paramount to achieve excellent tuberosity fixation. Barring

any surgical limitations, a comprehensive structured rehabilitation program is usually successful for regaining function. If therapy is not working, the patient should be evaluated for tuberosity and humeral component positioning.

Loss of tuberosity fixation is probably the most devastating event to the functional success of the surgery.[22] This can ordinarily be avoided with secure fixation technique and anatomic reduction. If the subscapularis and lesser tuberosity displace in the early postoperative period, it is of utmost importance to make an early diagnosis and a timely repair. Late repairs do not have a great success rate, and we therefore emphasize the need to maximize the chances for success at the first surgical intervention. Tuberosity malreduction will also greatly affect the functional results.[3] Overreduction will put undue tension on the rotator cuff and lead to stiffness, tissue attenuation, cuff tears, and superior migration of the prosthesis. Underreduction will lead to impingement of the greater tuberosity beneath the acromion and/or of the lesser tuberosity beneath the coracoid/conjoined tendon complex.

Malposition of the humeral component is another issue that can be avoided in the operating room by the attentive surgeon. The stem must place the top of the head 5 to 10 mm above the greater tuberosity and in 20 to 40 degrees of retroversion. The head size must restore proper anatomy and offset. This combination will avoid impingement from a high riding tuberosity or loss of motion from the excess soft tissue tension created by a proud or oversized humeral head.

Instability is a problem that may present in two manners. First, if there is failure of the tuberosities and/or rotator cuff, superior migration of the prosthesis may result. With failure of the coracoacromial arch, anterior–superior subluxation or dislocation of the humeral head may result during attempted elevation of the arm. Second, anterior, posterior, or inferior dislocation can result from unrecognized capsular injury or with inadequate and unbalanced tissue tension.

Late Treatment

In some instances a patient is too sick due to massive injury or medical comorbidities to undergo surgery in the acute phase of his or her proximal humerus fracture. In other situations a patient may appear as a result of late referral or late presentation to the health care system. If the patient is elderly or otherwise infirm, then it is best to begin rehabilitation and give him or her a trial of conservative management. Many of these patients will achieve a satisfactory level of function and pain relief. If the patient is younger and more active, we will attempt a prosthetic reconstruction several months postinjury so long as the patient has a clear understanding of the less reliable results.[2532]

There is a subset of these patients that, treated nonoperatively, will continue to have pain with motion and will be severely affected in many aspects of their life. Due to the long duration since the original injury, we frequently are faced with bony malunion, rotator cuff loss, general muscle atrophy in the shoulder girdle, and marked soft tissue contractures. Classically we could offer these patients a choice of an arthrodesis or a resection arthroplasty.[9] Recently, we have addressed this difficult subset of patients with the Delta (reverse) shoulder prosthesis. The criteria of a functioning deltoid and adequate bone stock are essential when employing this prosthesis. This component affords substantial improvement in function and pain relief compared with resection arthroplasty or arthrodesis (Fig. 23A–D).

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30 Malunions, Nonunions, and Other Complications of Proximal Humerus Fractures

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SUMMARY 939

Fractures of the proximal humerus are relatively common, accounting for approximately 5% of all fractures,[62] and their prevalence can be expected to increase as life expectancy and associated osteoporosis increase. Fortunately, most proximal humerus fractures can be treated nonoperatively with expected good outcomes; initial surgical intervention is required in only 20% of acute fractures.[70] Unfortunately, a small but not insignificant subset of those patients whose initial treatment was either conservative or surgical fails treatment and proceeds to develop either complications from the original injury or complications associated with the management of the injury. The spectrum of such complications of proximal humerus fractures, including neurovascular injuries, avascular necrosis of the humeral head, hardware complications, malunion, and nonunion, can prove extremely problematic and challenging to treat. These patients often have debilitating pain, limitation of range of motion, and loss of function of the shoulder leading to significant disability. Treatment of these complications of fractures of the proximal humerus can prove extremely challenging due to a variety of factors: osteopenic bone, extensive scarring, previous instrumentation, avascular bone, and neurologic injury. This chapter will provide an overview of the

cause, evaluation, and classification of these challenging problems and will include a critical discussion of the treatment options with the goal of providing a simplified treatment algorithm.

COMPLICATIONS OF INJURY

Neurologic Injury

With neurologic injury there are common patterns associated with certain fracture patterns. The incidence of neurologic injury is reported to be between 21% and 50%.[12,24,84] There have been numerous reports that outline the risk of temporary and permanent nerve or vascular injury with fractures and fracture–dislocations of the proximal humerus. The literature is not always clear; some articles quote neurapraxias, without clarifying which ones have resolved spontaneously. Nerve injuries are more common than previously recognized. De Laat et al. evaluated nerve lesions in primary shoulder dislocations and surgical neck fracture–dislocations with a prospective clinical and electromyographic (EMG) study. Electrophysiologic evidence of nerve lesions was found in 45% of the 101 patients evaluated. The most commonly injured nerves were the axillary, suprascapular, radial, and musculocutaneous. These authors stressed the importance of early diagnosis and treatment to prevent lasting impairment in those that involve more than a transient neurapraxia. In three other electrophysiologic studies of surgical neck fractures associated with glenohumeral dislocations, the axillary and other nerves were injured 20% to 30% of the time. Age was a factor; in those older than 50 years, the brachial plexus injury rate was 50%.[12,28,82]

The patient- and fracture-related factors associated with neurologic injury include the elderly and hematoma formation. Most have partial or complete recovery; 5% to 8% have persistent motor loss. The three common shoulder injuries associated with nerve injuries include the anterior shoulder dislocation, the two-part greater tuberosity fracture with an anterior dislocation, and the surgical neck fracture.[15,17] The most common single injury causing a nerve injury in the shoulder is the isolated two-part greater tuberosity fracture with an anterior dislocation.[84] The most common isolated fracture location is the surgical neck. This includes the two-part, three-part, and four-part surgical neck fractures. With a posterior fracture–dislocation, the axillary neurapraxia rate is 31% to 37% of the cases.[12,60] Following a four-part fracture, the nerve injury rate is 6.1%. Of this percentage, only one-third fully recover,[95] whereas following the three- and four-part fractures with a subsequent failed open reduction and internal fixation (ORIF), the permanent nerve injury rate rises to 17.4%, or approximately 8.7 times that of the permanent neurapraxias with the injury before treatment.[77] Thus, an ORIF as an intermediate step before arthroplasty has additional and significant risk. Worse yet, permanent loss of the axillary nerve occurs following blunt trauma in 50% of the cases.[12] Even an isolated anterior dislocation has a 10% to 20% axillary injury rate.[15,64] In summary, the risk increases with advancing patient age, the surgical neck fracture site, an associated dislocation, hematoma formation, blunt trauma, and a failed ORIF.

The diagnosis of nerve injury is more difficult with acute fractures, owing to pain, swelling, and immobilization; later, the physical examination for each motor group is more straightforward.[17,74,104] The major muscle groups to evaluate include the three divisions of the deltoid, the spinati, the internal rotators, trapezius, serratus anterior, rhomboids, and the biceps and triceps. Within the internal rotators, the subscapularis can be isolated with the Gerber lift-off test.[36] The two external rotators, the infraspinatus and the teres minor, can be isolated by palpation and inspection and by the external rotation lag signs before electrical confirmation.[88] These tests are difficult to perform in most patients with the late sequelae of trauma and cannot be performed with an acute injury.

The sensory examination, which has often been used for the evaluation of the deltoid, unfortunately does not necessarily correlate with the motor integrity: EMG studies have documented the unreliability of sensory examination.[8,12] Specifically, the sensation can be intact in the axillary distribution, with disruption of the motor fibers. This has important implications in patient management as well as in the medical–legal exposure. If there is any question about the nerve integrity at any stage in the patient's course, EMG and nerve conduction studies may better delineate the patient's status preoperatively. This permits rational treatment plans (i.e., timing of nerve exploration, followed by decompression, primary or secondary repair or graft, or even in selected cases, muscle transfers). The shift in recent years has been to surgically explore those individuals who have complete loss without any recovery in any one motor group by 3 to 4 months.

The treatment of nerve injuries will depend on the nerves involved, the type of injury, and the age and health of the patient. Nerve recovery is better with younger patients and with less distance from the injury site to the motor end-plate when reinnervation can occur. The prognosis for recovery from a sharp injury is better than from a blunt injury. Certainly, an early exploration and repair are justified with a penetrating injury with complete motor loss in a given motor group. The debatable area is the closed fracture with or without an accompanying dislocation and an associated brachial plexus deficit. The monograph *Traumatic Brachial Plexus Injuries* by Alnot and Narakas is an excellent guide for the complex cases that arise.[1,2]

Vascular Injuries

Vascular injuries are rare with proximal humeral fractures and fracture–dislocations. They are often masked by collateral circulation early on. The incidence of vascular injury is thought to be 0.3% to 3.0% with long bone fractures.[16] Conversely, in those with documented vascular injuries, 10% to 40% had long bone fractures or dislocations; 20% to 42% originally had negative physical findings.

The patient factors associated with vascular injury include the location of the fracture or dislocation, the mechanism of injury, and the age of the patient. More information is available on the association of brachial plexus injuries with vascular injuries. The vascular injuries are less frequently recognized because of the very extensive periscapular collateral circulation.[16] Traffic accidents account for 80%. Two main patterns predominate.[3] In the first and most common pattern, the shoulder is driven down and the head and cervical spine are flexed to the opposite side. This causes a traction injury to the plexus and vascular structures at the base of the neck. In the second, there is an acute narrowing of the costoclavicular space with a crush of the neurovascular structures of the thoracic outlet from a blow to the anterior shoulder girdle. The outlet is narrowed with the arm at the side, or worse when it is abducted. Associated proximal humerus fractures need not even be significantly displaced for a vascular injury to occur at the surgical neck level.[40] Fracture patterns associated with vascular injury usually involve fractures at the surgical neck of the humerus.[61,63,98] The artery at this level forms a "tethered trifurcation" where the anterior and posterior humeral circumflex vessels and subscapular trunks arise from the axillary artery (Fig. 30-1). The vascular injuries are associated with direct trauma, with a dislocated humeral head or a displaced upper humeral shaft. In a three-part greater tuberosity fracture, the circumflex vessels can be caught in the surgical neck site. A thrombus can then develop in the axillary artery.[92] In this case report, intimal damage caused by traction injured the brachial artery and caused it to develop spasm. Thrombus removal alone was insufficient, but circulation was restorable with either a direct repair or by a vein graft. Intimal tears of the axillary artery are described in four other cases at the subscapular and circumflex branches.[43] In two, an anterior dislocation alone occurred. In two, there were three- or four-part fractures with anterior dislocations. Vessel repair restored circulation in all.

The diagnosis of vascular injury often requires a high level of suspicion when ischemia is noted. The physical findings may be subtle, for normal distal pulses may be intact in up to 27% with major arterial injuries about the shoulder. In addition to shoulder pain with the surgical neck fracture level, distal coolness and tingling develop. Palpation of the axillary artery may be precluded by pain, muscle spasm, and swelling. The brachial and radial pulses will be diminished or absent in over 70%. With time, pain on passive extension of the fingers occurs.[40] The six "Ps" to summarize vascular injury are pain, pallor, paralysis, paresthesias, poikilothermia, and pulselessness. In addition, there may be a large or expanding hematoma, pulsatile external bleeding, unexplained hypotension, or a bruit. Usually, a vascular injury accompanies a nerve injury at the same level. These signs and findings are indications for vascular investigation along with imaging. The single-injection trauma angiography is accurate in the operating room setting. If more time is available, then formal angiography can be obtained in the radiographic suite with retrograde femoral arteriogram. In addition, more sophisticated studies include digital subtraction angiography and Doppler arterial pulse volume recordings.

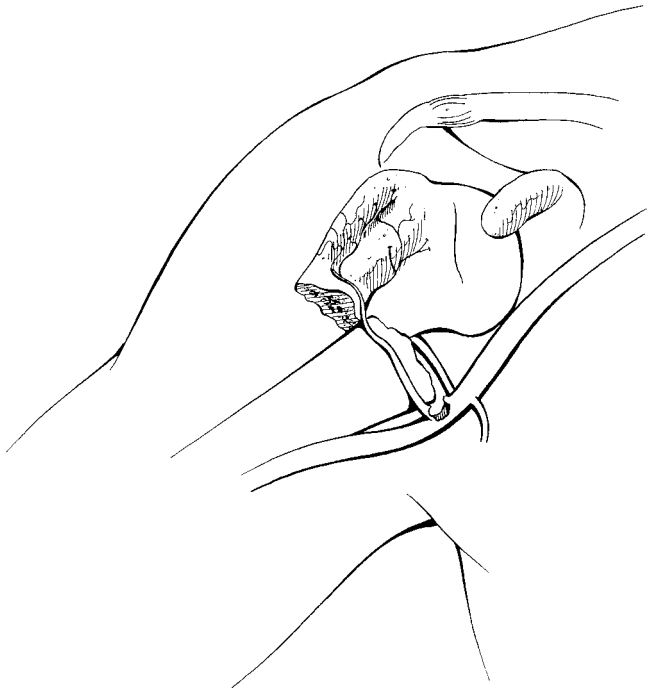

Figure 30-1 A "tethered trifurcation" forms where the anterior and posterior humeral circumflex vessels and subscapular trunks arise from the axillary artery.

Most authors agree that vascular injuries should be repaired. This is done immediately if there is acute ischemia. When performed on a delayed basis, it is to provide better nutrition for associated fracture and nerve healing.[3] At the time of emergency vascular repairs, the nerve repairs are best deferred until later when a good level of transection can be determined for the stretch injuries. The optimal time of nerve repair is 2 weeks to 3 months and may be combined with the elective vascular reconstructions. The one contraindication to a vascular repair is when the distal circulation is good through the collateral

circulation and there is a complete plexus avulsion. Here, no plexus reconstruction is possible.

At the time of acute vascular repairs, the tradition has been to rigidly fix all fractures with plates and screws. More recently, the need for rigid fixation has been debated as more fractures are pinned percutaneously or with limited pin, intramedullary rod, and tension band techniques. This avoids much of the periosteal stripping.[22,23,76] In support of this transition, Sturzenegger et al. noted five times the osteonecrosis rate in treating multifragmented proximal humerus fractures with AO buttress plates as with tension band wiring.[96]

Avascular Necrosis

Osteonecrosis, avascular necrosis, and ischemic necrosis are terms to describe the loss of circulation to the terminal articular surface. This is followed by collapse in most cases. Although rare, it can occur after two-part and minimally displaced fractures (Fig. 30-2). It occasionally occurs with a three-part displaced proximal humeral fracture, but most commonly occurs after four-part fractures or fracture–dislocations. Although the amount of osteoporosis and severity of the trauma may play a role, occlusion of the arcuate artery of Laing in the proximal portion of the biceps groove, which provides vascular supply to the anterior and superior two-thirds of the humeral head, is a major contributing factor.[37,57] This artery is the terminal branch of the ascending branch of the anterior circumflex humeral artery and enters the humeral head near the top of the groove of the long head of the biceps tendon. This point of rigid fixation is vulnerable to displacement of the shaft with fractures, and, especially, with fracture–dislocations. The more segments that are fractured and displaced at the time of a humeral shaft fracture, the greater the increase is in the incidence of injury to this artery. If the articular surface is no longer attached to any tuberosity and the medial soft tissue hinge at the neck of the humeral head is disrupted due to displacement of the shaft and the humeral head (classic four-part fracture), the incidence significantly increases. Neer reported two cases of osteonecrosis out of 33 ORIFs of three-part proximal humeral fractures.[71] Sturzenegger et al. noted six times the incidence of osteonecrosis with use of plates for the proximal humerus as compared with when they used wires and pins to provide what they termed "minimal fixation."[96] Although there are isolated reports without osteonecrosis following ORIF of four-part fractures, many of these have short follow-up. For example, Lee and Hansen reported a case of a vascularization of the humeral head after ORIF of a four-part fracture at 2 years, which then went on to collapse at 3.5 years.[59] In Sturzenegger et al.'s report of ORIF of three- and four-part fractures, three cases developed sympathetic dystrophy with osteonecrosis following hardware complications. Screw penetration into the joint can cause traumatic arthritis. Collapse of the joint makes the use of screws even more problematic.[65,73,96,105] The timing of replacement if this complication occurs is important. The longer one waits, the more likely it is that both sides of the joint will need to be replaced. Either use of the plate that compresses the artery or stripping to place the plate on the fracture sites at the lateral edge of the biceps groove contributes to injury of the artery with subsequent osteonecrosis. Nonunion, malunion, and eventual osteonecrosis are the frequent complications associated with ORIF of displaced four-part fractures.[10,75]

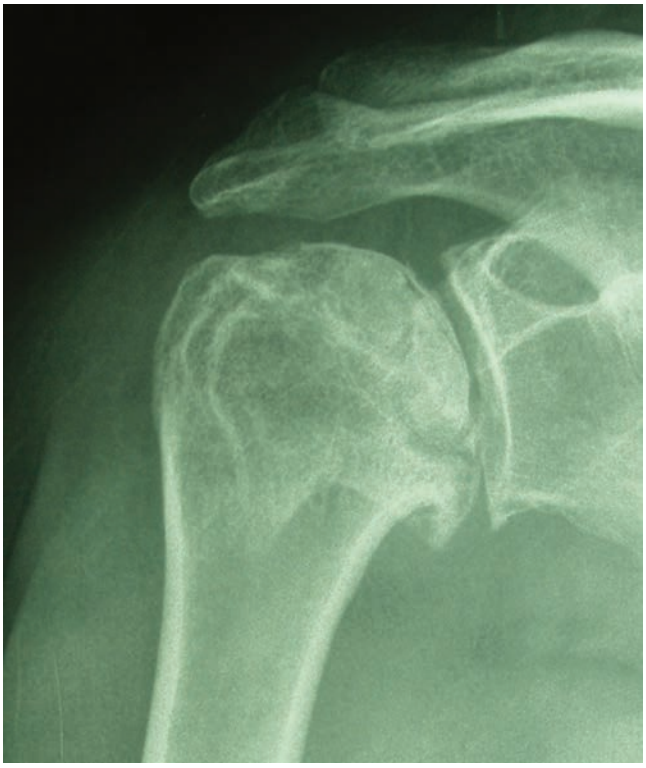

Figure 30-2 Avascular necrosis after a minimally displaced healed proximal humerus fracture.

Another attempt to decrease the incidence of osteonecrosis and extend the indications for ORIF of four-part fractures is a closed pinning and external fixation.[44,52–54] Although this has been a successful technique in Europe, Gerber et al. reported that when absolute anatomic reconstruction is not established, the result will be a failure.[35] There has been a significant increase in the number of nonunions, infections, and axillary nerve palsies following the closed pinning attempts. There is a steep learning curve. As with every new or resurrected technique, a set of complications also emerges.

In those patients in whom posttraumatic arthritis has developed from avascular necrosis, humeral head replacement may suffice. If the glenoid surface is significantly

damaged, and in particular if the concavity of the glenoid is altered with bony erosion at one edge, a total shoulder is preferred. Boileau et al.[13] reported good and predictable outcomes when treating patients with posttraumatic avascular necrosis of the humeral head with prosthetic arthroplasty. In fact, those patients in their series with avascular necrosis had significantly better outcomes than those with nonunions of the surgical neck or proximal humerus malunions. Prosthetic arthroplasty reliably provided pain reduction, restoration of range of motion, and improvement in function in another small series.[25]

MALUNIONS

For symptomatic patients with proximal humerus malunions, realistic nonsurgical options to reduce pain and improve function do not exist, and surgical reconstruction is often necessary. Surgical treatment of proximal humerus malunions proves especially challenging due to associated disruption of normal anatomic relationships, soft tissue scarring and contracture, rotator cuff pathology, postsurgical changes, neurologic impairment, and osteoporosis. This array of concomitant pathology renders the achievement of the operative goals of restoring premorbid functional status and relieving pain exceptionally challenging. The treatment of these complex problems requires thorough preoperative evaluation to determine the causative factors for the malunion and sound understanding and skilled application of the surgical techniques available to treat the entire spectrum of osseous and soft tissue pathology.

Etiology

Proximal humerus malunion results from either inadequate reduction of the displaced fragments or loss of fixation following closed reduction, closed reduction and percutaneous pinning, or ORIF. Although malunions sometimes occur following ORIF, they occur more commonly after nonoperative treatment. The higher incidence of malunion with nonoperative treatment may be secondary to the acceptance or lack of recognition of a significantly displaced fracture. Nonsurgical treatment of a displaced proximal humeral fracture may be a selected option in patients who are poor surgical candidates due to medical illness or have other severe injuries that preclude early surgical treatment of the proximal humeral fracture. Occasionally, a malunion occurs because the treating physician failed to appreciate the extent of displacement either due to lack of experience or inadequate or incomplete imaging studies. The malunion seen after internal fixation usually is secondary to inadequate fragment fixation obtained in the poor cancellous bone of the proximal humerus. This can result in postoperative loss of fracture reduction. It can also occur due to inadequate fragment reduction at the time of surgery.

Other contributing factors in proximal humerus malunions include inadequate immobilization, inadequate length of immobilization, or soft tissue interposition at the fracture site. Excessively aggressive rehabilitation can result in loss of fracture reduction or fixation.

Clinical Evaluation

Eliciting a careful history is essential in the evaluation of a patient with proximal humerus fracture malunion. The history should determine the mechanism of injury and subsequent treatment, with the goal of determining the cause of the malunion. Errors in diagnosis, such as a missed injury, may have occurred. Conditions that contribute to malunion include osteoporotic bone, premature or aggressive rehabilitation, high-energy multitrauma, and inadequate stability of operative fixation and inadequate reduction. Alcohol or steroid use may contribute to the development of humeral head avascular necrosis, which may provide additive joint incongruity to that attributable to the malunion.

The pain or disability associated with a proximal humerus fracture malunion varies considerably and must be addressed in terms of the patient's goals. This assessment is critical. A relatively painless malunion with adequate passive range of motion and strength may not require surgical management, especially in a sedentary patient with limited expectations for upper-extremity function. This is typically seen in the older patient with an isolated surgical neck fracture that heals with a varus malunion but the humeral head remains spherical and without posttraumatic arthritis.

Essential to the clinical evaluation of the patient with a malunion is a complete neurovascular examination of the involved upper extremity. From the initial trauma, there may have occurred associated permanent axillary nerve or brachial plexus injuries, especially if fracture fragments were initially displaced medial to the coracoid process. Axillary nerve injury is often associated with inferior subluxation of the proximal humerus (Fig. 30-3). Possible neurologic injury from previous surgery must also be ascertained. If prior nerve injury is suspected, electromyelographic examination can be helpful in determining the extent of injury and the prognosis for neurologic recovery.

The long head of the biceps tendon interposition can contribute to a malunion of the proximal humerus. Rotator cuff injury may have occurred during the initial trauma and must be addressed at the time of surgery. Iatrogenic injury from previous surgery can include detachment of the origin of the anterior deltoid or deltoid denervation,[39] transection of the long head of the biceps, or subscapularis tendon detachment.

Loss of motion is one of the primary management problems associated with proximal humeral malunion.

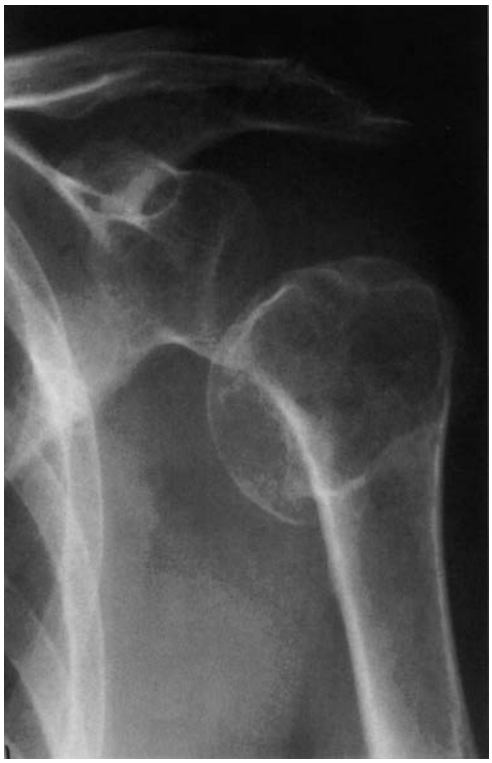

Figure 30-3 Anatomic neck malunion in a 64-year-old woman with an associated axillary nerve injury and deltoid paralysis. (Reproduced with permission from Warner JJP, Iannotti JP, Flatow EL, eds. *Complex and revision problems in shoulder surgery,* 2nd ed. Philadelphia: Lippincott Williams & Wilkins, 2005.)

Assessment of the degree of loss of passive versus active arcs of motion is necessary and should be considered in terms of the patient's current disability and treatment goals (Fig. 30-4). The progression or improvement of the patient's pain, weakness, and loss of motion must be considered as it relates to the rehabilitation program. If the passive motion is maintained, the rotator cuff is intact, and the surface of the joint is congruent, good function can be present without surgery (Fig. 30-5).

The surgeon should keep in mind the possibility of shoulder sepsis complicating any malunion resulting from prior surgery. If infection is suspected, appropriate hematologic studies are required and aspiration arthrogram is warranted.[25]

Radiographic Evaluation

Plain Radiographs

The most important aspect in the injury evaluation is to determine the position of the fracture fragments. Bone quality and the likelihood of healing must also be assessed. An adequate radiographic evaluation is required and includes an anteroposterior view in the plane of the scapula, axillary lateral, and transscapular lateral views[89] (Fig. 30-6). This series usually provides sufficient information to determine a treatment plan for most patients. Additional radiographic views such as the apical oblique view, which demonstrates posterolateral humeral head compression fractures, and the transscapular lateral view, which may be helpful in the evaluation of superiorly displaced greater tuberosity malunion, can be obtained.

Computed Tomography Scans

Computed tomography (CT) scans of proximal humerus malunions are needed when the plain radiographs are indeterminate. CT scans have been recommended to evaluate the degree of tuberosity displacement and for clearly imaging articular impression fractures, head-splitting fractures, and chronic fracture–dislocations (Fig. 30-7).[9,49,68] Morris et al. reported that CT identified axial malposition of the greater and lesser tuberosity that was not appreciated on plain radiographs in 10 and 18 out of 18 patients, respectively.[68] Additionally, several authors have recommended the utilization of three-dimensional CT reconstructions of the proximal humerus to better comprehend the position of malunited tuberosities (Fig. 30-8).[25,55]

Other Radiographic Modalities

Magnetic resonance imaging (MRI) can demonstrate associated soft tissue problems of the deltoid, rotator cuff, biceps tendon, and glenoid labrum, which can be helpful in the management of patients who may have concomitant osseous and soft tissue pathology (Fig. 30-9). Avascular necrosis can be detected earlier with MRI than with plain films, knowledge of which may affect treatment plans. It should be noted that patients with hardware from prior operative intervention may have signal artifact, which can diminish the quality and utility of the MRI or CT scans.

Classification of Proximal Humerus Malunions

There is presently no universally accepted classification system for proximal humerus malunions that provides a basis for comparing various subsets of malunions and different treatment outcomes. The absence of such a system lends to some difficulty in interpreting the relevant literature and comparing different treatments for specific subsets of malunions or posttraumatic sequelae across all reported cases. A few authors have recognized this shortcoming and have proposed classification systems useful in determining the treatment approach for proximal humerus malunions and evaluating outcomes.

Beredjiklian et al.[7] attempted to provide a method for systematic evaluation of both osseous and soft tissue

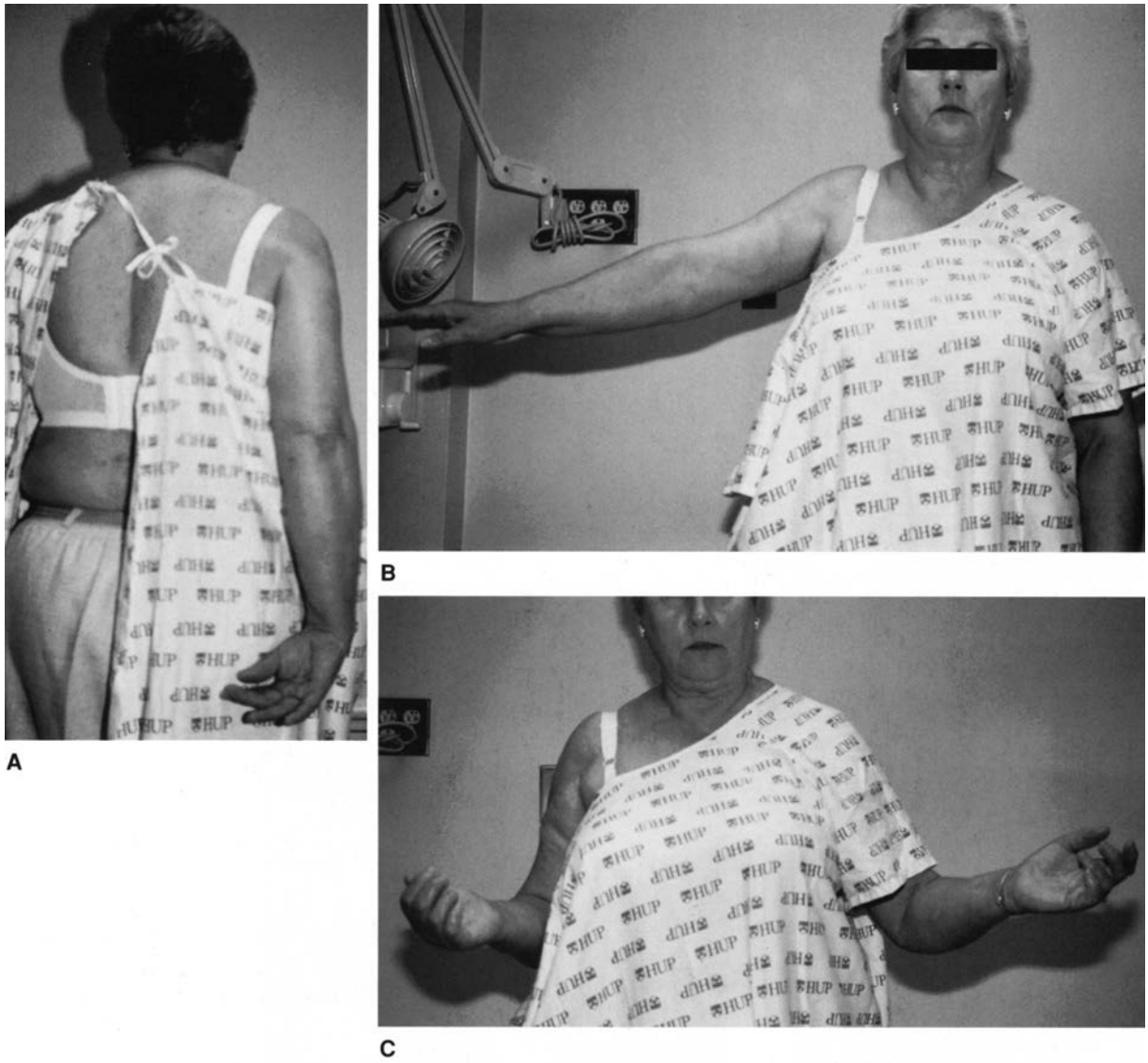

Figure 30-4 **(A)** The patient's functional disability from the marked loss of internal rotation, **(B)** abduction, and **(C)** external rotation, primarily resulting from capsular contracture, necessitating open capsular release. (Reproduced with permission from Warner JJP, Iannotti JP, Flatow EL, eds. *Complex and revision problems in shoulder surgery,* 2nd ed. Philadelphia: Lippincott Williams & Wilkins, 2005.)

abnormalities. Osseous abnormalities were categorized as malposition of the greater or lesser tuberosity of greater than 1 cm (type I malunion), incongruity or step-off of the articular surface of more than 5 mm (type II malunion), and malalignment of the articular segment by more than 45 degrees of rotation in any plane (type III malunion). Soft tissue abnormalities were classified as soft tissue contracture, rotator cuff tear, and subacromial impingement. Any given patient often has multiple osseous and soft tissue sequelae of their posttraumatic deformity, and this classification system is useful in enabling a holistic awareness of all involved pathology with the aim of treating each component.

In an attempt to simplify the surgical treatment and prognosis in treating the posttraumatic sequelae of the proximal humerus, Boileau et al.[13] proposed a general classification system for proximal humerus fracture sequelae that was not specific for but did include proximal humerus malunions. Intracapsular/impacted fracture sequelae (category 1) included both cephalic collapse and necrosis (type 1) and chronic dislocation or fracture–dislocation (type 2) in which a proximal humerus arthroplasty could be performed without a greater tuberosity osteotomy. Extracapsular/disimpacted fracture sequelae (category 2) included both surgical neck nonunions (type 3) and severe tuberosity malunions (type 4) in which the

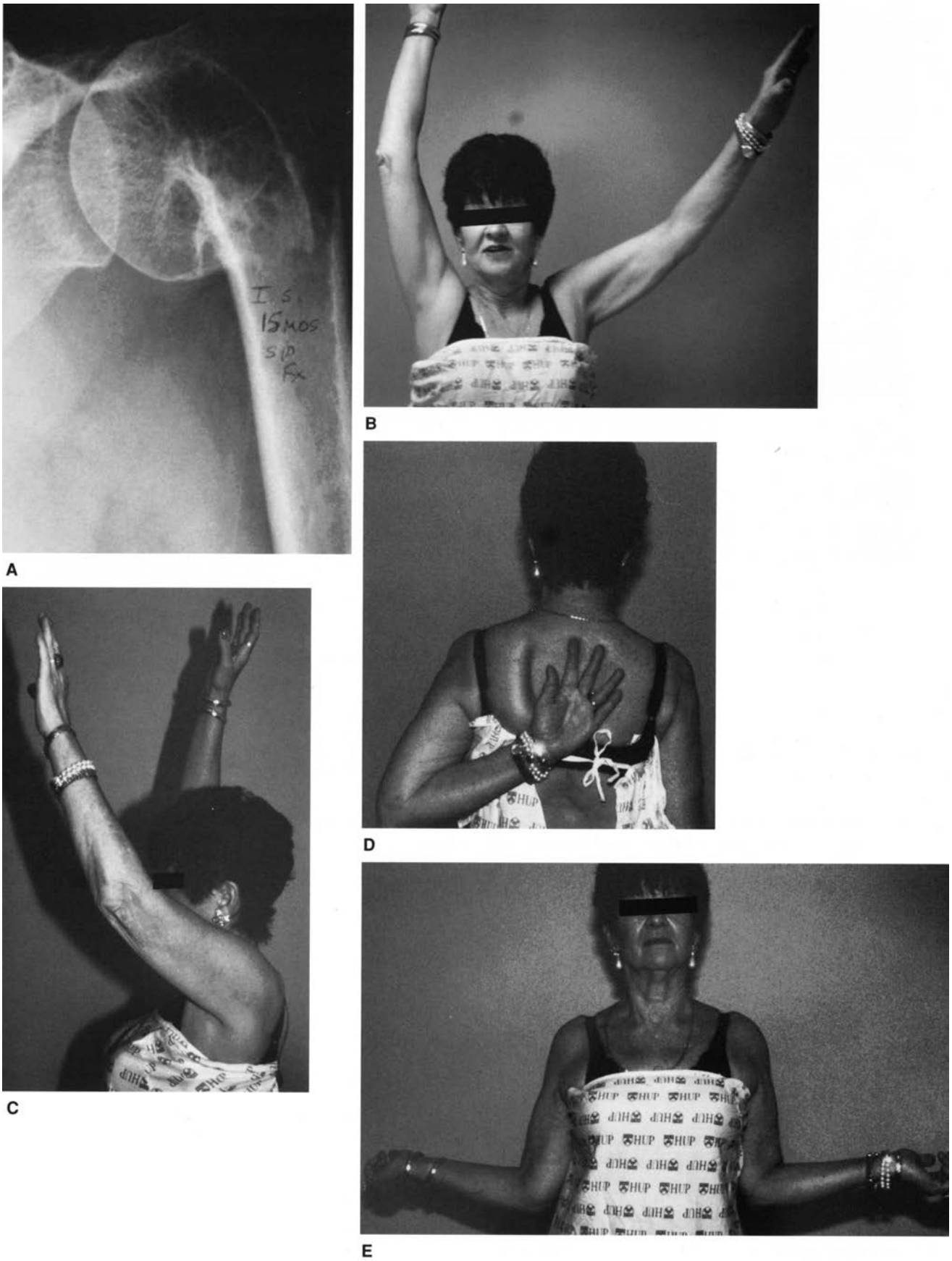

Figure 30-5 **(A)** A 60-year-old woman had a humeral neck malunion after prior surgery. The patient had excellent function in **(B)** abduction, **(C)** forward flexion, **(D)** internal rotation, and **(E)** external rotation. (Reproduced with permission from Warner JJP, Iannotti JP, Flatow EL, eds. *Complex and revision problems in shoulder surgery,* 2nd ed. Philadelphia: Lippincott Williams & Wilkins, 2005.)

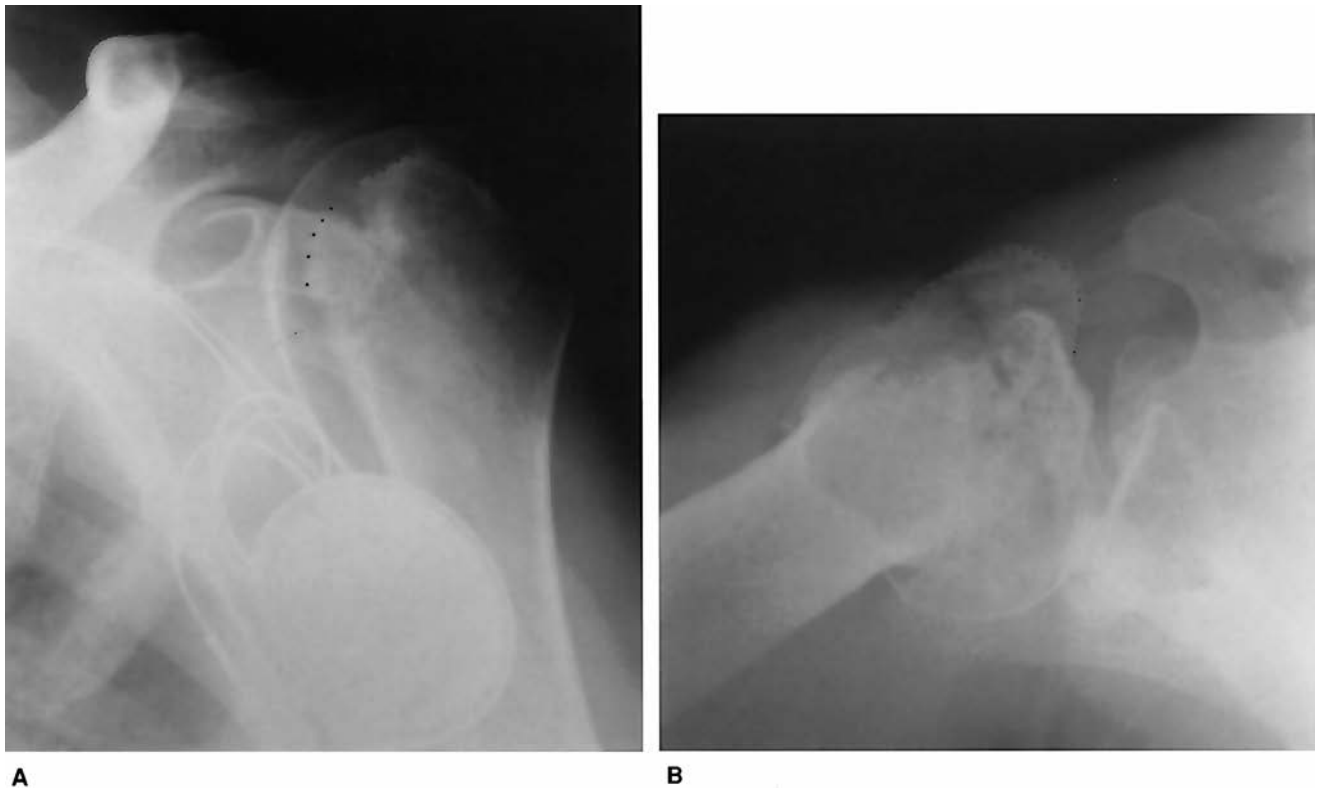

Figure 30-6 A 60-year-old retired but active man sustained a fall that produced pain. **(A)** An anteroposterior and inadequate scapular Y-view did not demonstrate the posterior humeral head dislocation. Malunion of a lesser tuberosity fracture with a posterior dislocation was initially unrecognized because of the lack of an axillary view at the time of the initial injury. **(B)** The axillary view obtained 6 months after injury clearly demonstrates posterior subluxation, malunited tuberosities, and posttraumatic arthritis. The dotted line represents the greater tuberosity. (Reproduced with permission from Warner JJP, Iannotti JP, Flatow EL, eds. *Complex and revision problems in shoulder surgery,* 2nd ed. Philadelphia: Lippincott Williams & Wilkins, 2005.)

proximal humerus could not be reconstructed without a greater tuberosity osteotomy. This classification system enabled the prediction of posttraumatic deformities that could be reconstructed with an arthroplasty with a predictably good outcome (category 1) versus those that would yield an unpredictable and likely poor result with arthroplasty (category 2) predicated on the need for greater tuberosity osteotomy and the associated possibility of postoperative displacement, malunion, or nonunion. This series excluded cases in which malunions could be treated with osteotomy and internal fixation without the use of shoulder arthroplasty in treating posttraumatic sequelae.

The most commonly utilized classification of proximal humerus malunions has been a simple modification of Neer's classification of acute proximal humerus fractures.[70] Several authors have utilized this classification and applied it to describe their series of malunions.[5,14,31,77,97] As this is a commonly understood classification system, it will be utilized to structure the following discussion of specific subtypes of proximal humerus malunions.

Specific Fracture Malunions

Two-Part Anatomic Neck Malunion

Isolated anatomic neck fractures are extremely rare, and malunions of this type have not been reported. Subsequent avascular necrosis of the articular surface is a common posttraumatic sequela of this type of fracture. A prosthetic hemiarthroplasty would be selected as the treatment if there is avascular necrosis with head collapse. Theoretically, an anatomic neck malunion could be treated by osteotomy and internal fixation, although risk of subsequent avascular necrosis would be of significant concern.

Two-Part Greater Tuberosity Malunion

Malunion of the greater tuberosity fracture is probably the most common proximal humeral malunion. Greater tuberosity fractures are often retracted superiorly and posteriorly by the deforming forces of the attached

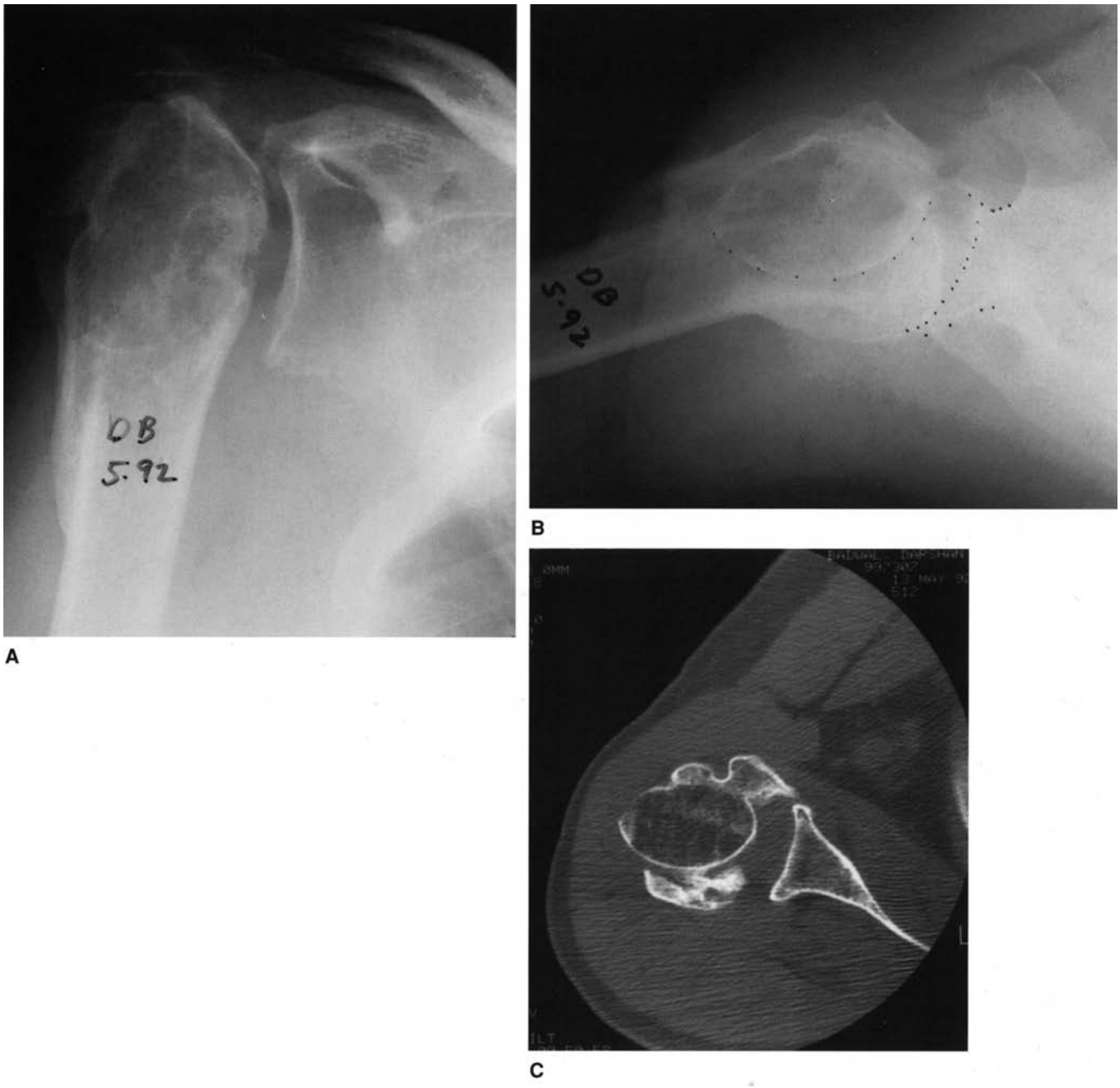

Figure 30-7 A 45-year-old businessman sustained a fall in India that was treated with nonoperative care. He had only mild pain with the activities of daily living and therefore did not consent to surgery. This patient had only 70 degrees of elevation and 0 degrees of external rotation and internal rotation to the buttock. **(A)** Malunion of a four-part fracture is poorly defined on the anteroposterior view. **(B)** The axillary view improves the recognition of the displaced humeral head fragment (widely spaced dotted line), and **(C)** the computed tomography scan best defines the united tuberosities. (Reproduced with permission from Warner JJP, Iannotti JP, Flatow EL, eds. *Complex and revision problems in shoulder surgery,* 2nd ed. Philadelphia: Lippincott Williams & Wilkins, 2005.)

supraspinatus, infraspinatus, and teres minor muscles. The articular surface is unaffected by this fracture type and maintains its appropriate relationship with the humeral shaft. Posterior displacement of the greater tuberosity can lead to a rigid, bony block to external rotation, which can be evident on physical examination. In extreme cases, anterior glenohumeral instability can result as the posteriorly displaced greater tuberosity impinges on the posterior glenoid in external rotation.[21] Similarly, superior displacement of the greater tuberosity can block abduction and forward elevation and lead to subacromial impingement as the malunited tuberosity encroaches on the subacromial

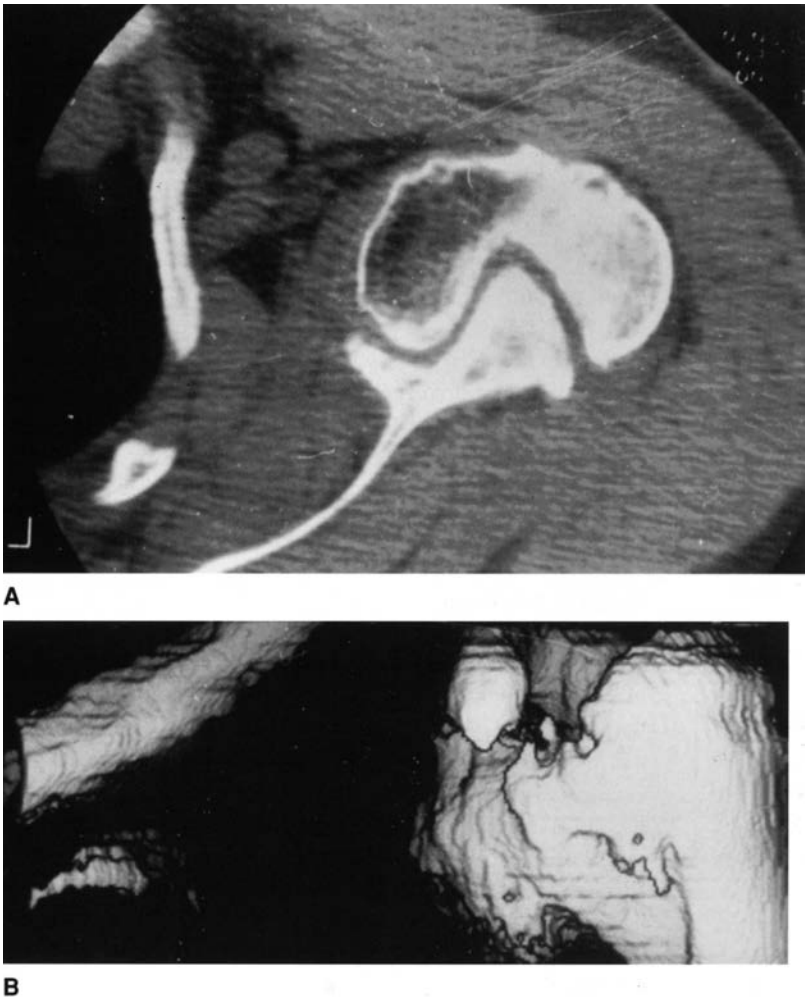

Figure 30-8 A 40-year-old laborer sustained a four-part fracture dislocation that was treated by attempted open reduction and internal fixation 15 years before referral to our center. **(A)** A computed tomography scan and **(B)** three-dimensional reconstruction of this malunited four-part fracture dislocation demonstrate the humeral head in a subcoracoid location and the humeral shaft articulating with the glenoid. This patient performed heavy labor for 15 years with what was essentially a fused "glenohumeral" joint, but pain increased over the years, and he was eventually treated with excision of the malunited head fragment and fusion of the proximal humerus to the glenoid. Successful fusion allowed him to return to heavy labor. (Reproduced with permission from Warner JJP, Iannotti JP, Flatow EL, eds. *Complex and revision problems in shoulder surgery,* 2nd ed. Philadelphia: Lippincott Williams & Wilkins, 2005.)

space.[21,48] Tuberosity malposition can result in rotator cuff weakness secondary to the shortened musculotendinous length of the external rotators.[91] Additionally, severe subacromial impingement can lead to rotator cuff attrition or tears. Thus, it is critical to examine the integrity of the rotator cuff through both physical examination and intraoperative evaluation.

Greater tuberosity malunions that heal to the humeral shaft result in less deformity and soft tissue contracture. These malunions are easier to mobilize and reduce to their anatomic site because they move with the humerus when the arm moves and are displaced less. On the other hand, greater tuberosity malunions that heal by fibrous tissue to the posterior part of the humeral head or glenoid neck result in severe shortening of the capsule and attached rotator cuff and can rarely be brought to their anatomic site and still achieve passive internal rotation to the abdomen.

Satisfactory closed reduction of chronically displaced, isolated greater tuberosity fracture is impossible, and malunion of this fracture is best treated by prevention through primary surgical management of the acute fracture. Conversely, fractures associated with an anterior glenohumeral dislocation are often successfully reduced with reduction of the dislocation.[52] Healing of these fractures must be monitored closely when they are treated by closed means because of the tendency for later displacement.[67]

Radiographic Evaluation

Careful radiographic evaluation of the suspected greater tuberosity is required; three high-quality orthogonal views of the shoulder should be obtained.[90] Small fragments isolated to the superior facet of the greater tuberosity often displace superiorly and are more commonly diagnosed on anteroposterior radiographs. Larger fragments containing the superior and posterior portion of the rotator cuff often displace posteriorly behind the humeral head and are more difficult to appreciate on anteroposterior radiographs. The scapular lateral view may be particularly useful in delineating posterior and superior displacement of the greater tuberosity, and the axillary view is particularly useful in determining posterior displacement. Malunion of the greater tuberosity is a result of not obtaining the correct

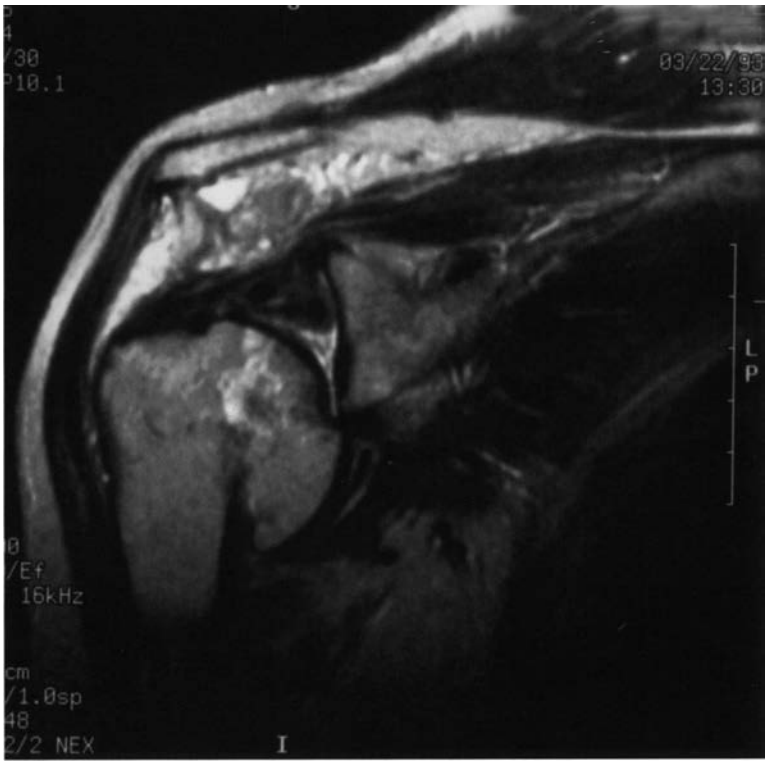

Figure 30-9 Magnetic resonance (MRI) scan demonstrates a posttraumatic arthritis defect in the humeral head, early avascular necrosis, and marked deltoid and supraspinatus atrophy. In this case, the MRI scan is helpful in diagnosing the avascular necrosis, determining the degree of atrophy, and detecting posttraumatic arthritis. (Reproduced with permission from Warner JJP, Iannotti JP, Flatow EL, eds. *Complex and revision problems in shoulder surgery,* 2nd ed. Philadelphia: Lippincott Williams & Wilkins, 2005.)

views, obtaining poor-quality views, or not obtaining follow-up x-ray films. In any case, malunion of the greater tuberosity is preventable.

A CT scan of the proximal humerus can be particularly useful in evaluating the greater tuberosity malunion. As stated previously, Morris and associates[68] evidenced that CT scanning enabled accurate evaluation of tuberosity displacement that was inaccurately identified with plain radiographs.

Surgical Indications

The Neer classification of proximal humerus fractures defines 1 cm as the criterion for significant displacement. In the case of greater tuberosity malunion, we think that for most patients the same criterion should be applied to correct the pathologic limitation in motion, rotator cuff weakness, and impingement. In an active patient with malunion of greater than 5 mm in the superior direction, symptoms of subacromial impingement can occur and can warrant surgical intervention.

Surgical Technique

In patients with acceptable passive arcs of shoulder motion, greater tuberosity malunions can be surgically managed with the same techniques used for the acute fracture.[30] For small fragments, up to 3 cm in size, with superior displacement, satisfactory exposure can be achieved with a superior incision within the Langer lines centered on the anterolateral corner of the acromion. The deltoid is detached from the anterior acromion and split a distance of 4 cm from the acromion. Subacromial scar tissue is excised when present. For larger fragments or malunions with posterior displacement, a wider and more extensile exposure is required. In these cases an extended deltopectoral approach is performed.

The greater tuberosity fragment is mobilized by sharp dissection of a fibrous union or by osteotomy of a bony union. The greater tuberosity osteotomy is frequently biplanar,[25] beginning at the anterior callous. Craig described the use of several drill holes prior to osteotomizing the tuberosity to mark the site and minimize fracture and fragmentation.[21] He emphasized that care must be taken during the osteotomy to protect the axillary nerve posteriorly as it emerges from the quadrilateral space. When discrimination between tuberosity and fracture callus is difficult, good-quality radiographs and a CT scan can assist in making this assessment. The donor defect from which the greater tuberosity has been displaced is adjacent to the bicipital groove. It is often filled with fibrous tissue or sclerotic bone and requires débridement and light decortication down to a bed of bleeding bone in preparation for the reduced greater tuberosity. Care should be taken to avoid removing all of the dense, cortical bone so as to preserve adequate bone stock for fixation. With severely posteriorly displaced fragments, excision of the posterior capsule is required to mobilize the greater tuberosity and its attached rotator cuff to allow the greater tuberosity to reach its site of reattachment. The rotator

interval is usually torn, and dissection of the scar in this area is necessary to mobilize the greater tuberosity fracture fragment. The rotator cuff must be brought to length, and any coexistent cuff tear should be repaired first. Temporary traction sutures placed in the rotator cuff insertion allow control of the greater tuberosity fragment to assist in lateral advancement during lysis of adhesions and contracture release. Prior to definitive repair of the rotator interval and fixation of the tuberosity fragment, it is important to inspect the anterior glenoid and labrum through the rotator interval to rule out associated pathology. This is especially true in cases in which the greater tuberosity fracture occurred in the setting of a traumatic anterior glenohumeral dislocation. If a Bankart lesion is identified, some authors advocate repair through the rotator interval prior the bony repair.[21,91] In some cases partial takedown of the subscapularis may be necessary to achieve exposure of the anterior–inferior glenoid. Bankart repair would be recommended in the young, active patient and is usually not required in the older and more sedentary patient. In the senior author's experience only lesions that include a significant bony glenoid component and demonstrate instability prior to surgery are repaired. The vast majority of patients with greater tuberosity fracture associated with an anterior dislocation either do not have a classic soft tissue Bankart lesion or the lesion is minor and does not require repair.

The greater tuberosity fragment can usually be anatomically reduced. In some cases, it cannot be anatomically reduced, in which case the greater tuberosity is returned anteriorly as far as possible and below the top of the humeral head. It is necessary that the patient's arm can be brought passively to the abdomen prior to wound closure. The surgeon may use the supraspinatus, infraspinatus, and teres minor facets of the greater tuberosity as anatomic landmarks for anatomic reduction.

Two to four heavy, nonabsorbable sutures (5-0 or #2 Fiberwire, Arthrex, Naples, FL) are placed vertically in a figure-eight configuration through the greater tuberosity and then through drill holes in the humeral shaft. Horizontal fixation can be achieved by placing intraosseous sutures to the lesser tuberosity area (Fig. 30-10).[79] Incorporation of the sutures in the rotator cuff tendon adjacent to the greater tuberosity fragment is important because the tendon substance is often stronger than that of the osteopenic bone of the tuberosities.

Alternatively, internal fixation of the greater tuberosity with screws and washers could be used in normal bone if the tuberosity fragment is large. In such cases where there is a large fragment of bone with good bone quality, one or two interfragmentary bone screws can be used, but should not be used without the additional use of the suture fixation as described above. Hardware loosening may require later removal.[101] Screw fixation should be avoided in osteopenic bone. Plate fixation is usually not necessary for the management of an isolated greater tuberosity malunion and may lead to subacromial impingement. Wire fixation does not seem to offer any distinct advantage over heavy nonabsorbable suture but does have the disadvantage of material failure and subsequent migration requiring removal.

After reconstruction, the subacromial space is examined with the surgeon's index finger. If it is tight or if a rotator cuff repair was performed, a subacromial decompression is performed to protect the bony and soft tissue repairs.

Passive and active assisted range of motion is begun on the day of surgery, but internal rotation behind the back is avoided for 6 weeks. Active motion is begun at 6 weeks, followed soon by resistance exercises. For cases with severe posterior displacement and difficult mobilization of the fragment, we utilize an abduction orthosis to hold the arm in 0 degrees of rotation, and internal rotation to the abdomen is avoided for the first 4 weeks.

In patients with greater tuberosity malunions and loss of passive arcs of glenohumeral motion, an arthroscopic capsular release without correction of the malunion is usually unsuccessful in achieving acceptable passive arcs of motion with a minimum of 120 degrees of forward elevation. In cases of malunion, there is a significant extracapsular scarring, and therefore we prefer the extended deltopectoral approach with open capsular release at the time that the malunion is corrected. Additionally, for large greater tuberosity fragments with metaphyseal-diaphyseal extension, the axillary nerve limits the extent of a deltoid split, further necessitating the use of a deltopectoral exposure. In cases with internal rotation contracture of greater than 20 degrees, subscapularis coronal plane Z-lengthening is performed. Cases with mild malunion and less severe internal rotation contracture require separation of the subscapularis from the underlying capsule and excision of the capsule and surrounding scar from the subscapularis tendon and muscle. In these cases where osteotomy is not required, an arthroscopic capsular release with excision of extracapsular scar is preferred.

Results

Morris and colleagues[68] reported three greater tuberosity malunions that underwent osteotomy and repositioning. Shoulder elevation and external rotation motion arcs were both improved by 60 degrees. If significant shoulder stiffness and weakness have not yet occurred, the surgeon might expect the good healing rate and functional results that Flatow and coworkers[30] found in their series of acute, displaced greater tuberosity fractures treated by the nonabsorbable suture method of internal fixation.

Eleven of the 39 cases reported by Beredjiklian et al.[7] involved an isolated malunion of the tuberosity (type I malunion). Eight of these patients were treated definitively with an osteotomy of the tuberosity and soft tissue reconstruction, and seven of these reconstructions corrected the bony deformity to within 5 mm of anatomic reduction. Pain relief was significant in all cases and rated as minimal

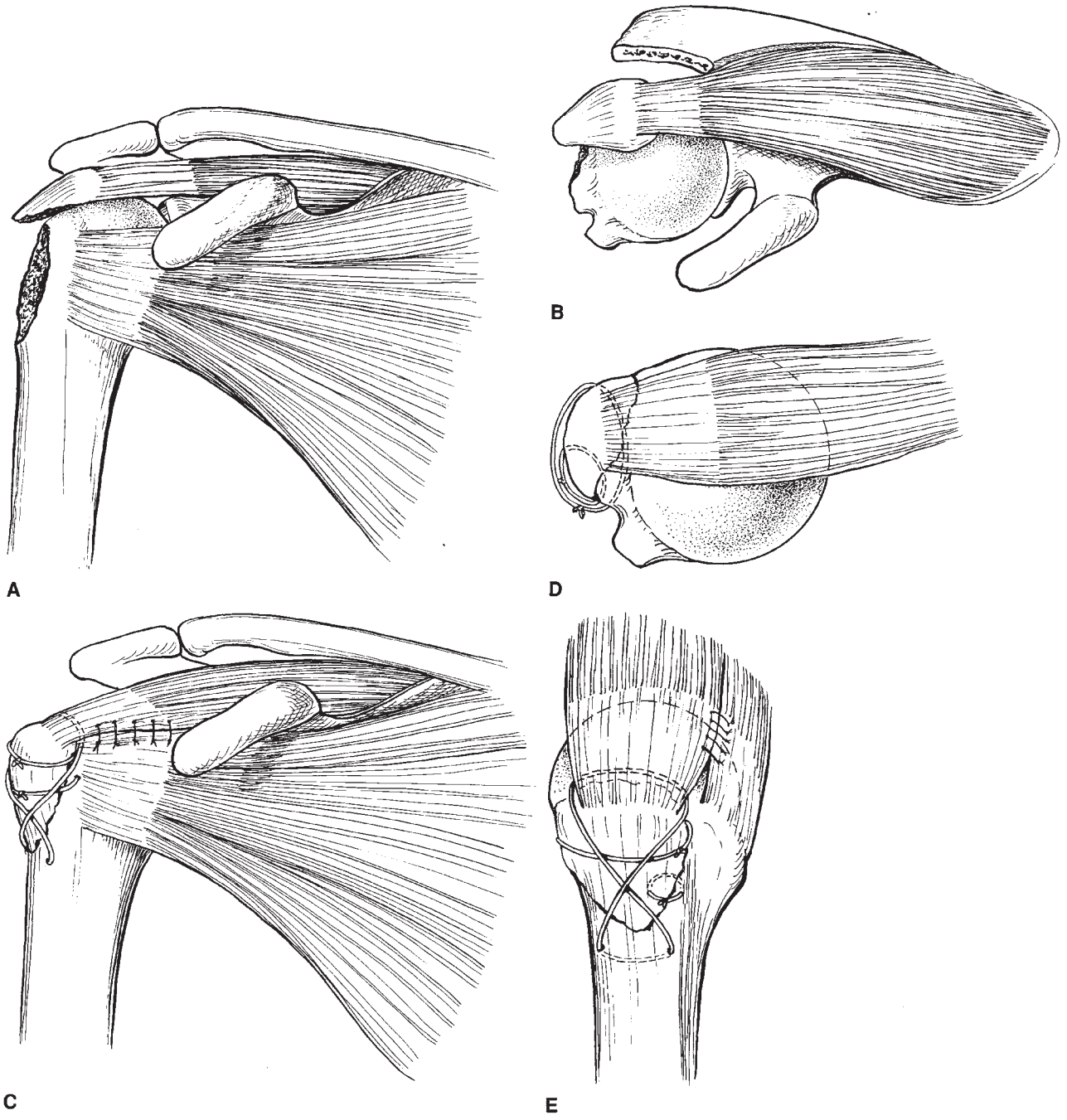

Figure 30-10 **(A,B)** Isolated greater tuberosity fracture with superoposterior displacement. **(C–E)** Reduction of the greater tuberosity and internal fixation using transosseous horizontal sutures and vertical figure-eight sutures. The horizontal sutures between the tuberosities are placed between the bone fragments, which helps to reduce the fragments, and in the rotator cuff tendon insertion sites, which provides the best tissue for maintaining the reduction during the postoperative evaluation. The vertical figure-eight suture also passes through the tendon insertion and then passes through a drill hole 2 cm distal to the metaphyseal fracture line. The vertical suture prevents superior displacement. The rotator interval between the subscapularis and supraspinatus is repaired, significantly improving stability. (Reproduced with permission from Warner JJP, Iannotti JP, Flatow EL, eds. *Complex and revision problems in shoulder surgery,* 2nd ed. Philadelphia: Lippincott Williams & Wilkins, 2005.)

or none in 88%. Functional capacity improved in 75% of patients, and, overall, six of eight patients had a satisfactory result. The best outcomes among their 39 cases were seen in the group who had isolated malposition of the greater or lesser tuberosity and had all osseous and soft tissue abnormalities corrected.

Two-Part Lesser Tuberosity Malunion

Two-part lesser tuberosity fractures are rare but often associated with posterior shoulder dislocations. If a malunion fragment is large, it may act as an obstacle to internal rotation and may also involve the articular surface of the humeral head. Associated subscapularis weakness may be seen with significant medial displacement due to altered musculotendinous length.

Radiographic Evaluation

Trauma series radiographs or CT scans can assist in determining size and displacement of the lesser tuberosity fragment. Morris and associates[68] reported that plain radiographs were inadequate in determining lesser tuberosity position when compared with surgical findings and that CT evaluation improved accuracy.

Surgical Technique

A deltopectoral approach can provide access to the lesser tuberosity fragment. Open reduction and internal fixation with anatomic reduction of associated articular involvement can be performed with heavy nonabsorbable sutures. The smaller-sized lesser tuberosity fragment can be excised[56] and the subscapularis tendon repaired directly to the proximal humerus with intraosseous sutures.[9] A chronically retracted fragment may require capsular release to mobilize the fragment. When necessary, we perform this from the inner aspect of the joint in the midcapsule. This approach avoids detachment of the subscapularis.

Surgical Neck Malunion

Isolated surgical neck malunions usually involve anterior angulation and varus deformity secondary to anterior displacement of the shaft by the pectoralis major[27] and abduction of the head by the rotator cuff.[70] This deformity, if severe, can cause limitation of forward flexion and abduction with associated impingement.[19] However, in many cases varus malunion does not result in significant loss in internal rotation and external rotation.

Radiographic Evaluation

Plain radiographic views of the proximal humerus in various degrees of rotation are required to accurately assess angulation. Comparison radiographs of the contralateral shoulder can assist in estimating the degree of deformity and planning the required osteotomy. Beredjiklian et al.[7] provided an excellent description of the method of determining the magnitude of this deformity radiographically.

Surgical Indications

If the surgical neck malunion reveals an increased anterior angulation greater than 45 to 55 degrees, forward elevation will be limited.[9,48] Varus deformity of similar magnitude can cause decreased abduction and forward elevation. These indications were supported by Beredjiklian et al.,[6,7] who classified malalignment of the articular segment of more than 45 degrees with the humeral shaft in any plane as a subtype of proximal humerus malunions requiring intervention.

Surgical Technique

If clinically indicated, osteotomy and internal plate fixation are performed at the malunion site. We prefer the use of a blade plate or a locking proximal humerus plate (Synthes, Paoli, NJ). Open capsular release is usually necessary to restore satisfactory passive arcs of motion and is performed before osteotomy. Bone from the osteotomy can be used for the graft or harvested from the iliac crest. For patients with intraarticular fracture or posttraumatic arthritis/avascular necrosis, hemiarthroplasty is our preferred method of surgical management.

Results

Solonen and Vastamaki[93] described a valgus wedge derotational osteotomy with T-formed AO plate fixation through a deltopectoral approach in seven young patients with preoperative varus angulation ranging from 40 to 60 degrees. Five of the seven patients achieved normal or near-normal results with significantly improved range of motion. Their two poor results were secondary to soft tissue pathology rather than failures in fixation or union of the osteotomy.

Three- and Four-Part Malunions

Three- and four-part malunions are complex problems that can result in severe deformities and significant complications. These posttraumatic sequelae usually require prosthetic replacement. In selected three-part injuries osteotomy of the fracture fragments followed by internal fixation may be attempted if there is no humeral head avascular necrosis or joint incongruity and the bone quality is good.

In three-part malunions either the greater or lesser tuberosity is displaced along with the surgical neck. If the greater tuberosity is left intact, the head will be externally rotated and abducted secondary to the intact superior and posterior portion of the rotator cuff. The shaft will be pulled anteromedially by the pectoralis, and the lesser tuberosity will retract medially. Fractures in which the greater tuberosity is displaced will result in the articular surface being internally rotated by the pull of the subscapularis. The greater tuberosity is posterosuperiorly displaced

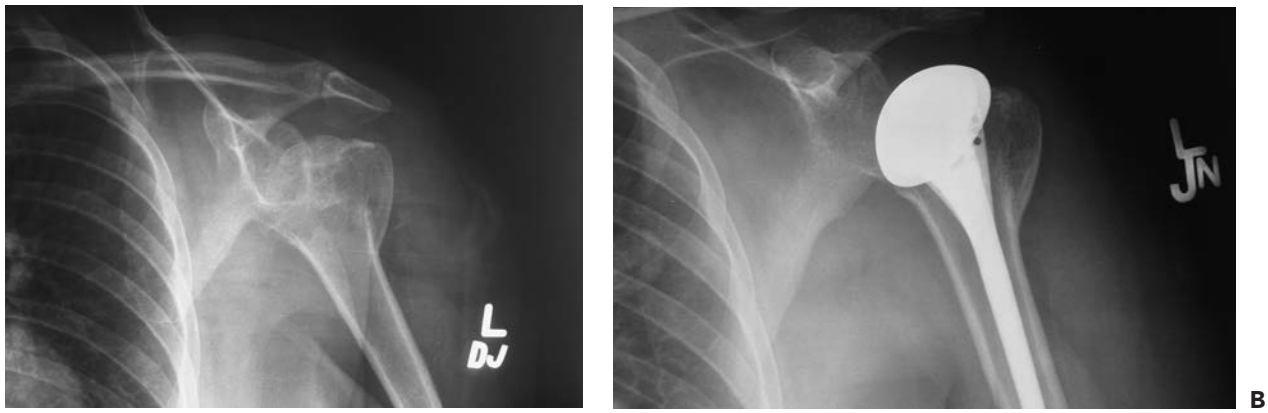

Figure 30-11 A 50-year-old female with posttraumatic arthritis and minimal displacement of the greater tuberosity treated with noncemented hemiarthroplasty without tuberosity osteotomy.

by the intact cuff, and the humeral shaft again is retracted anteromedially. Malunions of this magnitude often lead to severe loss of function, decreased range of motion, and disabling pain. An increased incidence of posttraumatic degenerative arthritis and avascular necrosis of the humeral head is also prevalent.[34]

Four-part malunions are challenging to treat as both tuberosities and the surgical neck are significantly displaced, leading to extreme humeral head distortion. These malunions are associated with significant soft tissue injuries and adhesions, joint incongruity, and an increased incidence of osteonecrosis.[34] Patients with four-part malunions typically have severe pain and disability secondary to restricted range of motion, avascular necrosis, soft tissue injuries, and contractures.

Radiographic Evaluation

Thorough preoperative radiographic studies are required in the treatment of three- and four-part malunions because tuberosity malunion is commonly present. We recommend CT scanning with three-dimensional reconstructions to determine tuberosity position, articular surface congruity, and bone integrity.

Surgical Technique

Most of these types of deformities occur in the elderly, and prosthetic replacement is usually required. The integrity of the glenoid articular surface determines whether humeral hemiarthroplasty or total shoulder arthroplasty is performed, and this determination can be made intraoperatively if preoperative radiographs are indeterminate (see Fig. 30-7).

An extensive deltopectoral approach with preservation of the deltoid origin is our preferred technique. Dissection of subacromial and intraarticular scarring are required to mobilize the rotator cuff and tuberosities, while avoiding neurovascular injury. Capsular contracture always exists, and an extensive capsular excision is required. Rotator cuff mobilization is enhanced by global excision of the capsule. The rotator cuff should be carefully inspected for tears, and these should be repaired.

Tuberosity position must be assessed before surgery and intraoperatively. One centimeter of displacement is a general guideline as the indication for surgical correction of a tuberosity fracture malunion. Greater tuberosity malunion with posterior displacement often blocks external rotation or forward elevation and results in postoperative anterior dislocation. Lesser tuberosity malunion may permit excessive posterior subluxation or block internal rotation because of coracoid impingement.

In some cases of tuberosity malposition, avoiding an osteotomy can be achieved with the use of a small stem that is shifted in the medullary canal to accommodate for tuberosity malposition, and an eccentric humeral head is also helpful to adapt the prosthesis to the malposition (Fig. 30-11). When these measures cannot restore a near-normal relationship between the tuberosities and the humeral head, a tuberosity osteotomy should be performed. Tuberosity reconstruction requires osteotomy with an oscillating saw or osteotome. Large degrees of tuberosity displacement require osteotomy before osteotomy of the humeral head segment. Small displacements can be assessed after osteotomy of the humeral head, and in some cases, tuberosity osteotomy is unnecessary (Fig. 30-12). The bicipital groove is a helpful landmark for tuberosity osteotomy. The osteotomy should produce a tuberosity fragment long enough to ensure contact with the bony humeral shaft on repositioning and large enough so that adequate rotator cuff is attached. The attached rotator cuff may need to be mobilized so as to achieve needed length as described previously.

Tuberosity fixation is achieved by using several heavy nonabsorbable sutures. Two sutures are passed through the middle portion of the tuberosity-to-tendon interface and the anterior flange of the prosthesis. Two more sutures are passed through both tuberosities, one each at the superior

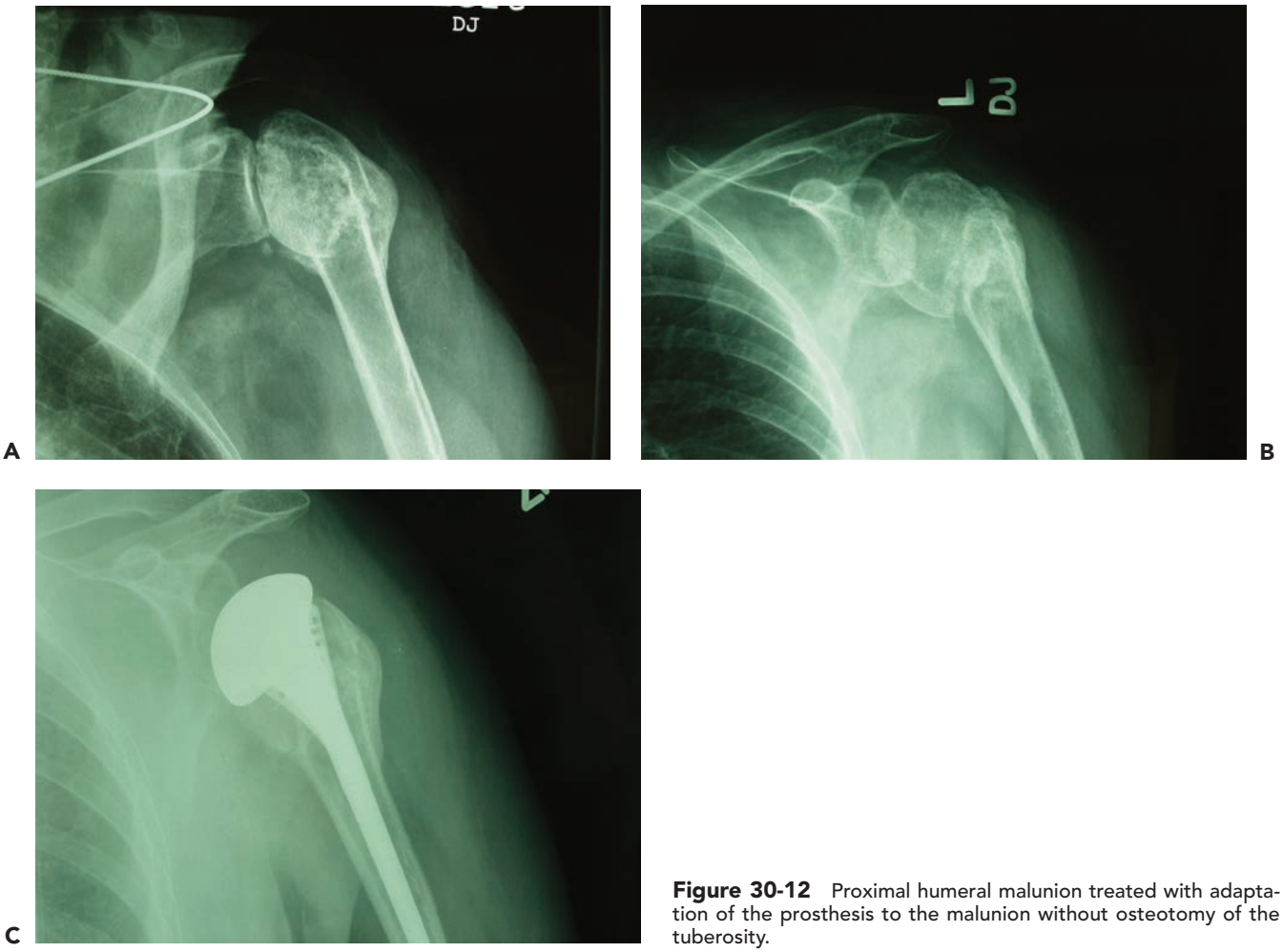

Figure 30-12 Proximal humeral malunion treated with adaptation of the prosthesis to the malunion without osteotomy of the tuberosity.

and inferior ends. Holes are drilled through the lateral and anterior aspect of the proximal humeral shaft. Two sutures are passed through these holes and, in vertical tension band figure-eight fashion, passed through the superior aspect of the tuberosities at the cuff insertion. These two sutures assist in bringing the tuberosities inferior to ensure contact of the tuberosities and the humeral shaft and placement below the top of the prosthetic humeral head. Local bone graft is usually available from the discarded head fragment and is used to increase tuberosity healing. Impingement can be avoided by making certain that the tuberosities are below the superior level of the humeral head.

A modular shoulder arthroplasty system, with a number of head component size options, allows better soft tissue tensioning. Intraoperative prosthetic changes can be made to improve glenohumeral stability and tuberosity position.[25] Humeral shaft bone quality and stability of the humeral component determine whether a cemented or noncemented humeral prosthesis is used.

Postoperative immobilization in a SCOI (Don Joy) brace with slight abduction and neutral rotation for 4 weeks helps protect the tuberosity repair and allows for balanced scarring in both the anterior and posterior aspects of the shoulder. The brace is removed several times a day for pendulum, active assisted, and passive range-of-motion exercises and activities of daily living.

Results

There is a relative paucity of literature regarding the surgical treatment of three- and four-part fracture malunions. The majority of series address the entire spectrum of posttraumatic sequelae of proximal humerus fractures in whole without focusing particular attention on malunions. This leads to a rather confusing body of literature in which comparisons between series are difficult.

In general, the results of prosthetic arthroplasty of three- and four-part malunions are inferior to those of prosthetic treatment of similar acute fractures.[102] Pain relief is usually achieved, but shoulder range of motion and strength are often limited. Patients with these malunions should be prepared for "limited-goals" outcomes following prosthetic replacement.

Tanner and Cofield[97] reported their experience in performing proximal humeral arthroplasty in 49 shoulders

with acute or chronic fractures of the proximal humerus. The chronic fracture group was composed of 27 shoulders with a mean time from initial injury to arthroplasty of 20 months. Sixteen of these 27 shoulders were documented as having malunion with incongruity of the articular surface. All of the patients in the acute fracture group and 25 of 28 shoulders in the chronic fracture group had satisfactory pain relief. Active abduction in the acute and chronic fracture group was 101 and 112 degrees, respectively. Complications were noted to be more frequent in the chronic fracture group and were related to surgical difficulty, extensive tissue scarring, and distortion of anatomy. Note was made that patients with the best function and motion had incurred less surgical insult to the cuff and tuberosity mechanism and did not require a tuberosity osteotomy. Cofield warned that late reconstruction of malunited proximal humerus fractures is "exceedingly complex" and the results in terms of motion and strength are limited; thus, these procedures should be classified as "salvage procedures."[18]

Frich and coworkers[31] found better pain relief in four-part fractures treated with arthroplasty acutely than those treated after a surgical delay, which in their series was of at least 4 months' duration. Of 11 patients in the chronic four-part fracture group treated with prosthetic replacement, only one had a good result, compared with three excellent results and six good results in the 15 patients treated with immediate replacement. There were no good or excellent results for the nine patients with chronic three-part fractures. There was a high incidence of instability for the group with delayed surgery attributed to increased difficulty in soft tissue tensioning, including rotator cuff reconstruction. The authors noted a remarkably limited range of motion in their series of chronic fractures and concluded that "treatment of chronic proximal humerus fractures to a large extent is a soft tissue problem and that the preoperative condition of the soft tissue around the shoulder determines the end result."

Dines et al.[25] reported on a series of 20 shoulder arthroplasties performed for chronic posttraumatic changes of the proximal humerus including malunion, nonunion, impression fractures, and osteonecrosis. Preoperative malunion of the tuberosities or humeral head was noted in 8 of 20 patients, and 12 patients required tuberosity osteotomy at the time of arthroplasty for malunited tuberosities. Fair, good, or excellent results were achieved in 90% of patients. Ninety and seventy-five percent of patients had pain relief at rest and with activity, respectively. Average postoperative motions were forward elevation of 111 degrees, external rotation of 30 degrees, and internal rotation to L2. However, patients requiring tuberosity osteotomy had resultant motion of 15 degrees less forward elevation and 9 degrees less external rotation than those not requiring an osteotomy. Additionally, patients having undergone a tuberosity osteotomy had less improvement in the Hospital for Special Surgery shoulder scoring system than those not requiring an osteotomy (73.6 vs. 82.3, respectively). Thus, avoidance of tuberosity osteotomy is advised whenever feasible. The authors recommended the use of a modular-designed prosthesis to facilitate soft tissue tensioning and tuberosity repair.

In a study of 39 consecutive patients with three- and four-part fractures treated with hemiarthroplasty, Bosch et al.[14] noted that the outcome of primary or secondary hemiarthroplasty was inversely proportional to the time between the injury and the prosthetic replacement. Their series included 17 patients with secondary humeral head replacements greater than 4 weeks postinjury. Limited description of the presence or types of malunions was provided. All patients had satisfactory pain relief, and the only factor found to negatively influence outcome was the length of time from injury to operation.

Seventeen of 23 shoulders with failed treatment of three- and four-part proximal humerus fractures subsequently treated with arthroplasty reviewed by Norris et al.[77] were classified as malunions. All of the shoulders initially treated nonoperatively and 54% of those initially treated surgically had malunion as a complication. Tuberosity osteotomy was required in 57% of cases, and the authors warned that tuberosity osteotomy is a "formidable procedure" with technical difficulty and potential for complications. Pain was satisfactorily relieved in 95% of patients, average forward elevation increased from 68 to 92 degrees, and average external rotation increased from 6 to 27 degrees postoperatively. They concluded that late glenohumeral arthroplasty, albeit technically difficult, is a satisfactory reconstructive procedure when primary treatment of complex proximal humerus fractures is unsuccessful. It was also noted by these authors that results are inferior to those for acute humeral head replacement.

Beredjiklian et al.[6,7] retrospectively reviewed their experience in treating 39 patients with proximal humerus malunions categorized by the presence of osseous and soft tissue abnormalities as previously described. Overall, the result was satisfactory for 69% and unsatisfactory for 31% of patients at an average of 44 months postoperatively. It is notable that 96% of patients with a satisfactory result had operative correction of all osseous and soft tissue abnormalities, whereas 66% of patients with an unsatisfactory result had incomplete operative correction of these abnormalities. Thus, they concluded that operative management of these patients is successful only if all osseous and soft tissue abnormalities are corrected at the time of surgery. Additionally 74% of patients with malunions and joint incongruity treated with prosthetic arthroplasty had a satisfactory result. The rate of complications in their study approached 30% and attests to the technical difficulty in treating proximal humerus malunions.

Sixteen of the 71 shoulders treated with arthroplasty for the posttraumatic sequelae of proximal humerus fractures in Boileau and colleagues'[13] series were characterized as

having severe malunions of the tuberosities. Overall, the results of arthroplasty for this heterogeneous group, including cephalic collapse, locked dislocations, surgical neck nonunions, or severe tuberosity malunions, were encouraging with a significant reduction in pain and improvement in anterior elevation (74 to 102 degrees) and external rotation (0 to 34 degrees). However, the isolated results for those patients requiring tuberosity osteotomy for prosthesis implantation (16 patients with severe malunions of the tuberosity and six patients with surgical neck nonunions) were less favorable. All of the patients in this group had either fair or poor results and did not regain active elevation above 90 degrees (mean active elevation of 82 degrees vs. mean of 123 degrees in patients not requiring osteotomy). The authors concluded that greater tuberosity osteotomy is the most likely reason for poor and unpredictable results after shoulder arthroplasty for the sequelae of proximal humerus fractures and recommended avoiding an osteotomy whenever possible, even if that entails accepting distorted proximal humerus anatomy. Complications occurred in 27% of the cases in this series.

Antuna et al.[5] evaluated the long-term outcome of 50 patients who underwent shoulder arthroplasty as treatment for proximal humeral malunions with a mean of a 9-year follow-up. Shoulder arthroplasty resulted in statistically significant pain relief with significant improvements in postoperative motion: Active elevation improved from 65 to 102 degrees and external rotation improved from 12 to 32 degrees on average. Despite these promising results, 50% of patients in the series had an unsatisfactory result as determined by the Neer result rating with a 20% overall complication rate. Of note, there was statistically significant less postoperative motion in those patients who had undergone previous operative treatment of their initial fracture or who required a tuberosity osteotomy at the time of arthroplasty. Ten of the 24 shoulders that required tuberosity osteotomy had complications including tuberosity nonunion (four), tuberosity malunion (three), and tuberosity resorption (three). These results further attest to the technical difficulty in treating this complex group of patients.

Mansat and colleagues[66] evaluated the efficacy of shoulder arthroplasty for the late sequelae of proximal humerus fractures. Eight of the 28 patients in their series had proximal humerus malunions. Three of these cases required osteotomy of the tuberosities during prosthetic replacement despite the use of a modular prosthesis, and all three of these patients had unsatisfactory results as determined by Constant criteria. The authors noted that patients with malunion or nonunion had the least favorable results when compared to those with simple posttraumatic arthritis or avascular necrosis.

In the case series reported to date, prosthetic reconstruction utilized a standard unconstrained humeral component. The use of the reverse total shoulder should be considered in patients when a tuberosity osteotomy is required, particularly in the older patient. The results of the reverse shoulder have not been published for treatment of severe malunions requiring tuberosity osteotomy, but one would expect better functional results than those reported with unconstrained hemiarthroplasty or total shoulder replacement (Fig. 30-13).

There is also a role for the use of a resurfacing humeral hemiarthroplasty that does not require the use of an intramedullary stem for fixation of the prosthetic. With humeral surface replacement the volume of remaining humeral head after reaming should allow for at least 70% contact with the undersurface of the prosthetic and the bone quality should be sufficient to prevent bone indentation with firm digital pressure after reaming. In addition, the tuberosity should not be malunited or require osteotomy. If these surgical criteria are satisfied, then use of a resurfacing humeral component may allow for greater adaptation of the prosthetic to the proximal anatomy, thereby avoiding osteotomy of the tuberosity, surgical neck, or humeral shaft (Fig. 30-14).

Glenohumeral Surgical Fusion

Glenohumeral fusion of a proximal humerus malunion is indicated only in selected cases. A fusion may be required in the individual with concomitant rotator cuff and deltoid dysfunction.[79] A shoulder infection that cannot be eradicated with hardware removal, irrigation with débridement, and intravenous antibiotics may ultimately require fusion.[38]

Conclusion

Fortunately, proximal humerus malunions are relatively uncommon injuries. However, when present they are painful and severely debilitating with little response to conservative, nonsurgical measures. As evidenced by the significant rate of complications experienced even in the clinical series of accomplished, respected shoulder specialists, these posttraumatic sequelae are among the most challenging and technically difficult disorders that a shoulder surgeon will face. Certainly, the best treatment for any proximal humerus malunion is prevention by adequate treatment of acute fractures. Several authors, citing the increase in complications and worse functional outcomes following late arthroplasty for proximal humerus malunions, warn against making the mistake of assuming that failed primary treatment can simply be corrected with late prosthetic arthroplasty.[14,31,77,79,91,97] Thus, they advocate prompt and proper acute treatment of proximal humerus fractures to avoid the later challenge of treating a malunion.

When faced with a proximal humerus malunion, it is critical to obtain an accurate history and perform a thorough physical examination. Workup should include adequate plain radiographs supplemented with CT scans, three-dimensional CT reconstructions, and MRI scans when uncertainty remains following review of plain films. The ultimate goal of every preoperative evaluation should be

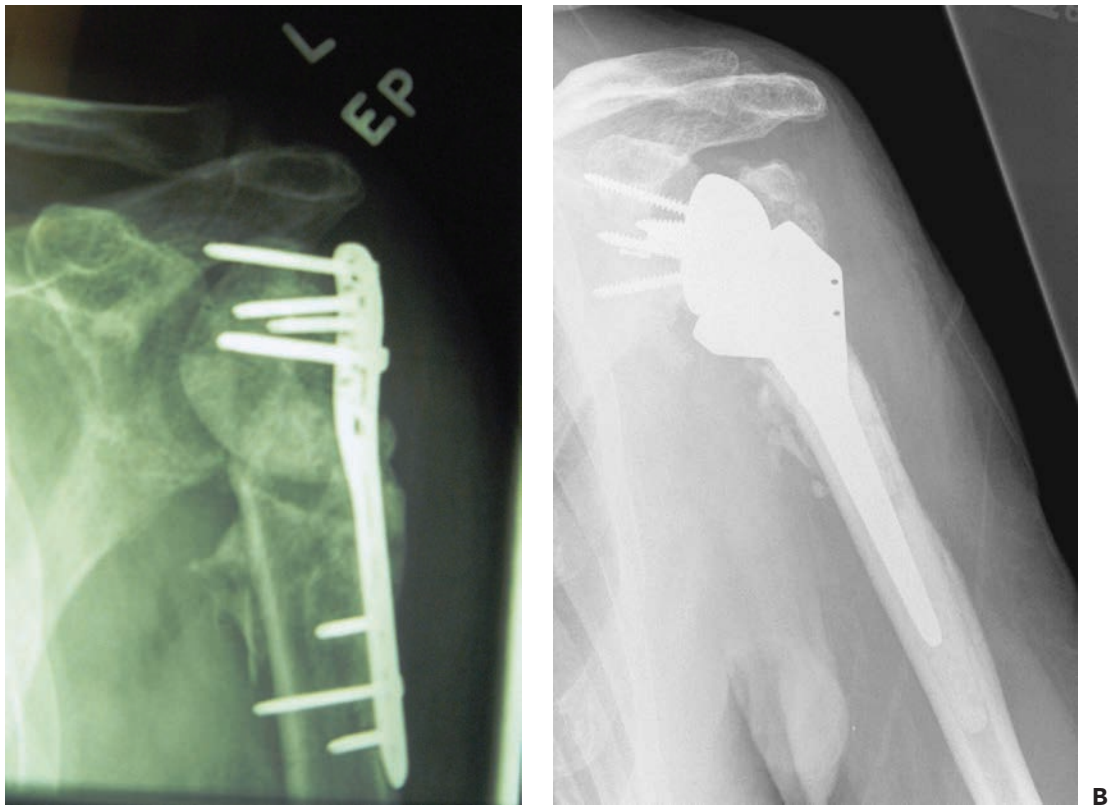

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Figure 30-13 The results of the reverse shoulder have not been published for treatment of severe malunions requiring tuberosity osteotomy, but one would expect better functional results than those reported with unconstrained hemiarthroplasty or total shoulder replacement.

maximal understanding of all involved pathology including osseous and soft tissue deformities so that an appropriate surgical plan can be formulated with the aim of treating all components of the posttraumatic pathology. The results for surgical correction of isolated tuberosity malunions are promising and favorable outcomes can be expected. Although results of prosthetic arthroplasty for complex proximal humerus malunions are inferior to acute arthroplasty, results in the literature evidence good potential for pain relief and some improvement in function with the likelihood for limited-goals function. It is essential to counsel patients preoperatively so as to equate postoperative expectations with that which has been evidenced to be attainable.

When it comes to the surgical treatment of proximal humerus malunions, we recommend an approach that addresses all osseous and soft tissue abnormalities present in each posttraumatic shoulder. The treatment algorithm recommended by Beredjiklian and colleagues[6,7] provides a comprehensive, systematic method of evaluating and treating these complex cases in such a manner (Fig. 30-15). It is obvious from the literature that the necessity for a greater tuberosity osteotomy automatically increases the likelihood for complications and decreases the likelihood for favorable, predictable outcomes. Therefore, we recommend avoidance of tuberosity osteotomy during arthroplasty by utilizing a modular prosthetic system and approximating the tuberosity–to–humeral head relationship. Thus, tuberosity osteotomy is avoided unless absolutely necessary for prosthetic implantation. When all osseous and soft tissue abnormalities are addressed surgically with appropriate technique and carefully monitored postoperative rehabilitation, the outcome is satisfactory in most cases. The use of the reverse total shoulder prosthesis in the older patient with severe malunion requiring prosthetic replacement and tuberosity osteotomy may provide better function than that seen with unconstrained arthroplasty.

NONUNIONS

Fractures of the proximal humerus are nondisplaced in the vast majority of cases and typically heal uneventfully with closed treatment. Unfortunately, a small percentage of these fractures, treated nonoperatively or operatively, develop nonunions. Patients that develop this complication are often debilitated with persistent pain and limited function. The technical challenges of previous scarring, poor bone stock, and prior hardware can make these cases extremely challenging, but successful treatment of proximal humerus nonunions resulting in relief of pain and restoration of function can be extremely rewarding.

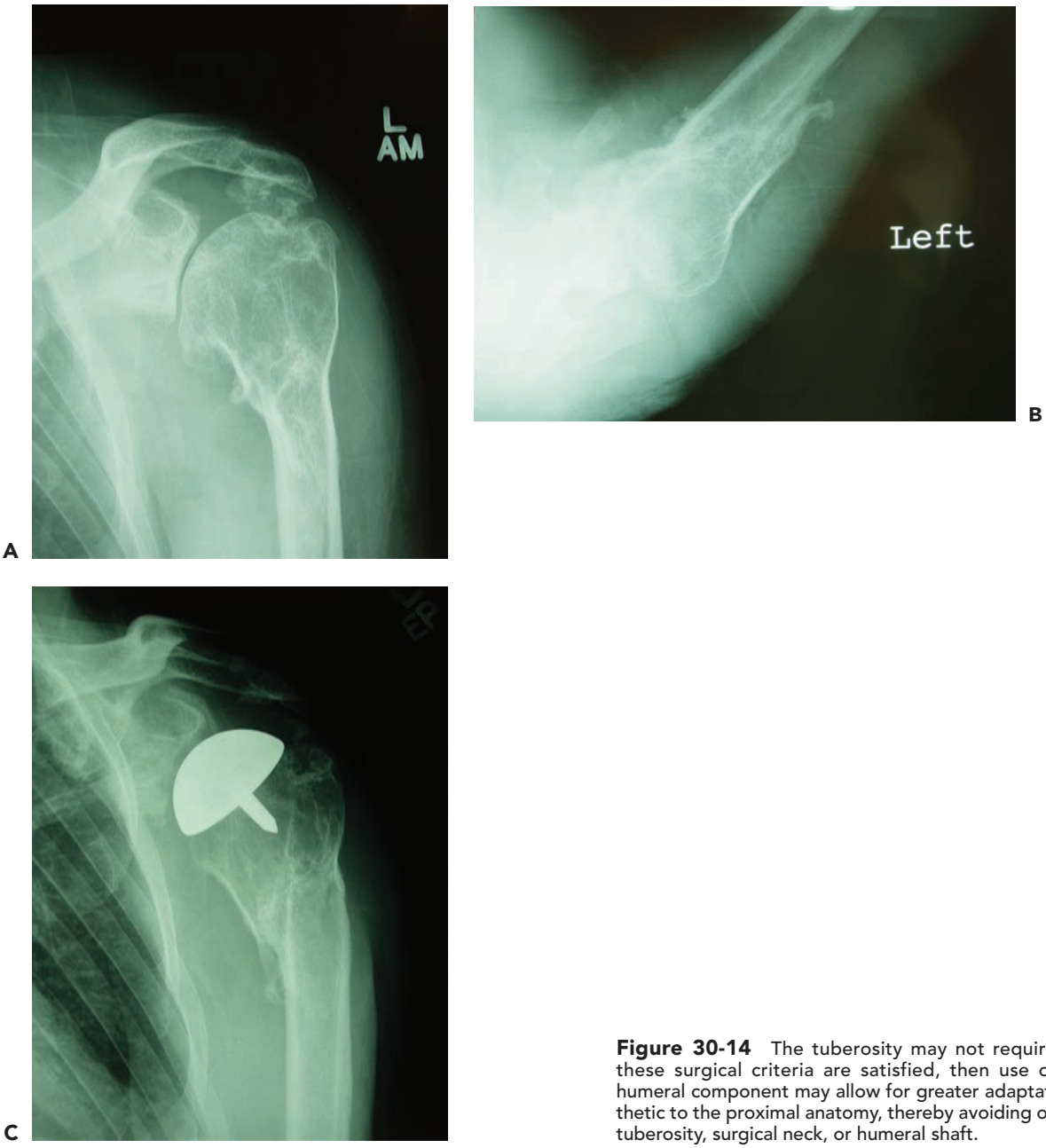


Figure 30-14 The tuberosity may not require osteotomy. If these surgical criteria are satisfied, then use of a resurfacing humeral component may allow for greater adaptation of the prosthetic to the proximal anatomy, thereby avoiding osteotomy of the tuberosity, surgical neck, or humeral shaft.

Etiology

The paucity of literature reporting the incidence of proximal humerus fracture nonunions suggests that they are a rare phenomenon. However, nonunions may be commonly seen after displaced two-part surgical neck fractures or after cases in which inadequate open reduction and internal fixation was used as the primary treatment.[19,103] In fact, up to 23% of surgical neck fractures may go on to nonunion.[72] Other fracture patterns that have a higher incidence of nonunion include those with displaced tuberosities, which are treated closed, and four-part fractures, which are treated either closed or open. Factors contributing to the development of nonunions may be attributed to either patient factors, fracture site factors, sequelae of inappropriate primary fracture treatment, or any combination thereof.

Patient factors that predispose to proximal humerus fracture nonunion include osteoporosis, metabolic bone disease, diabetes, drug or alcohol addiction, nutritional deficiency, smoking, and general noncompliance.[87,100] Each of these comorbidities can significantly impact the healing potential at the initial fracture site, the rate of complications following operative treatment, and the ability of the patient to comply with a postoperative rehabilitation protocol. These factors may have been overlooked or underestimated in the initial decision-making process contributing

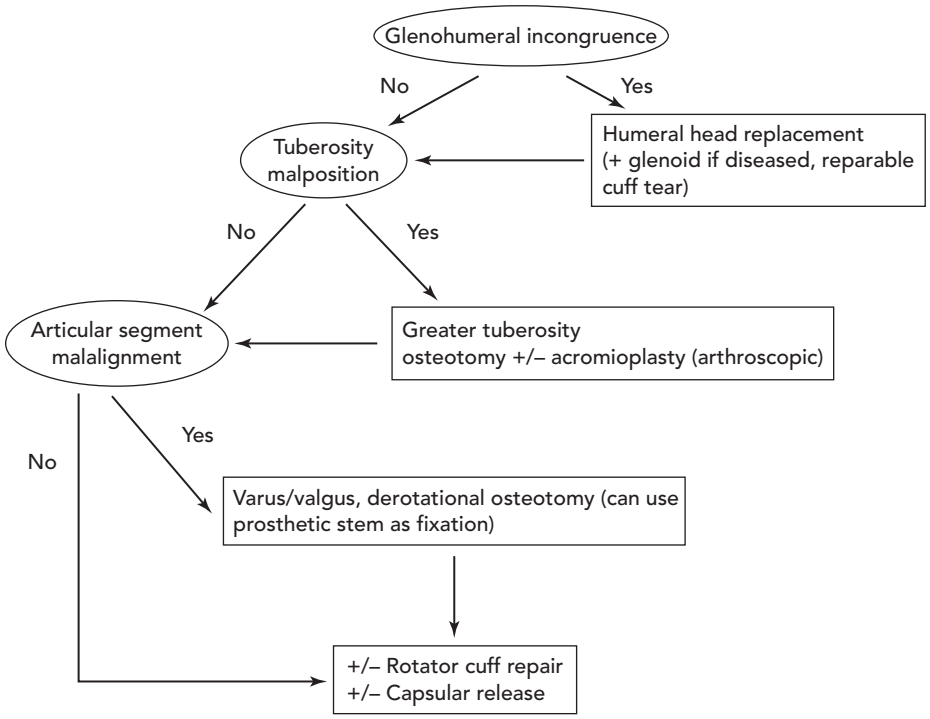


Figure 30-15 Algorithm for the categorization and operative treatment of malunions of the proximal aspect of the humerus. (Modified form Beredjiklian P, Iannotti JP, Norris TR, et al. Operative treatment of malunion of a fracture of the proximal aspect of the humerus. *J Bone Joint Surg Am* 1998;80:1484.)

to the development of the complication. Healy et al.[41] documented in their series of 25 patients with proximal humerus nonunions that the most common associated factor was the presence of one or more significant medical comorbidities in 64% of cases. The results of treatment in this group were unsatisfactory overall, with only four patients having good results. Thus, it is imperative to evaluate patients for these and other coexistent medical comorbidities prior to surgical intervention for treatment of an established nonunion. Glenohumeral joint stiffness, prior to the fracture, has also been identified as a predisposing patient factor to the development of surgical neck nonunions. One small series identified four patients that developed pseudarthroses secondary to preexisting glenohumeral stiffness, either from rheumatoid arthritis or prior glenohumeral fusion.[86] In this situation, humeral motion occurs in the fracture site rather than the glenohumeral joint, leading to nonunion.

Fracture site factors also contribute to the development of nonunions. Soft tissue interposition between the proximal and distal fracture fragments can prevent adequate contact of opposing bone surfaces, impeding callous formation and fracture reduction. The long head of the biceps tendon, the deltoid muscle, or the rotator cuff tendons are potential impediments.[32,69] Additionally, the dense cortical bone in the distal fragment of a surgical neck fracture and the generalized decreased quality of metaphyseal bone in the proximal humerus seen in this elderly population predispose to poor bone healing. Surgical neck fractures are also subject to the deforming forces of surrounding musculotendinous units: The pectoralis major pulls the proximal shaft anteromedially and the rotator cuff tendons rotate and abduct the proximal head or tuberosity fragments. These forces can prevent adequate reduction of the fracture segments and contribute to nonunion. Lastly, synovial fluid from the adjacent joint can dilute the fracture hematoma and inhibit callous formation.

Finally, proximal humerus nonunion can result as the iatrogenic sequela of inadequate primary fracture treatment. Closed treatment can be problematic as the weight of the arm causes distraction across the fracture site, which can be accentuated with the use of a hanging arm cast. Nine of 16 patients with established nonunions in Neer's[71,72] series followed closed treatment, and distraction was implicated as the major cause in each of these cases. Attention to detail is necessary when treating these fractures with immobilization; the arm must be immobilized across the front of the body to neutralize the medial pull of the pectoralis, and the elbow must be kept in front of the midline in the coronal plane to prevent apex anterior angulation and loss of reduction.[32] Inadequate operative treatment can also contribute to nonunion, especially if severe osteopenia is overlooked at the time of initial intervention. Epidemiologic studies have linked proximal humerus fractures with fractures of the distal radius and proximal femur, especially in elderly women.[29,45] This factor should not be overlooked as plate and screw fixation can be problematic in osteopenic bone, leading to inadequate fixation, hardware loosening, fracture site motion, and nonunion. Lastly, premature institution of range-of-motion exercises following closed or open treatment contributes to nonunion. It is imperative that the fracture parts have consolidated and move as a unit prior to the initiation of shoulder motion.

Clinical Evaluation

The degree of pain and functional loss must be carefully assessed with a thorough history and physical examination. Patients typically have little ability to perform basic tasks or activities of daily living due to a flail arm—loss of deltoid and rotator cuff ability to move the arm in space. Pain is variable, but most patients complain of at least moderate rest pain and significant pain with daily activities.[83] Severe pain with more strenuous activities is not uncommon. In rare cases, pain may be minimal and function adequate for a low-demand, sedentary lifestyle justifying benign neglect; this is especially true in the face of significant associated comorbidities and confounding factors.

In cases in which prior operative treatment was attempted, complete review of all records and operative reports is essential, and the potential of an infected nonunion following operative intervention must always be included in one's thought process. This concern warrants further evaluation with a complete blood count, acute phase reactants, aspiration, and culture to screen for sepsis.[25]

Physical examination is often striking in that most shoulder motion occurs at the fracture site rather than the glenohumeral joint.[11,72,78] This may be misinterpreted as falsely adequate passive range of motion, but the patient typically has minimal to no active range of motion of the shoulder (0 to 45 degrees of forward elevation). Severe atrophy of the deltoid, spinati, and periscapular musculature is common, and motor function can be difficult to assess because of the pain, weakness, and mechanical disability of the nonunion. Regardless, it is essential to examine for at least modest firing of the deltoid and rotator cuff musculature to rule out preexisting neurologic dysfunction from the initial injury and/or deltoid dehiscence from any previous surgical intervention. A thorough neurologic examination including motor and sensory function should be performed. If uncertainty exists, an EMG should be obtained to differentiate pain and weakness from neurologic injury. The skin and soft tissues should be examined for integrity as bony prominences from the underlying nonunion can result in overlying skin compromise. Drainage from prior incisions is an ominous herald of an infected nonunion.

Evaluation of the patient's medical comorbidities is also essential. Confounding variables such as severe osteopenia, nutritional deficiency, metabolic bone disease, and medical illnesses with the potential to adversely affect healing must be recognized and investigated. Blood chemistry can provide screening information to assess metabolic bone state, liver function, kidney function, and glucose and fat metabolism. If a medical condition is discovered that affects bone healing, referral to an internist or endocrinologist is warranted.

Radiographic Evaluation

An adequate radiographic evaluation is required and includes an anteroposterior view in the plane of the scapula, an axillary lateral view, and a transscapular lateral view.[89] Radiographs should be critically evaluated for presence of a nonunion, avascular necrosis of the humeral head, posttraumatic arthritis of the glenohumeral joint, and displacement of the tuberosities. A surgical neck nonunion, either hypertrophic or atrophic, is typically obvious; however, additional radiographic views such as the apical oblique view, which demonstrates posterolateral humeral head compression fractures, and the transscapular lateral view, which may be helpful in the evaluation of superiorly displaced greater tuberosity malunion or nonunion, can be obtained. If suspicion of intraarticular extension exists as occurs with a head-splitting fracture, a CT scan can be valuable in delineating this pattern. In addition, a CT scan will clearly define the position of the tuberosities.[68] A significant amount of bone loss at the nonunion site may contribute to shortening of the humerus with associated shortening and dysfunction of the deltoid. A scanogram or plain radiograph of the opposite humerus can be obtained to determine the loss of height that needs to be addressed in the operative treatment.[80]

Classification of Proximal Humerus Nonunions

The classification of proximal humerus nonunions follows Neer's system of classifying acute proximal humerus fractures.[70] This classification is based on a four-part system involving the humeral head, the lesser and greater tuberosities, and the shaft as the four parts. Acutely, when any one of the four major segments are separated more than 1 cm or angulated greater than 45 degrees with respect to another part, the fracture is considered displaced. Unimpacted two-part fractures of the surgical neck are generally associated with a higher incidence of nonunion than other fracture types.[72] Nevertheless, tuberosity nonunions and complex three- and four-part nonunions do occur.

Specific Fracture Nonunions

Tuberosity Nonunion

Plain radiographs in three orthogonal views typically define a tuberosity nonunion. However, CT scanning can be helpful in accurately defining the location and size of united tuberosities.[68] Nonunions of the tuberosities impose significant functional deficits on shoulder function, making the presence of any united tuberosity an indication for surgery. There is typically no role for nonoperative treatment of these nonunions unless the patient has medical contraindications to surgery. Greater tuberosity nonunions function similar to a massive rotator cuff tear, rendering a large portion of the rotator cuff incompetent and leading to significant weakness in external rotation and forward elevation.[21] Associated impingement by a superiorly retracted greater tuberosity can further contribute to pain, rotator cuff

pathology, and disability.[21] Likewise, lesser tuberosity nonunions render incompetence of the subscapularis, a major anterior stabilizer of the shoulder. These patients have weakness in internal rotation as evidenced by the Gerber lift-off test[36] and can have anterior instability. Additionally, a medially displaced lesser tuberosity can cause painful coracoid impingement with limited internal rotation.

Surgical Technique

The surgical technique for repair of a greater tuberosity nonunion is similar to that described in the preceding section on malunions. Repair can be performed through a superolateral incision centered on the anterolateral corner of the acromion for small fragments. As described, the united fragment is débrided of fibrous tissue and scar down to viable bone. Extensive circumferential rotator cuff release is often required to enable reduction of the fragment back into its anatomic bed lateral to the bicipital groove.[21] Concomitant rotator cuff tears should be addressed prior to reduction of the tuberosity fragment. After débridement and preparation of the donor bed, the fragment is secured with two to four heavy, nonabsorbable sutures (#5 Ticron or #2 Fiberwire, Arthrex, Naples, FL) as described. Alternatively, internal fixation of the greater tuberosity with screws and washers could be used in normal bone if the tuberosity fragment is large. When using screw fixation, suture fixation should be used as a supplemental means of fixation to avoid loss of fixation due to fracture of the tuberosity proximal to the screw fixation.

A deltopectoral approach can provide access to the lesser tuberosity fragment. Open reduction and internal fixation with anatomic reduction of associated articular involvement can be performed with heavy nonabsorbable sutures. The smaller-sized lesser tuberosity fragment can be excised[56] and the subscapularis tendon repaired directly to the proximal humerus with intraosseous sutures.[9] A chronically retracted fragment may require capsular release to mobilize the fragment. When necessary, we perform this from the inner aspect of the joint in the midcapsule. This approach avoids detachment of the subscapularis. Our postoperative rehabilitation protocol does not differ from that utilized after the reconstruction of tuberosity malunions.

Surgical Neck Nonunion

Nonunions of the proximal humerus most commonly occur at the surgical neck.[18,19,41,72,80] Patients typically present with pain, disability, and palpable crepitus at the nonunion site. Minimal active motion is present as the rotator cuff attached to the proximal segment has no distal attachment to the upper extremity. The nonunion is usually quite obvious on plain radiographs, although occasionally a CT scan is necessary in differentiating a fibrous union from a nonunion. Osteopenia of the proximal segment and bone loss at the nonunion site are commonly identifiable on plain films. Occasionally, osteopenia and humeral head cavitation, accelerated by the accumulation of joint fluid at the nonunion site, preclude proximal fixation.[72]

The only role for nonoperative treatment is in the medically debilitated patient that would not tolerate surgical intervention or in the patient with medical or social comorbidities that would preclude postoperative compliance. Thus, surgical indications are debilitating pain and loss of function in a cooperative, motivated, and reliable patient. Surgical intervention in these cases can be extremely challenging secondary to extensive capsular contracture, scarring from prior surgical intervention, metadiaphyseal bone loss, distorted anatomy, and osteopenia of the humeral head.[32] Therefore, it is imperative that the patient is counseled preoperatively to enable realistic understanding of these challenges and their relevance to postoperative complications, rehabilitation, and expectations.

Numerous techniques have been described for treating surgical neck nonunions.[4,11,13,20,25,26,32,33,41,47,58,69,77,80,85,87,94] These techniques can be classified into four main groups: intramedullary nails, intramedullary nails with tension bands, open reduction and plate fixation, and arthroplasty. Choice of surgical technique is largely based on the condition of the proximal fragment at the time of intervention. Absolute requirements for any method of open reduction include a viable humeral head with maintained articular surface and adequate bone stock to enable fixation. The presence of avascular necrosis of the humeral head, advanced posttraumatic arthritis, or an osteopenic, cavitary humeral head necessitates hemiarthroplasty.

Surgical Techniques

Intramedullary Nails with and without Tension Bands.

Open reduction and internal fixation with intramedullary nails can occasionally be successfully utilized in surgical neck nonunions (Fig. 30-16). An extended deltopectoral approach is used, avoiding excessive soft tissue detachment from the nonunion fragments. The fibrous tissue and pseudarthroses at the nonunion site is removed, along with any preexisting hardware. The bone ends are decorticated. The nonunion fragments are aligned using the bicipital groove as a reference for proper version. Care is taken not to remove excessive bone to maintain deltoid length. If one fragment has a spike, this may be impaled into the other nonunion fragment, thereby improving fixation and fracture stability. If the humeral shaft can be narrowed and reduced within the cancellous portion of the humeral head, this is done to improve the stability and to increase bone contact (Fig. 30-17).

When Ender intramedullary rods are used, they should be modified by placing a superior hole to the already existing large proximal hole. This preoperative rod modification permits more inferior seating of the rod into the rotator cuff, diminishing the incidence of postoperative subacromial impingement due to hardware. One Ender rod is placed in the greater tuberosity, and one is placed in

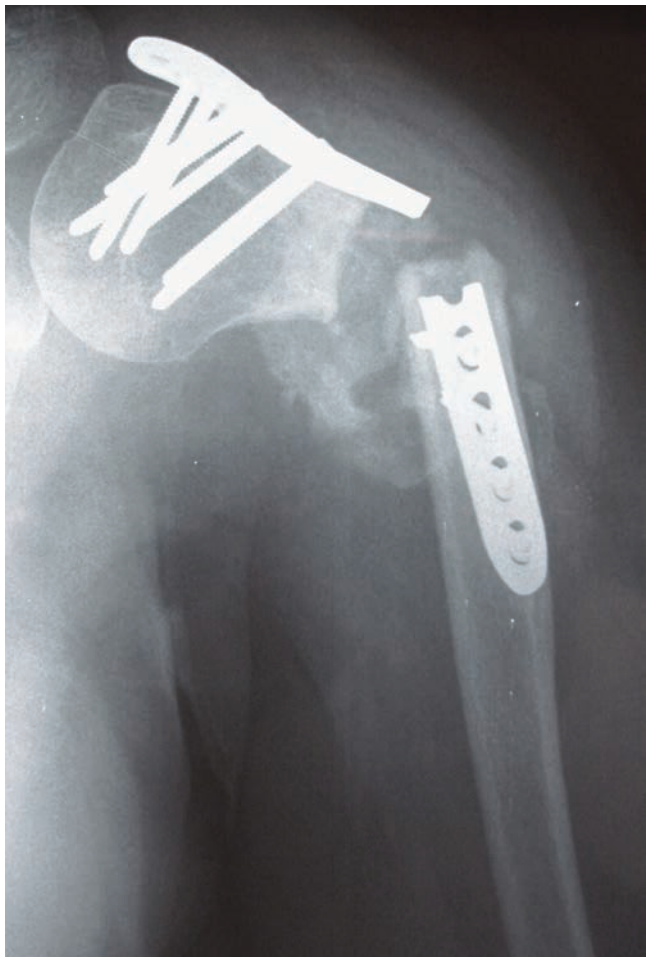


Figure 30-16 Intramedullary locking rod to treat a surgical neck nonunion after failed locked plating.

the lesser tuberosity; both are then placed into the distal intramedullary canal of the humeral shaft.

A hole is then drilled in the humeral shaft approximately 2 cm below the nonunion site, and 18-guage wire or 1-mm nonabsorbable suture is passed through the hole in the superior hole of the Enders rods. A figure-eight tension band technique is used. An additional wire or suture may be used at right angles to the first. We do not use tension band wiring alone, preferring the additional longitudinal and torsional stability afforded by the intramedullary fixation.[50]

Depending on the nonunion type (i.e., atrophic, oligotrophic, or hypertrophic), bone quality, and apposition at the nonunion site, bone grafting may be required.[42] We prefer to use cancellous iliac crest autograft packed at the nonunion site. Corticocancellous strips can be used if needed to restore humeral length.[87] We have not used allograft bone for treatment of the atrophic nonunion and prefer autogenous bone when available.

A sling is placed at the time of surgery. The postoperative physical therapy protocol depends on the quality of internal fixation and stability at the nonunion site noticed at surgery. If the surgeon is confident with the surgical repair, passive and active assisted motion of the shoulder can be effected immediately. Careful radiographic evaluation of healing determines when active motion can commence and when the sling is discontinued. As with any shoulder surgery, passive and active range of motion of the elbow, wrist, and hand are started immediately postoperatively.

Results. Neer applied the combination of an intramedullary nail, tension band construct, iliac bone graft, and abbreviated spica cast application successfully in 12 of 13 cases of surgical neck nonunion.[72] He noted that subsequent hardware removal secondary to symptomatic subacromial impingement and release of adhesions was routinely needed for pain-free, functional motion.

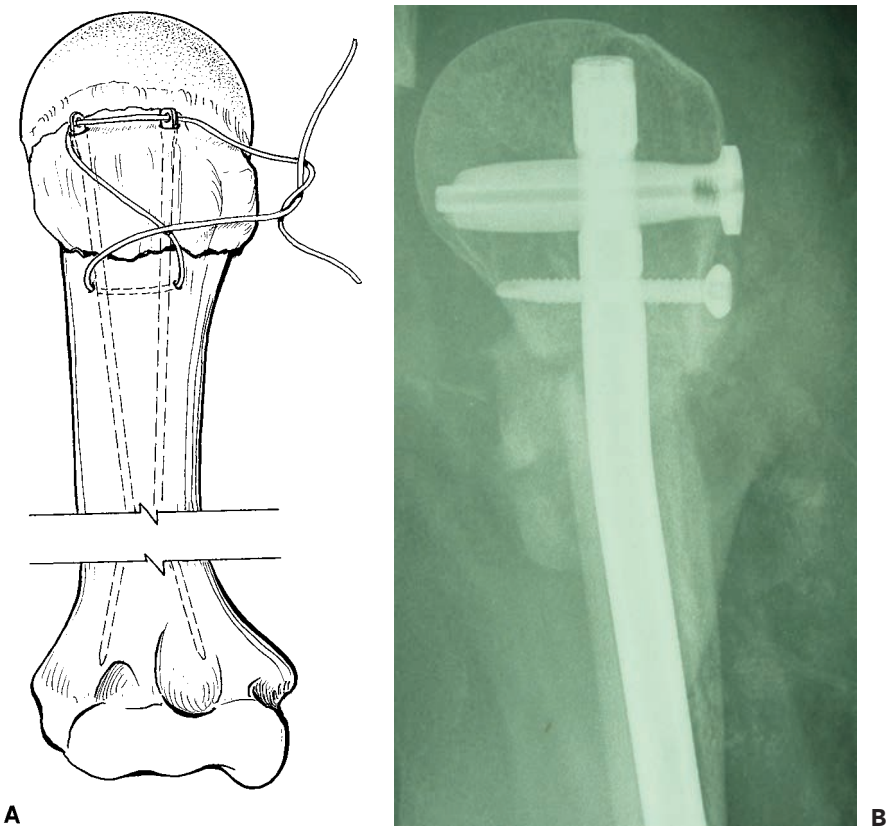
Another series reported on five patients treated with unreamed nails ± a tension band construct.[41] The only patient that evidenced healing at the nonunion was treated with a Rush rod and tension band. All five patients had impingement symptoms secondary to hardware leading to unsatisfactory results.

Nayak et al. reported on successful clinical union in all 10 patients treated with Rush rods, tension banding, and iliac bone grafting.[69] However, two patients evidenced persistent radiographic evidence of nonunion despite good clinical function. Additionally, 80% required reoperation for hardware removal secondary to painful subacromial impingement. The intramedullary rods were not incorporated into the tension band wiring, which may be a critical factor in this failure. Circumflex humeral artery laceration, axillary nerve injury with permanent sequelae, and one cortical perforation with Rush rod insertion were reported complications in this small series.

Norris et al. reported good results using intramedullary rods with a tension band construct.[80] These patients regained excellent motion, and the group did not evidence inferior results to those patients who underwent hemiarthroplasty. The success in this series is likely attributable to the incorporation of the tension band through the proximal holes in the Enders rods, which improves rotational stability and may prevent rod backout into the subacromial space.

These series confirm that intramedullary nails can be successfully utilized in the treatment of surgical neck nonunions, especially when a tension band is incorporated into the construct. However, the high incidence of subacromial mechanical impingement necessitating hardware removal makes this option unfavorable, especially in elderly patients with multiple anesthetic risks. The technical caveat of incorporating the tension band construct through proximal holes in the Enders rods is essential as it provides improved stability and rotational control and may limit backout of the intramedullary device.

Open Reduction and Internal Fixation with Plates. If good-quality bone stock is present and the humeral head fragment is large enough, plate and screw fixation is the procedure of choice. Internal fixation can be performed utilizing



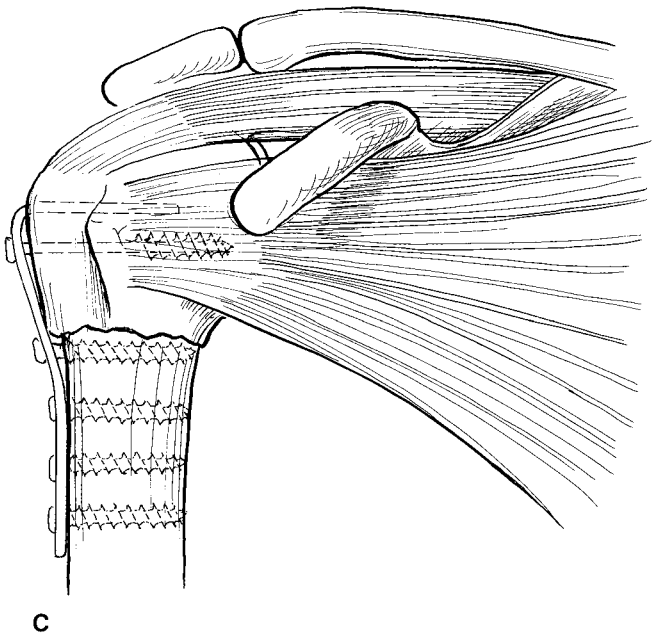


Figure 30-17 **(A-C)** If the humeral shaft can be narrowed and reduced within the cancellous portion of the humeral head, this is done to improve the stability and to increase bone contact. (Reproduced with permission from Warner JJP, Iannotti JP, Gerber C, eds. *Complex and revision problems in shoulder surgery,* 1st ed. Philadelphia: Lippincott-Raven, 1997.)

a variety of plates (T-plates, blade plates, or periarticular locking plates) (Fig. 30-18). The exposure required for plate fixation is greater than that needed for intramedullary rod and tension band fixation, and care must be taken to avoid further devitalization of the nonunion by excessive soft tissue stripping in applying this larger hardware. Humeral head avascular necrosis has been reported with the use of plate fixation of proximal humeral fractures and nonunions, and perhaps the associated increased surgical exposure contributed to this occurrence.[51,99]

The technique for ORIF with plates is performed with the patient in the 30-degree beach-chair position using a standard deltopectoral approach. As the subdeltoid space is dissected, the deltoid muscle is retracted laterally, and the pectoralis major and conjoined tendon are retracted medially. Care should be taken to protect the axillary and musculocutaneous nerves during exposure of this scarred surgical field with potentially distorted anatomy. If preoperative arthrofibrosis exists, arthrolysis of the glenohumeral joint is performed. Arthrolysis of a stiff joint will aid in postoperative rehabilitation and will limit force transmission to the nonunion site.[41] The nonunion site is identified and débrided of all interposing soft tissue, pseudocapsule, and fibrous tissue exposing raw bone ends. If the proximal fragment is small and difficult to control, a 2.5-mm Kirschner wire or the guidewire from a Synthes blade plate set can be useful as a joystick while reducing the nonunion and applying hardware. The wire is drilled into the proximal fragment lateral and posterior to the bicipital groove. Bone contact between the proximal and distal fragments is restored by impaling the diaphyseal fragment into the head of the proximal humerus. Minimal shaping of the distal fragment may facilitate this maneuver. The nonunion site is typically autografted with corticocancellous strips of iliac crest bone graft. As the long head of the biceps tendon is often interposed in the nonunion site, we tenodese the tendon in the area of the intertubercular groove if there is evidence of fraying and tenosynovitis so as to remove it as a source of postoperative pain.

The plate is then applied to the proximal humerus. If a blade plate is used, the guidewire is drilled from the lateral position across the humeral head into subchondral bone. A depth gauge is utilized to determine the appropriate length of the blade and the side plate is contoured to fit the impacted diaphyseal fragment. The rotator interval is incised so that the surgeon has increased exposure and to ensure that the blade of the plate does not violate the articular surface. Screws are placed using standard technique. Several techniques can be added to help prevent failure of the blade in the proximal fragment. As described, impaction of the bone fragments can add stability. Additionally, a cerclage wire or cable can be placed around the proximal shaft and plate to prevent screw pullout. Lastly, a tension band construct can be added by passing 1-mm tapes through the subscapularis and supraspinatus tendons and the proximal plate laterally.

To avoid impingement, care must be taken to avoid placing the plate too proximally. Internal fixation with the humeral head in varus or in excessive internal rotation is another pitfall to avoid with this technique.[99] Alternatively, a locking periarticular proximal humerus plate can be utilized (Synthes, Paoli, NJ).

Postoperative physical therapy follows the same guidelines as described for the intramedullary technique. The protocol may be somewhat accelerated because of the improved bone quality in these usually younger patients.

Results. Vastamaki[99] reported union in all 12 surgical neck nonunions that were internally fixed with an AO T-plate. Nine patients had relief of pain and some improvement in shoulder range of motion and strength. Three patients developed humeral head avascular necrosis.

Healy and coworkers[42] recommended adding a nonabsorbable suture weave through the rotator cuff, and then tying the suture to the T-plate. They felt that this construct reduces the rotator cuff distraction force that pulls the humeral head away from the proximal screw. Sonnabend[94] modified an AO semitubular plate into a blade plate and created two sharp spikes at the proximal end. Combining this technique with bone graft, he achieved union in five of six surgical neck nonunions. He thought there would be a decreased incidence of cutting-out with this construct compared with that of proximal screws. A more inferior position of the plate on the humeral head may be possible with a blade plate than with a T-plate, perhaps reducing the incidence of impingement. Instrum and colleagues,[46] in a cadaveric biomechanical study, revealed comparable fixation strength of these two modes of plate fixation. Palmer and colleagues[81] described the use of a modified 3.5-mm AO dynamic compression plate (DCP) plate with an interlocking screw technique in which screws were placed from the distal plate across the nonunion site and into the proximal fragment through a hole in the intraosseous portion of the 90-degree blade plate. They argued that this technique increases pull-out strength and reduces toggle at the nonunion site.

Galatz et al. recently reported excellent results in 11 of 13 patients treated with open reduction and internal fixation with blade plates or T-plates and bone graft.[32] These elderly patients had significant improvements in pain, motion (24 to 144 degrees of forward elevation), and function. They concluded that open reduction and internal fixation with autogenous bone graft results in excellent outcomes even in patients older than 65 years with significant medical problems.

Humeral Head Hemiarthroplasty Method. Humeral head prosthetic replacement can be necessary in the management of surgical neck nonunions, especially in cases in which internal fixation is precluded because of poor bone quality, small humeral head fragment size, comminution-associated articular surface defects, or head collapse caused

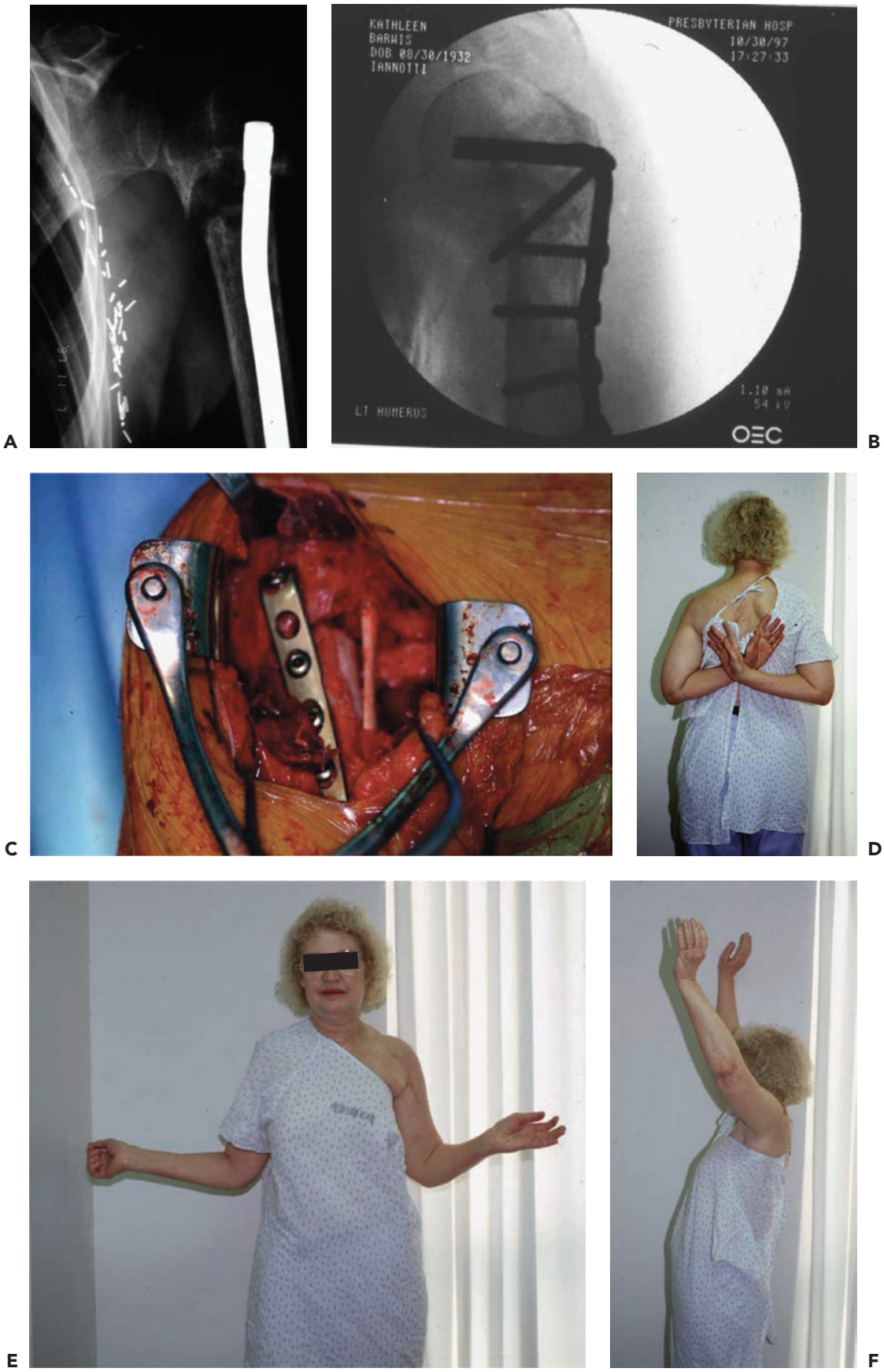


Figure 30-18 Surgical neck nonunion treated with a blade plate and bone graft. It healed with an excellent clinical result in a middle-aged patient.

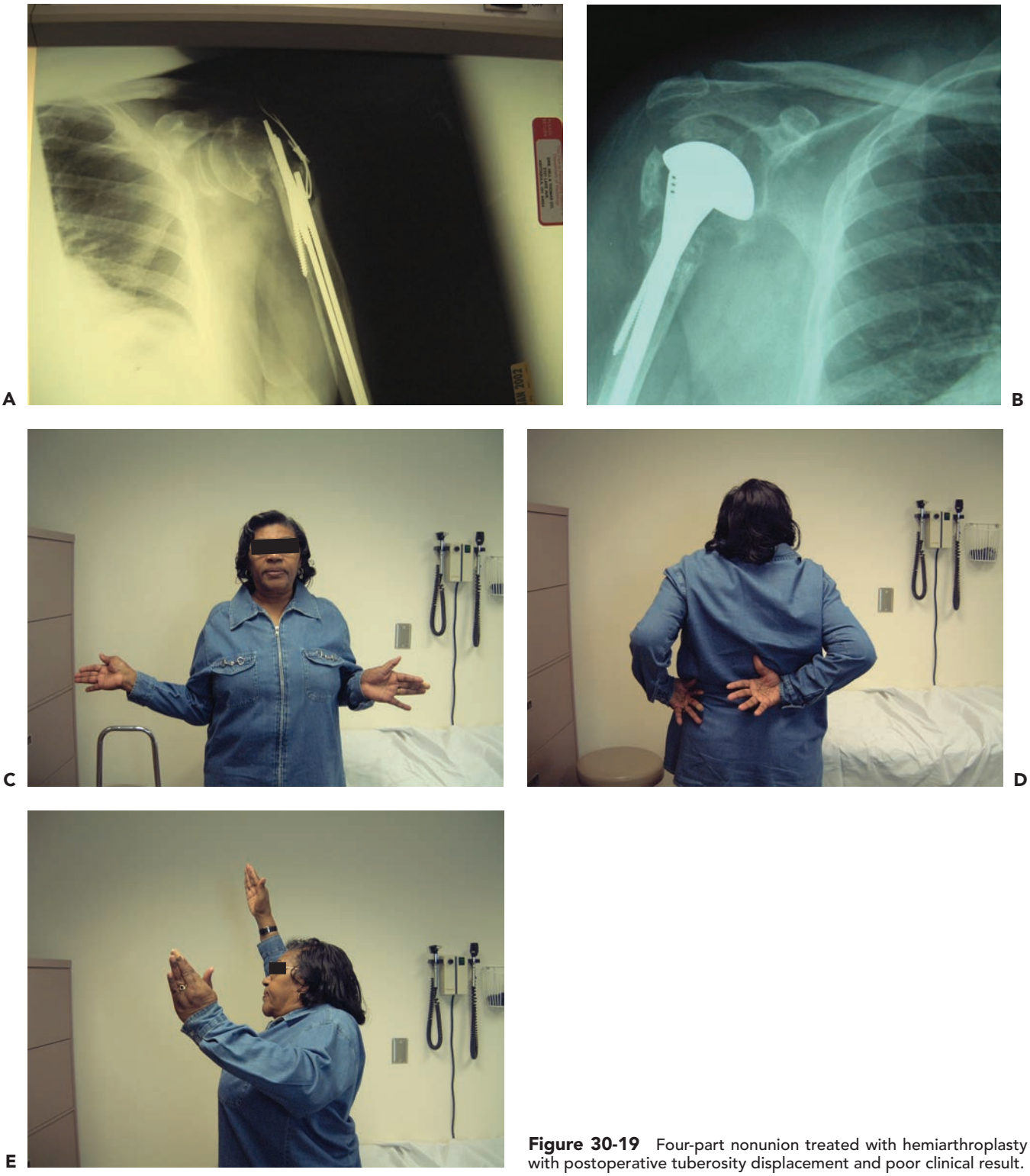


Figure 30-19 Four-part nonunion treated with hemiarthroplasty with postoperative tuberosity displacement and poor clinical result.

by avascular necrosis. In these circumstances the tuberosities are small with poor bone quality, and tuberosity nonunion after prosthetic arthroplasty is common. When tuberosity nonunion occurs, the functional results are often poor (Fig. 30-19). Total shoulder arthroplasty is indicated if concomitant glenoid articular surface defects exist. In these cases the tuberosities are osteotomized and cement fixation is required for stem fixation. Osteotomy of the tuberosities is at the bicipital groove and osteotomy of the articular surface, essentially creating a four-part fracture (Fig. 30-20).

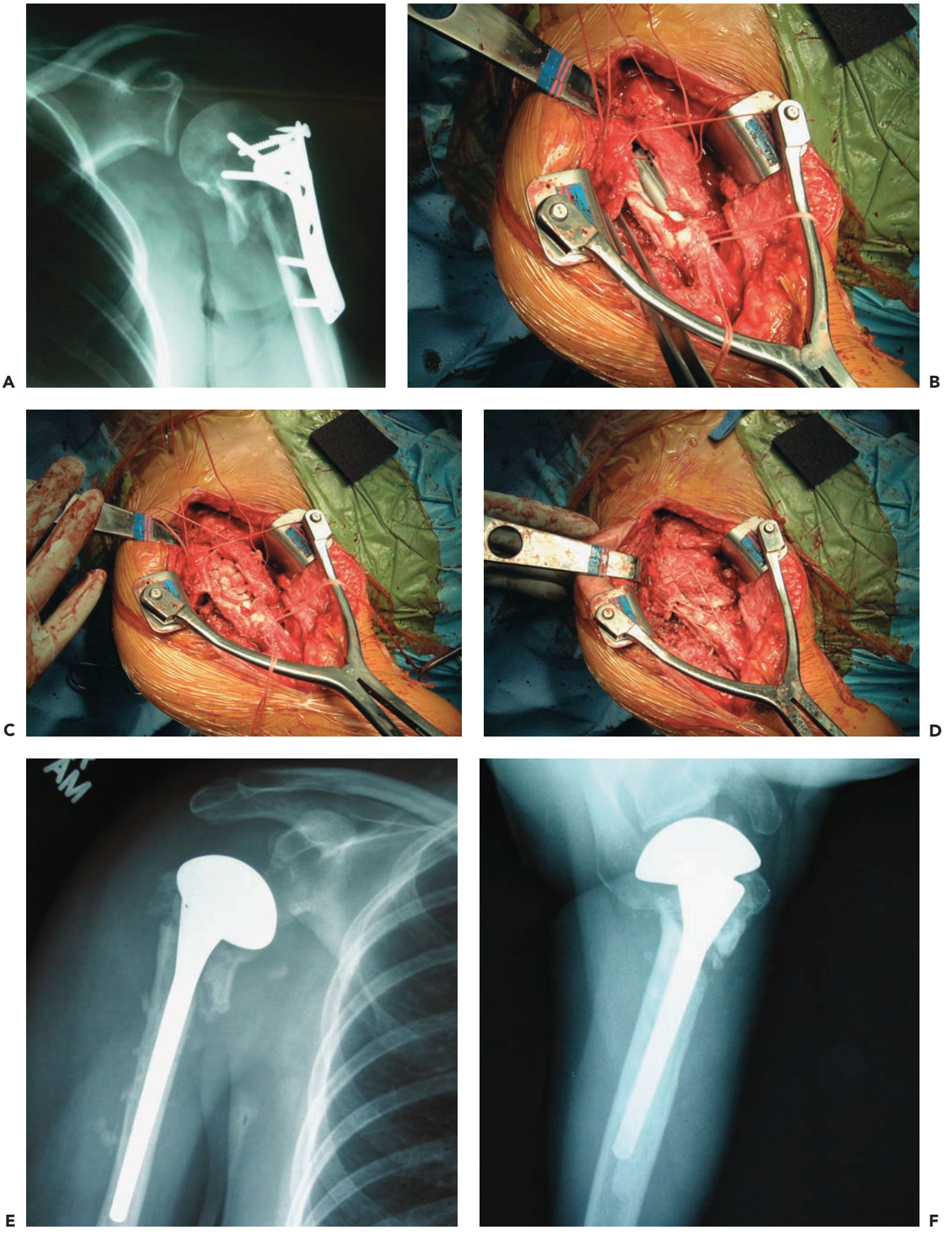


Figure 30-20 Three-part nonunion treated with hemiarthroplasty with good tuberosity healing and good function postoperatively.

Neer advocated the use of iliac crest grafts between the preserved tuberosities to facilitate bony contact and healing of the tuberosities to the shaft and to maintain proper musculotendinous length of the deltoid and rotator cuff musculature.[72] Alternatively, bone graft can be obtained from the humeral head if sufficient. Norris[75] described a technique that can be utilized to avoid splitting the tuberosities individually. A hole can be made in the humeral head through the rotator interval, and the tuberosities can be shish-kebabed as a ring below the prosthetic collar. This technique is desirable as it will improve tuberosity healing and minimize loss of fixation; however, it is technically demanding and fracture of the tuberosity ring can easily occur when passing the prosthetic through the rotator interval into the humeral shaft. We utilize similar surgical technique and postoperative rehabilitation protocol to that described in the preceding section on malunions.

Three-Part and Four-Part Nonunions

The preceding discussion has focused on surgical neck nonunions of the proximal humerus, which are by far the most common. In contrast, three- and four-part nonunions are not very common. Malunions of the tuberosities more commonly accompany surgical neck nonunions than do true associated tuberosity nonunions. Nevertheless, these late sequelae of proximal humerus fractures do occur and warrant discussion.

As in surgical neck nonunions, the critical evaluation of three- and four-part nonunions involves assessment of the quality and vascularity of bone attached to each of the tuberosity fragments and of the integrity of the vascularity and congruity of the articular surface. In those rare cases in which the bone quality is sufficient and the articular cartilage is satisfactory, open reduction and internal fixation with tension suture stabilization of the free tuberosity is preferred. If, however, as is typically the case, the articular segment is incongruous or avascular, prosthetic arthroplasty is the preferred surgical option.[77] Technical themes warranting attention remain the maintenance of humeral length, mobilization of the rotator cuff, and secure fixation of the tuberosities with the addition of autogenous bone graft. Again, our preferred technique for hemiarthroplasty in this setting is discussed in the section on proximal humerus malunions.

In older patients with poor bone quality, a reverse total shoulder replacement is likely to give better clinical results because this prosthetic is less dependent upon healing of the tuberosities (Fig. 30-21).

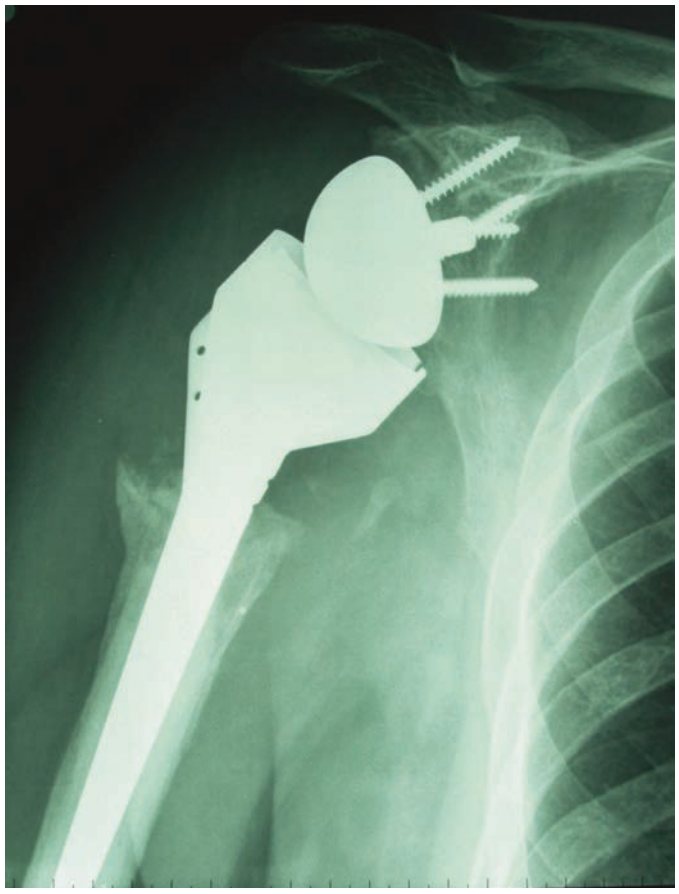
Results

The results of prosthetic treatment of surgical neck and three- and four-part nonunions will be discussed in this section. There is a relative paucity of literature regarding the prosthetic treatment of proximal humerus fracture nonunions. Most series include small numbers and discuss the entire spectrum of posttraumatic sequelae of proximal humerus fractures in whole without focusing particular attention on specific nonunion type. Thus, a rather confusing body of literature exists in which comparisons between series are difficult. In general, the results of prosthetic arthroplasty for three- and four-part nonunions are inferior to those of prosthetic treatment for similar acute fractures.[14,31,72,77,102] As is true for late prosthetic treatment of malunions, pain relief is usually achieved, but shoulder range of motion, strength, and function are often limited. Accordingly, patients should be prepared for "limited-goals" outcomes following prosthetic replacement. Patients should expect excellent pain relief, but younger, active patients may be disappointed by a lack of overhead motion and strength.

Norris and Turner[78] described four elderly patients with surgical neck nonunions who had satisfactory results in pain relief and function with cemented humeral head hemiarthroplasty using local bone graft. No complications were reported. In another report, however, Norris[77] reported higher complication rates and poorer ultimate shoulder function in the prosthetic treatment of proximal humerus nonunions if a history of prior, failed attempt at open reduction and internal fixation existed. Likewise, Frich and associates[31] evidenced decreased forward elevation and increased pain scores in surgical neck nonunions treated with prosthetic replacement when compared to the results of patients treated with hemiarthroplasty for acute surgical neck fractures.

Nayak and colleagues'[69] series of surgical neck nonunions treated with operative intervention included seven elderly patients treated with hemiarthroplasty. Tuberosity osteotomy was required in all seven of these patients. Those patients treated with prosthetic replacement had better pain relief, but less improvement in range of motion (active forward elevation of 20 degrees improved to 110 degrees) than those treated with internal fixation (20 to 140 degrees). Interestingly, this difference in motion existed despite the presence of mechanical impingement symptoms necessitating hardware removal in 8 of 10 patients treated with intramedullary fixation and tension band construct. Complications including postoperative axillary neuropathy, presumed nonunion of the tuberosities, inferior subluxation of the prosthetic humeral head, and subacromial impingement were reported in the hemiarthroplasty group.

Six patients in Healy and colleagues'[41] review of 25 surgically treated proximal humerus nonunions were treated with hemiarthroplasty and rotator cuff reconstruction. Three patients had good results; two, fair results; and one, a poor result. Although these patients had good pain relief, their shoulder motion and strength was limited; the average range of motion only improved to 72 degrees of total elevation, 30 degrees of external rotation, and internal rotation to the midlumbar spine. Dines et al.'s[25] series of posttraumatic sequelae of proximal humerus fractures treated with hemiarthroplasty included six proximal humerus nonunions. The classification of nonunion type was not included. Active



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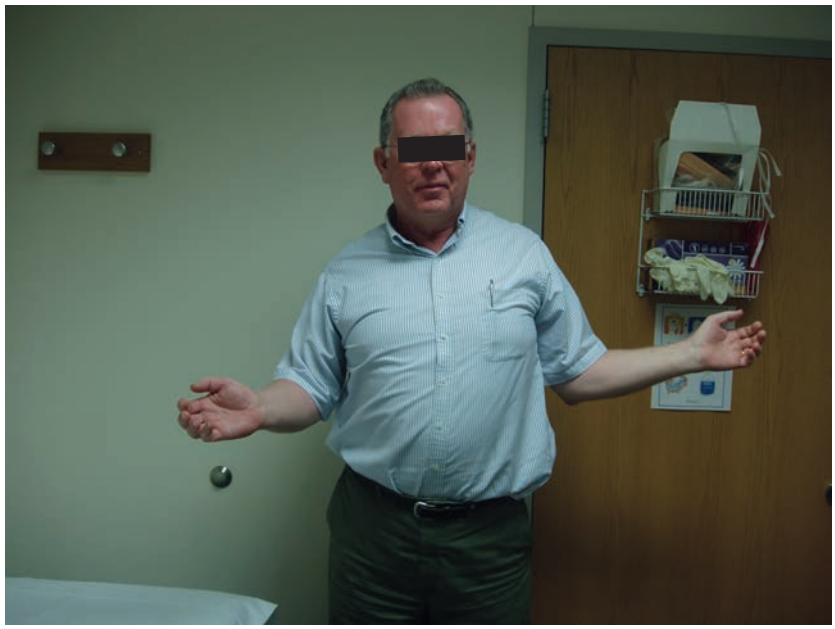

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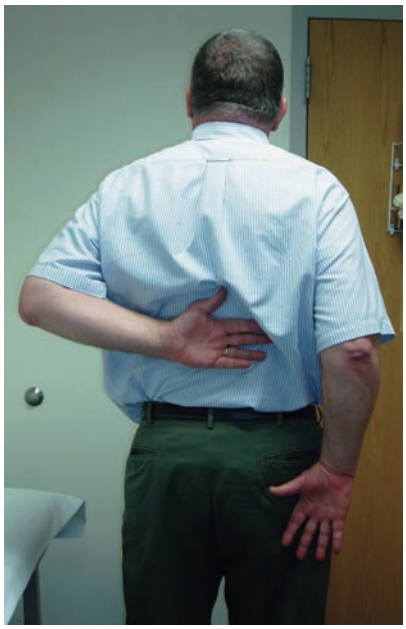

D

Figure 30-21 Proximal humeral arthroplasty with nonunion tuberosity treated with reverse total shoulder arthroplasty with a good clinical result.

forward elevation improved from 48 degrees to 120 degrees postoperatively, and all six patients had good or excellent results as determined by the Hospital for Special Surgery (HSS) scoring system. Five of the six patients required tuberosity osteotomies during the hemiarthroplasty.

Antuna and colleagues[4] have published the largest series of proximal humerus nonunions treated with shoulder arthroplasty. All 25 patients in this series had surgical neck nonunions and nine of the patients had three- or four- part nonunions. Shoulder arthroplasty resulted in significant pain relief and improvement in range of motion. Mean active elevation improved from 41 degrees preoperatively to 88 degrees postoperatively. Eleven patients required tuberosity osteotomies at the time of arthroplasty, and 12 patients were reported to have nonunion, resorption, or malunion of the greater tuberosity following arthroplasty; this likely attributes to the lack of above shoulder strength and motion. Additional complications included reoperation for periprosthetic fracture, reoperation for instability, and reflex sympathetic dystrophy. On the basis of a modified Neer result rating system, there was one excellent result, 11 satisfactory results, and 13 unsatisfactory results. Of significant note, the nine patients with a three- or four-part nonunion had significantly less pain relief and were less satisfied with the results of the arthroplasty than were those patients with only a surgical neck nonunion.

Boileau and colleagues'[13] classification of posttraumatic sequelae of proximal humerus fractures included nonunion of the surgical neck (type III). Their series included six cases of surgical neck nonunions, all of which required a greater tuberosity osteotomy to enable prosthetic implantation. In terms of postoperative range of motion and function, these patients did dismally improve in active elevation from 50 degrees preoperatively to only 63 degrees postoperatively. These patients also had less improvement in Constant scores when compared to those patients treated with hemiarthroplasty for avascular necrosis, locked dislocations, or tuberosity malunions. The authors discovered that the most significant factor predictive of poor functional outcome was the need for a tuberosity osteotomy at the time of prosthetic implantation. In fact, all six of these patients requiring tuberosity osteotomy had fair or poor results without active elevation above shoulder level. These results led the authors to conclude that "replacement arthroplasty should be abandoned in the treatment of surgical neck nonunions." Another recent series of posttraumatic sequelae of proximal humerus fractures treated with arthroplasty has also reported that proximal humerus nonunions yield the least favorable results among the spectrum of posttraumatic sequelae treated with shoulder replacement.[66]

Certainly, open reduction and internal fixation is preferred whenever bone stock, articular congruity, and vascularity of the proximal fragment are all satisfactory; however, when these criteria are not fulfilled prosthetic replacement remains the only viable treatment option. Again, these series repeatedly exhibit reliable improvement in pain but limited improvement in strength, motion, or overhead shoulder function. When criteria for open reduction and internal fixation of proximal humerus are not met (i.e., vascularity and quality of proximal bone, position of tuberosities, condition of articular surface, etc.), patients must be prepared for the reality of the challenging nature of this condition and the "limited-goals" expectation of the procedure. As is true for arthroplasty for the treatment of proximal humerus malunions, osteotomy of the tuberosities should be avoided at all costs. The use of a modular replacement system is key in this aspect and, by avoiding complications involved in failed tuberosity union following osteotomy, may improve motion, strength, and function.

Given the poor results of hemiarthroplasty for treatment of proximal humeral nonunion, strong consideration should be given to the use of the reverse total shoulder arthroplasty for these cases when they occur in the older, retired, or sedentary patient. Although results of the reverse shoulder have not been reported to date for specific treatment of these nonunions, it would be expected to yield better functional results than hemiarthroplasty.

SUMMARY

Although the vast majority of proximal humerus fractures that are treated either conservatively or operatively heal uneventfully with good return of function, a small but not insignificant number of patients will suffer debilitating disability from one or more of the sequelae of proximal humerus fractures. In general, the best treatment for these complex and challenging issues is avoidance with appropriate initial therapy as the literature repeatedly evidences superior outcomes for surgical intervention in acute fracture care than in the treatment of these late sequelae. Unfortunately, avoidance of these late complications is not always possible or realistic, especially in the tertiary care, referral setting. Thus, familiarity with the diagnosis, classification, and treatment options for this challenging spectrum of disorders is a mandatory component of the armamentarium of the shoulder surgeon. Comprehensive treatment of all osseous *and* soft tissue pathology in these problematic cases should always be the goal, regardless of surgical technique utilized. In general, preservation of congruency and vascularity of the articular segment of the proximal humerus warrants attempt at open reduction and internal fixation ± arthrolysis and/or rotator cuff reconstruction when warranted. However, in cases in which the articular segment is incongruous, avascular, and unsalvageable, prosthetic arthroplasty remains the only realistic treatment option. It is very clear that the addition of tuberosity osteotomies to any prosthetic replacement markedly diminishes the likelihood of good functional

results. Thus, whenever possible, a modular prosthesis should be utilized to enable implantation of the humeral head component without the requirement for osteotomy of the tuberosities. The use of a surface hemiarthroplasty in selected cases of malunion may allow adaptation of the prosthetic, further avoiding an osteotomy. The reverse shoulder arthroplasty may result in improved functional results in selected patients that require a tuberosity osteotomy.

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31 Fractures of the Clavicle

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INTRODUCTION

Fracture of the clavicle is common, and it has long been thought that its inherent reparative capacity will lead to rapid healing despite little more than symptomatic treatment.[273] Deformity has been described more often as a cosmetic concern since function is satisfactory despite malunion.[189,190] It has been suggested by many that primary operative intervention is meddlesome and will result in unnecessary complications.[219,273] However, although the standard of care has long been conservative therapy, operative management of acute clavicle fractures, particularly in polytraumatized patients and in selected cases of young athletes, has proven to be an excellent option.[8,136–141] Despite the proximity of major vascular, nervous, and cardiopulmonary structures, associated injury is uncommon.

This chapter will present to the reader the changing paradigms related to the management of fracture and related complications of the clavicle. A large percentage of clavicular fractures occur in children and heal readily with remodeling of deformity.[231,294] This is not always the case with displaced fractures in adults.

Interest in clavicular nonunion is relatively new[25] and has demonstrated that displaced fractures of the middle portion of the clavicle can in fact be troublesome injuries in adults.[142,188,307,309] Failure of bony union following clavicular injuries can lead to progressive shoulder deformity, pain, impaired function, and neurovascular compromise. Malunion may also contribute to weakness, pain, and neurovascular compromise.[86,87] Data on displaced distal clavicular fractures in adult patients have demonstrated sufficient difficulties with healing to consider primary operative treatment.[62,216–218] A recent investigation focusing on the results

of the treatment of displaced midclavicular fractures in adult patients suggests that this subset of fractures may also be prone to nonunion and delayed union.[307]

ANATOMY

The clavicle is unique among diaphyseal bones in many respects, among them its development, shape, structure, and anatomic relationships. It is the first bone to ossify in the embryo, occurring in the fifth gestational week, and the only long bone to ossify from a mesenchymal anlage (intramembranous ossification). While a number of investigations documenting the histologic evaluation of the embryonic clavicle report that ossification proceeds from two separate centers,[9,71,76,111,187] others assert that a narrow interconnection between these two centers is present from the outset, but may not be seen in any one particular histologic section and will therefore result in sampling error.[92,99,153] Dispute exists as to the cause of congenital pseudoarthrosis of the clavicle, with some suggesting that it is a result of the failure of these two centers to coalesce[150,211] and others believing that pressure from the subclavian artery inhibits ossification, leading to pseudoarthrosis.[180]

Growth in length initially occurs through expansion of the central ossification centers and later by enchondral ossification via epiphyseal growth centers acquired at each end of the bone. The medial (sternal) physis accounts for approximately 80% of the longitudinal growth of the clavicle.[235] Ossification of the sternal epiphysis of the clavicle occurs in the midteenage years and is very difficult to visualize on routine radiography.[69,235] The acromial epiphysis does not ordinarily form a secondary ossification center.[299] Both the sternal and acromial physes can remain open into the third decade of life, particularly the sternal physis, which typically remains open until approximately age 25 years in females and 26 in males.[133,234,235,299] As a result, apparent dislocations of the acromioclavicular or sternoclavicular joints are more likely to be physeal separation injuries in adolescents and young adults.[59,60,200,303]

A number of influences on clavicular length have been investigated. The influence of gender has been clearly shown. In a comparison between African-American males and females and white males, Terry[296] demonstrated that male clavicles are longer than female clavicles. Martin and Saller[193] emphasized that male clavicles in all races are longer than those of females. Dzigora[73] published an average length of 15.6 cm for clavicles of males and 14.3 cm for those of females of Russian ancestry. Investigations looking at body side reveal only the mildest divergence of clavicle length. The left clavicle tends to be longer than the right; this was observed by both Martin and Saller[193] and Dzigora.[73]

The clavicle is named for its S-shaped curvature with one apex anterior medially and another posterior laterally, resembling the musical symbol, clavicula.[205] The larger medial curvature widens the space for passage of neurovascular structures from the neck into the upper extremity via the costoclavicular interval. Men display significantly more angulation of the clavicle than do women.[118,162] The cause for this anatomic phenomenon appears to be the interaction of muscle and bone, since most authors find a positive correlation between increased musculature and clavicular angles. Body side also has significant influence regarding the medial clavicle angle, as confirmed by both Dzigora[73] and Bardeleben.[12] The increased musculature on the right side in right-handed people most likely determines these effects. However, another plausible explanation for clavicular curvature considers the fact that the clavicle is the first bone ossified during embryonic development (the fifth embryonic week) and later the first bone with which the developing thorax and the developing limbs of the upper extremity must conform.[209,250] The theory is that the S-form develops as a result of these interactions. Since the thoracic diameter is well accepted to be larger in males than in females, this would explain gender differences. However, Kummer and Lohscheidt[162] pointed out Pauwels' developmental principles of long bones: They are unable to significantly change the axis of an ossified bone. The axes are finished prior to ossification, in the essential stages of development. Since clavicles ossify early, an essential bone-shape altering factor would have to exist very early in development.

Inman et al. have suggested that the curvature of the lateral third of the clavicle contributes to range of motion of the shoulder girdle by allowing approximately 30 degrees of motion between the scapula and clavicle through the acromioclavicular joint.[126,127,179] According to their description, this motion occurs via inferior translation of the medial portion of the scapula leading to abduction of the scapula through the acromioclavicular joint, a motion that might be thought impossible considering the rigid interrelationship between the clavicle and scapula maintained by the stout coracoclavicular ligaments. However, the lateral curvature and 50-degree rotational motion of the clavicle on its longitudinal axis allow for inferior translation of the attachment of the coracoclavicular ligaments on to the posteriorly directed apex of the lateral clavicle along with the scapula. Inman et al. have likened this to the action of a crankshaft[126,127] (Fig. 31-1).

Others dispute this description, claiming that little motion occurs at the acromioclavicular joint and that the scapula actually rotates along with the clavicle.[48]

The clavicle is made up of very dense trabecular bone lacking a well-defined medullary canal. In cross section the clavicle transitions gradually between a flat lateral aspect, a tubular midportion, and an expanded prismatic medial end.[15,113] (Fig. 31-2)

The average measured diameter of the medullary canal over the course of the clavicle bone is depicted in Fig. 31-2. With its natural S-curve, the clavicle has a double funnel-shaped diameter. At the sternal end, the diameter measures

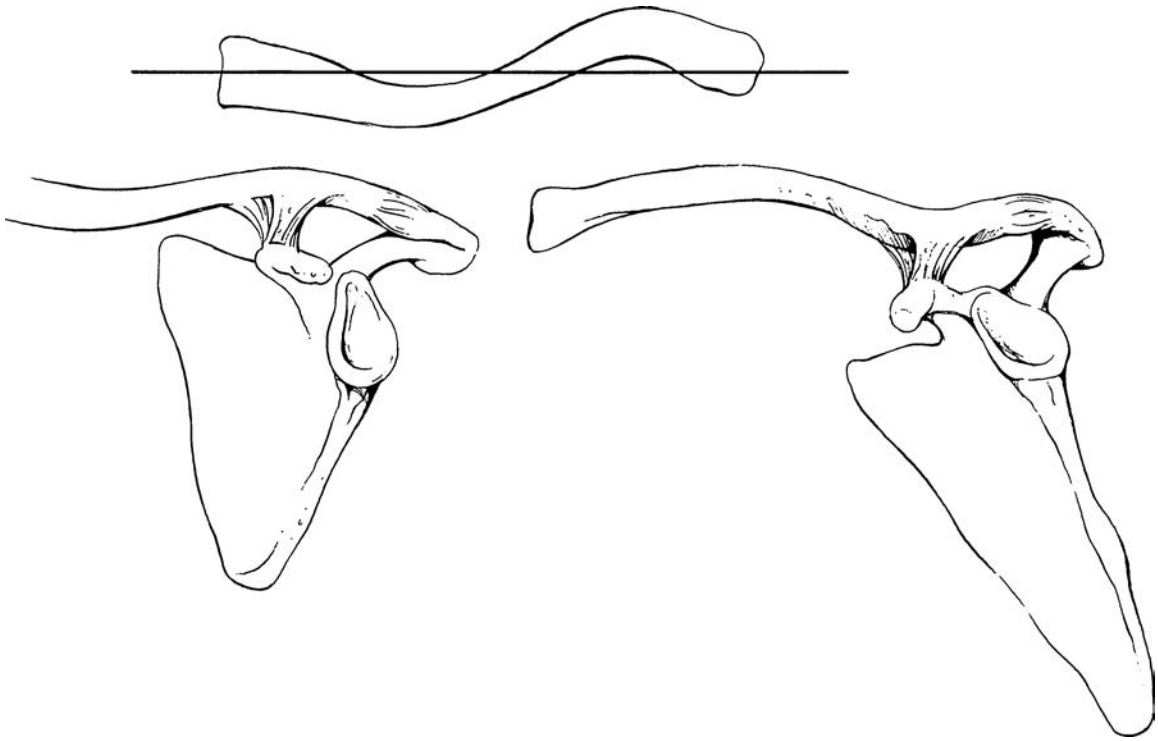


Figure 31-1 There are three axes of clavicular motion: anterior–posterior, superior–inferior, and rotational. According to Inman's description, the 50-degree rotational motion of the clavicle, in combination with the apex posterior curvature of the lateral clavicle, allows for inferior translation of the attachment of the coracoclavicular ligaments on the lateral clavicle. This in turn permits the medial aspect of the scapula to translate inferiorly, with the glenoid abducting through the acromioclavicular joint. This so-called crank-shaft mechanism provides 30 degrees of the total 60-degree contribution of scapulothoracic motion to shoulder abduction. It is important to note that other authors dispute this mechanism.

approximately 18 mm, and decreases in a linear manner to approximately 6.7 mm at the midpoint. Continuing on, the diameter increases in a similar fashion, eventually reaching 15 mm. The narrowest diameter is measured exactly at the meeting point of the medial convexity with the lateral concavity, the most frequent location of clavicle fractures. These peculiarities of the bone's curvature and cross-sectional anatomy as well as its bony structure become important when intramedullary fixation of the clavicle is considered[167] (see Fig. 31-2).

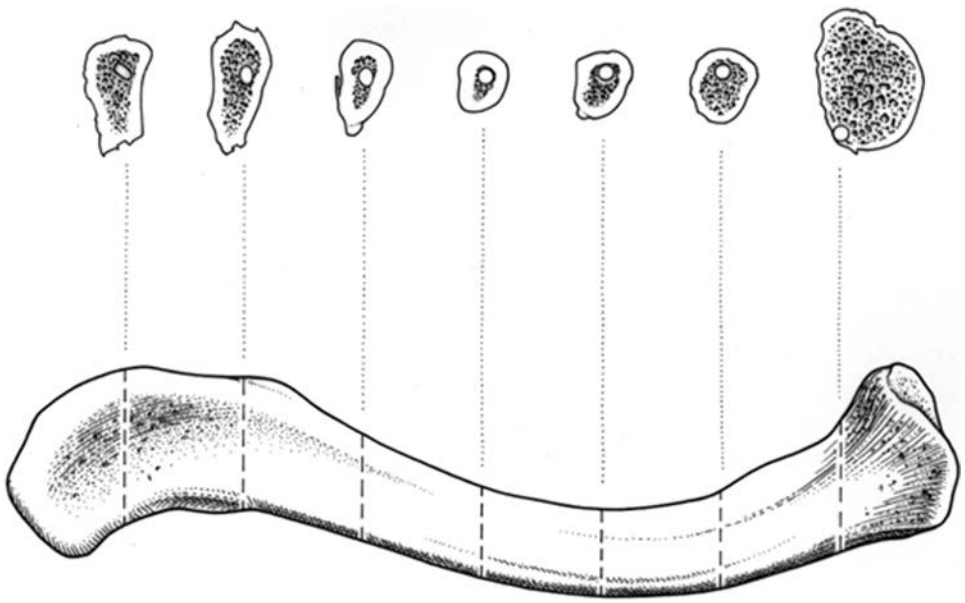


Figure 31-2 The diameter of the medullary canal cross section, the cortical thickness, and the position of the intramedullary nail within the clavicular canal: Contact point and the cortical thickness next to the respective positions become clear. In the medial and lateral portions of the bone are the danger zones for nail perforation, while in the middle third a relative narrowing is present.

The clavicle is subcutaneous throughout its length and makes a prominent aesthetic contribution to the contour of the neck and upper chest. The supraclavicular nerves run obliquely across the clavicle just superior to the platysma muscle and should be identified and protected during operative exposure to offset the development of hyperesthesia or dysesthesia over the chest wall.

The articulation of the clavicle with the trunk is stabilized by the stout costoclavicular and sternoclavicular ligaments. The subclavius muscle may also contribute to stability in this region of the clavicle. The coracoclavicular and acromioclavicular ligaments stabilize the relationship of the distal clavicle with the scapula. The upper portion of the insertion of the trapezius muscle and the anterior portion of the origin of the deltoid further stabilize the lateral clavicle through their attachments to its posterior and anterior aspects, respectively. Fractures in these regions of the clavicle tend to be relatively stable, provided that the described ligamentous and muscular relationships are not disrupted in the traumatic injury.

In displaced fractures and ununited fractures of the clavicle, the most common deformity includes medial–lateral shortening, drooping, adduction, and protraction of the shoulder girdle. The forces contributing to persistence or worsening of deformity following fracture include the weight of the shoulder as transmitted to the distal fragment of the clavicle primarily through the coracoclavicular ligaments as well as the deforming forces of the attached muscles and ligaments. The medial fragment is elevated by the clavicular head of the sternocleidomastoid muscle, which inserts onto the posterior aspect of the medial portion of the clavicle. The pectoralis major contributes to adduction and inward rotation of the shoulder.[121–124]

Bone density correlates strictly with the dynamic and static loading forces applied to a bone.[162,246] In the clavicle, the medial portion of the bone, where increased functional loading is most apparent, displays a clearly higher density. At the intersection of the middle to lateral thirds of the clavicle, there is a significant decrease of bone density. The midportion is the thinnest and narrowest portion of the bone and represents a transitional region, both in curvature and cross-sectional anatomy, making it a mechanically weak area.[113] This, in part, explains the increased frequency of fractures occurring in the middle and lateral thirds of the bone (80% of clavicular fractures occur in the middle third, 10% to 18% occur in the lateral third, and 2% to 10% occur in the medial third). Fractures of the medial third of the clavicle are extremely rare.[27,75,216,218,241,269]

Considering the intimate relationship of the clavicle to the brachial plexus, subclavian artery and vein, and the apex of the lung, it is surprising that injury to these structures in association with fracture of the clavicle is so uncommon. Brachial plexus palsy may develop weeks or years following injury, due to hypertrophic callus with or without malalignment of the fracture fragments leading to compromise of the costoclavicular space.[14,40,52,82,106,124,142,147,159,196,204,264,275,302,312] Narrowing of the costoclavicular space due to malunion or nonunion can also lead to a dynamic narrowing of the thoracic outlet.[13,18,31,51,89,181,210,254,289]

FUNCTION

A review of the comparative anatomy literature reveals that the clavicle has developed from the "os thoracale," a construct of the sternum and clavicle. This "distance-maintainer" between the breastbone and scapula was first observed in fossils of the earliest reptiles, who lived approximately 350 million years ago (Devon). The abilities of the "walking fish," of which the coelacanth is the most well known, were strictly dependent on the development of abduction in the front extremities. Further evidence for the phylogenetically early development of the clavicle is that it ossifies very early during ontogenesis. The functional consequence is, from an evolutionary perspective, the ability to abduct and raise the arms. Conversely, in animals without the ability to abduct the front extremities (i.e., sheep or horses), during ontogenesis the clavicle forms and is even temporarily ossified before completely disappearing by the time of birth.

A clavicle is not beneficial to running and jumping quadruped mammals.[17,18,40,60,179,243,244] In contrast to the quadrupeds, who derive stability and strength from close association of the shoulder girdle with the trunk, in simians the clavicle enhances upper extremity function for swinging through trees. It holds the glenohumeral joint and the upper extremity away from the trunk in all positions. The clavicle enhances overhead activity (combination of shoulder abduction and elevation), particularly in actions requiring power and stability, and resists those tensile forces that become so prominent in activities required by arboreal mammals. It is not surprising then that in cases of clavicle fractures in humans where there is shortening of the sternum–scapula distance, there is a subsequent limitation in shoulder abduction and elevation. The clavicle also serves as a bony framework for muscular attachments, provides protection for the underlying neurovascular structures, transmits the forces of accessory muscles of respiration (e.g., the sternocleidomastoid) to the upper thorax; and contributes to the aesthetics of the base of the neck.[179,209]

It is of interest that children with cleidocranial dysostosis (clavicular aplasia) adapt surprisingly well to an absence of clavicles. However, limitations have been observed with regard to overhead activities requiring strength, stability, and dexterity in cleidocranial dysostosis patients.[14,40,59,60,126,127,190,297] If these congenitally aclavicular children have notable functional deficiencies in comparison with normal children, then one must be concerned about what will happen when the learned coordinated

manipulation of the complex interaction of various muscle groups, ligamentous attachments, and interarticulations of the shoulder girdle is disrupted by sacrificing clavicular continuity in adult patients.[15,72] In fact, some studies have suggested that malunion alone (particularly shortening) can cause pain and inhibit function.[84,85,88]

Because some reports document good function following total or subtotal resection of the clavicle for infection, malignancy, or access to neurovascular structures in small series of patients,[1,55,80,90,108,126,187,310] some authors went so far as to encourage consideration of the clavicle as an expendable or surplus part of the skeleton.[108] Resection of the clavicle has been recommended both in the treatment of clavicular nonunion[36,214] as well as in the treatment of fresh clavicular fractures.[198,249]

It is certainly clear that some patients do very poorly following clavicular resection,[271,286] especially those with trapezial paralysis.[57,142,247,310] We therefore feel strongly that this procedure should be reserved for the unusual situation in which a salvage procedure becomes necessary. The clavicle plays an important functional role in the shoulder girdle, and every effort should be made to preserve or restore normal length and alignment in the treatment of clavicular disorders.

CLASSIFICATION

Surgeons interested in clavicle fractures have long distinguished midclavicular fractures from fractures of the medial or lateral end.[21,117] Following descriptions by Allman,[3] Rowe,[273] and Neer,[216–219] the clavicle has been divided into thirds for purposes of classification. This proves somewhat arbitrary when one considers that the majority of clavicular fractures occur at a distance from the lateralmost aspect of the bone, which falls on a roughly Gaussian distribution between approximately 30% and 60% of the length of the bone.[197] In the majority of reported series, separation of clavicular fractures as occurring in one of the thirds of the bone was most likely based upon interpretation from standard radiographs, rather than precise measurement. This implies that division of the large numbers of clavicular fractures occurring about the middle third–distal third junction as belonging to either the distal third or middle third group is often imprecise and may be arbitrary.

In Neer's defining work on distal clavicular fractures,[216–219] he considered fractures distal to the proximal limit of the trapezoid ligament as distal clavicular fractures and distinguished two types: Type I represents a fracture in which both the trapezoid and conoid ligaments remain intact and are attached to the medial fragment, thereby providing stable reduction of the fracture; a type II fracture consists of a fracture in which the trapezoid ligament remains attached to the distal fragment while the conoid ligament is ruptured and no longer maintains reduction of the medial fragment. In type II fractures, the loss of coracoclavicular ligament restraint on the medial fragment results in wide displacement of the fracture fragments, and in Neer's experience, an increased risk of nonunion[219] (Fig. 31-3).

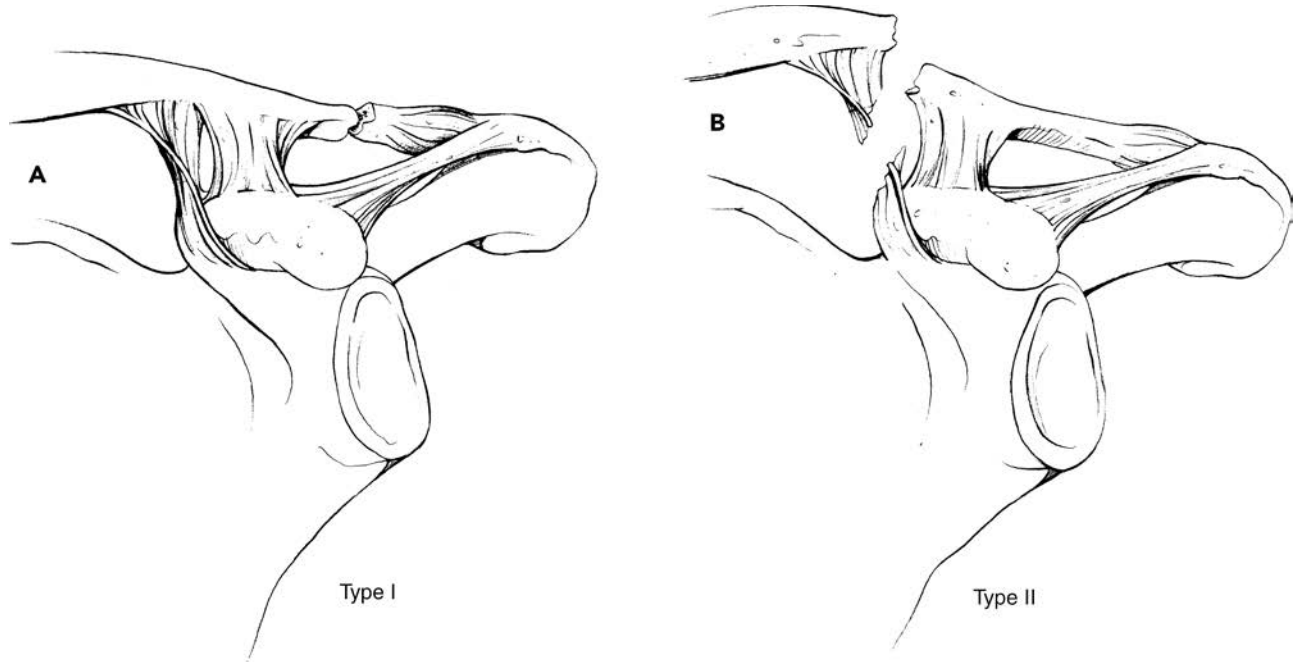


Figure 31-3 When the distal end of the clavicle is fractured, the ligaments may either **(A)** remain intact and serve to maintain apposition of the fracture fragments (type I) or **(B)** rupture, allowing wide displacement of the fragments (type II).

Rockwood subsequently recommended division of type II fractures of the distal clavicle into two subsets: fractures of the distal clavicle in which both the conoid and trapezoid ligaments remain attached to the distal fragment as type IIA and those in which medial fragment instability is a result of disruption of the coracoclavicular ligaments (Neer's original description) as type IIB.[270,272] It is unclear, however, how type IIA fractures differ from more distal midclavicular fractures, especially considering the fact that the most common site of midclavicular fracture is at the junction of the middle and distal thirds.[293] We believe Neer's classification to be more applicable as it serves the role of distinguishing fractures that might potentially be stabilized by ligamentous attachments as either stable (ligaments intact) or unstable (at least partial ligamentous disruption; see Fig. 31-3).

In unusual instances, fractures of the distal clavicle may be unstable in the absence of ligamentous injury. This occurs when both of the coracoclavicular ligaments remain attached to an inferior fracture fragment that lacks attachment to either of the primary medial and lateral fragments.[247]

Neer noted in his initial report that fractures of the distal clavicle are occasionally associated with extension into the acromioclavicular joint, and subsequently distinguished such fractures in his classification system as type III.[216–218] It has been suggested that some injuries diagnosed as type I acromioclavicular joint separation may in fact be intraarticular distal clavicular fractures, and that posttraumatic osteolysis of the distal clavicle[39,130,131,183,262] occurs in part as a result of an undetected intraarticular fracture.[57,217]

In children and adolescents, medial and lateral clavicular injuries most frequently take the form of physeal separation injuries, although metaphyseal fractures also occur.[234,235,303] In distal clavicular physeal injuries and metaphyseal fractures (so-called pseudodislocation of the acromioclavicular joint), the proximal fragment may displace and separate from the surrounding periosteum, while the thin distal clavicular epiphysis, with or without an attached metaphyseal fragment, retains its anatomic relationship to the acromion and the remainder of the shoulder.[90,146,183,184,235,254,304] The acromioclavicular and coracoclavicular ligaments are intact and remain attached to the periosteal sleeve.[234,235,303,309] Analogous patterns of injury occur at the sternal end of the growing clavicle.[36,143,176,258] Since both the medial and lateral physes typically remain open into the third decade of life,[133,234,299] it is important to realize that apparent acromioclavicular or sternoclavicular dislocations in some young adults may actually be physeal separation injuries.[36,54,143,176] As a result, these injuries can, in general, be expected to heal and become stable with nonoperative treatment. They may even remodel somewhat.

Fractures of the sternal end of the clavicle are uncommon and almost without exception treated symptomatically.[3,52,57,84,120,143,172,217,273,314] Craig has subdivided these as minimally displaced (type I), displaced (type II), intraarticular (type III), physeal separation (type IV), and comminuted (type V) fractures. Fractures in this region of the clavicle are so uncommon that the patterns of medial clavicular injury have rarely been described and studied, and it remains unclear how different fracture patterns might influence treatment or prognosis.

Fractures occurring between the medial limit of the coracoclavicular ligaments and the lateral limit of the costoclavicular ligaments represent by far the most common type of clavicular fracture. These fractures have not to this point been subclassified in a universally acceptable manner. Current publications use the Comprehensive Classification of Fractures (CCF) (Fig. 31-4)[47,141,274]: Type A fractures are simple transverse fractures, type B fractures are wedge fractures, and type C fractures are those in which the main fragments are separated by a zone of comminution and have no contact. This classification turns out to be very useful for the decision of which operative procedure should be carried out (plate vs. intramedullary nail). (See section titled Operative Treatment.)

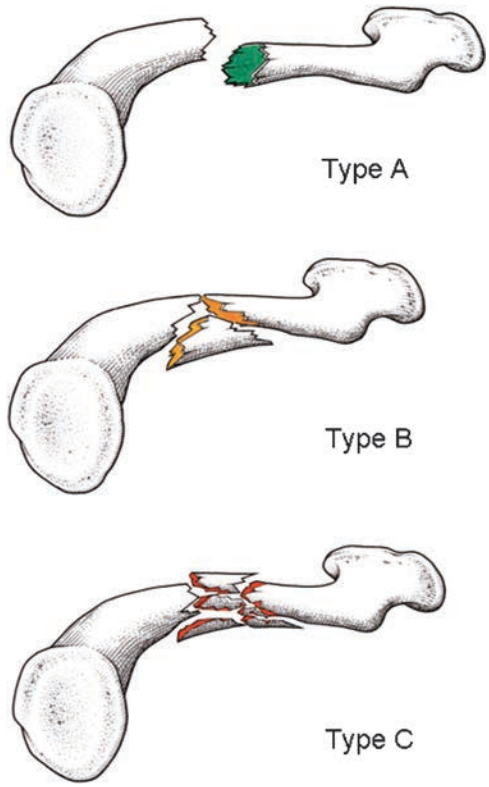


Figure 31-4 Type A fractures are simple transverse fractures, type B fractures are wedge fractures, and type C fractures are those in which the main fragments are separated by a zone of comminution and have no contact. (From Classification, Orthopaedic Trauma Association Committee for Coding and Classification. Fracture and dislocation compendium. *J Orthop Trauma* 1996; 10(5–9):1–154.)

One important element of a midclavicular fracture is the amount of displacement or deformity. Distinguishing *nondisplaced* or minimally displaced fractures (including greenstick and plastic bowing-type[28] fractures in children), which will heal with symptomatic treatment and little concern regarding either cosmesis or function, from *displaced fractures*, which may result in deformity, shoulder dysfunction, and an increased risk of nonunion, has been common practice up to now.[298] However, as seen in Fig. 31-5, the fracture displacement changes depending on body posture. In this particular x-ray, the difference is significant. Therefore, the criterion of fracture displacement is clearly not useful information regarding the choice between operative or conservative management.

Pathologic fractures of the clavicle occur, although they are unusual. Rowe reported fractures through eosinophilic granuloma, Pagetoid bone, and metastatic carcinoma involving the clavicle.[273] Fracture related to enchondroma[25] and arteriovenous malformation[208] have also been reported. Stress fractures of the clavicle have been described following radical neck dissection as a result of the devascularizing dissection and radiation osteitis that complicate the treatment of these tumors.[58,143,239,253,290] Fatigue fracture was also reported in a 12-year-old boy who, while attempting to improve his grades, had apparently been carrying an inordinately large number of books under the arm on the involved side for several months.[148] Stress fracture related to the use of a Dacron graft loop for coracoclavicular ligament reconstruction has also been reported.[70]

MECHANISM

In general, clavicle fractures suffered by adolescents and adults in all regions are the result of moderate or high-energy traumatic injury such as a fall from a height, motor vehicle accident, sport activity, or a blow to the point of the shoulder, and rarely a direct injury to the clavicle. Typical sports concerned include cycling, horseback riding, Alpine skiing, or motorcycling.[154–156,226–228,230,231,233] In children and the elderly, clavicle fractures usually occur following low-energy trauma.[3,52,119,136,137,139–141,228,229,231,273,277,284,287]

It has become clear that the clavicle fails most commonly in compression. Failure in compression is seen following falls onto the shoulder and direct blows to the

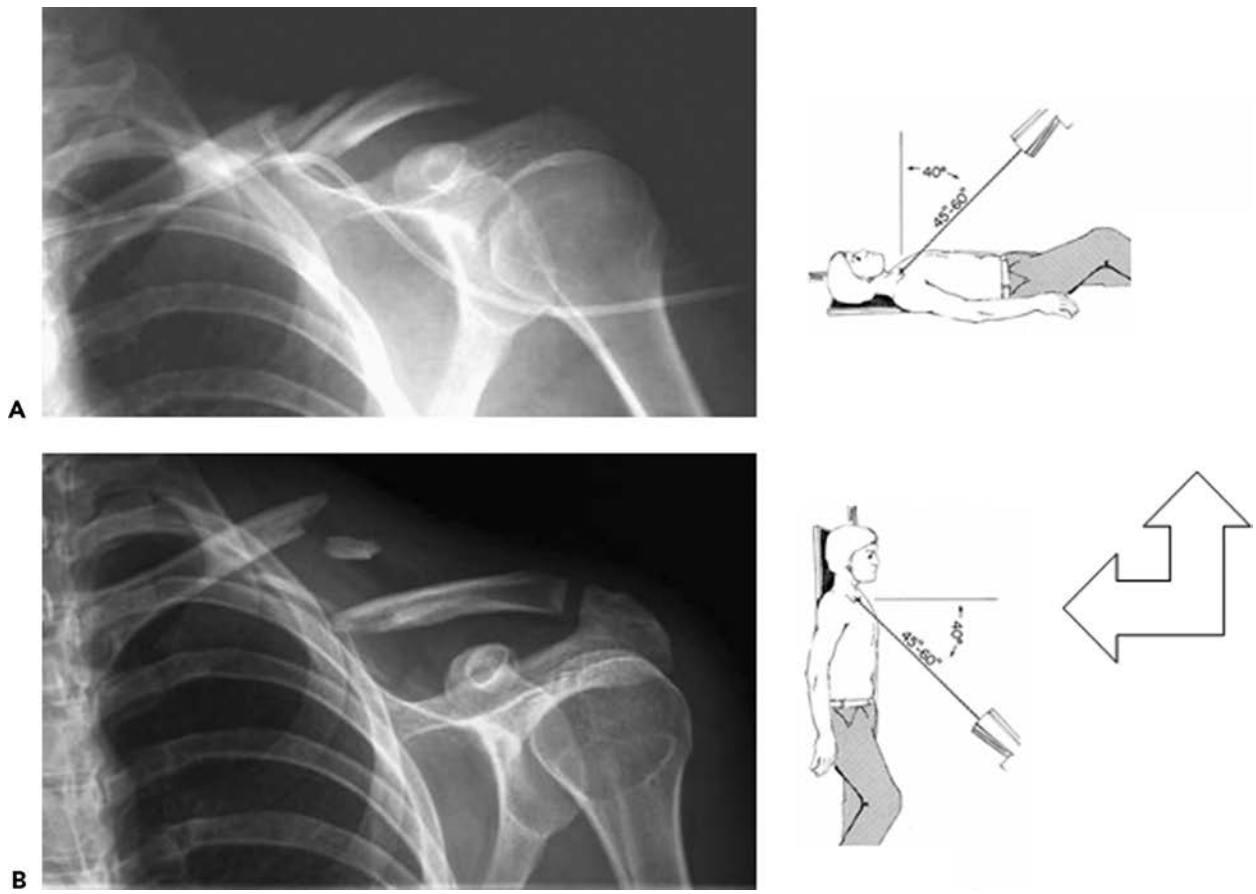


Figure 31-5 X-rays taken of a person with midshaft fracture in reclining **(A)** or standing **(B)** position. The position of the fracture elements changes dramatically depending on muscle tone.

point of the shoulder.[23,95,277,287] A direct blow to the clavicle, seen to occur in some stick-wielding sports such as lacrosse,[284] may also fracture the clavicle. Although a fall onto the outstretched hand has traditionally been considered a common mechanism of midclavicular fracture,[3] recent observations[287] bring this into question.

Stanley et al. studied 122 of 150 consecutive patients presenting to one of two separate hospitals in Sheffield with fractured clavicles. The detailed accounts of their injuries showed that 87% were the result of a fall onto the shoulder, 7% the result of a direct blow to the point of the shoulder, and 6% the result of a fall onto an outstretched hand.[287] Falls on the outstretched hand were the apparent mechanisms of 5 of 79 (6.3%) midclavicular fractures and 2 of 34 (5.9%) distal clavicular fractures, suggesting that direct injury to the shoulder is the most common mechanism of clavicular fracture at all sites. These authors hypothesized that even those patients who recall their injury as a fall onto the outstretched hand may have fallen secondarily onto the shoulder. This second impact may have been the injuring force, suggesting that isolated fall onto the outstretched hand is actually an unusual mechanism of injury.[287]

Neer has stated that distal clavicular fractures tend to be the result of a high-energy, direct blow to the shoulder.[119,216] However, distal clavicular fractures have also been identified in the elderly following lower energy injuries.[231]

EPIDEMIOLOGY

An understanding of the frequency and distribution of clavicular fractures is provided by data collected in Malmo, Sweden. Four percent of all fractures occurring in Malmo in 1987 involved the clavicle. This represented 35% of all fractures in the shoulder region. The overall incidence of clavicle fractures increased from 52 per 100,000 persons per year in 1952 to 64 per 100,000 persons per year in 1987, mostly as a result of an increase in sports-related injury and injuries following a fall.[231]

Seventy-six percent of the fractures occurred in the middle third of the clavicle, a figure that is similar to previous reports.[52,84,217,273] The average age overall in this subgroup was 21 years. However, the average age was 11 years for nondisplaced fractures, 25 years for simple displaced fractures, and 43 years for comminuted fractures.

Twenty-one percent of fractures in Malmo involved the distal clavicle with an average age of 47 years (median also 47 years). This is also comparable with the rate reported in some previous studies[52,58,172] but is double the rate reported in others.[79,119,217,218,273] The incidences of middle and lateral third fractures of the clavicle were comparable for middle-aged adults (approximately 35 to 60 years of age) in the Malmo experience.

Medial clavicular fractures represented only 3% of clavicular fractures.[231] Although many of the published studies report an incidence of 4% to 6%, even 3% is probably an overestimate based on inclusion of many of the more medial midclavicular fractures in this group.[79,119,217,218,273] Taylor measured the distance of 550 fractures from the lateral aspect of the clavicle and found only 0.5% in the medial third of the bone.[292,293] According to the data of Nordqvist et al., the average age of a person sustaining a medial clavicular fracture was 51 years, with a large proportion of fractures occurring in adolescent and young adult males and the elderly. The incidence of both lateral and medial clavicular fractures rose sharply after age 75, suggesting that these areas become substantially more susceptible to fracture when osteoporotic.[229]

EVALUATION

Clavicle fractures resulting from low- to moderate-energy traumatic injuries are easily diagnosed and are associated with few complications. The deformity and swelling associated with the fracture are usually apparent. The location of the fracture along the clavicle can usually be determined by close inspection and palpation, although distinction of fractures of the medial or lateral ends of the clavicle from dislocation of the adjacent joints can be difficult prior to radiographic examination. The patient typically resists all motion of the ipsilateral shoulder, is tender at the fracture site, and holds the arm against the trunk.

Open clavicular fractures are uncommon, even following high-energy traumatic injury, and are usually the result of a direct blow to the clavicle. Tenting of the skin by either one of the major fracture fragments or an intervening fragment of comminuted bone is not uncommon, but a true threat to the integrity of the skin is unusual.[263]

Neurovascular injury,[123] pneumothorax,[67,180,203,312] and hemothorax[167] have been reported in association with fracture of the clavicle, but are uncommon. In contrast to late dysfunction of the brachial plexus following clavicle fracture in which medial cord structures are typically involved, acute injury to the brachial plexus at the time of clavicle fracture usually takes the form of a traction injury to the upper cervical roots. Such root traction injuries usually occur in the setting of high-energy trauma and have a relatively poor prognosis.[17,169,282]

Vascular injuries may not always be apparent. These may consist of an intimal injury or a small puncture wound and can present from weeks to years later in the form of an aneurysm or pseudoaneurysm or thrombosis of the involved vein or artery.

The combination of clavicle fracture and fracture of the first five ribs in severely injured patients is an important indication of high-energy upper thorax trauma. It is not uncommon in such cases that accompanying injuries of

the mediastinal organs (i.e., aortic rupture, heart contusion, pericardial tamponade), the lungs (i.e., hemopneumothorax, lung contusions), and/or cervical and thoracic spine injuries occur.[6,7] The prevalence of pneumothorax in association with fracture of the clavicle is often quoted as being 3%, based on Rowe's study of over 600 fractures at Massachusetts General Hospital.[273] In that study, Rowe did not distinguish between moderate- and high-energy injuries and he did not distinguish isolated fractures from those injuries associated with ipsilateral scapular fracture or dissociation from the thorax, or ipsilateral upper rib injuries.[76,77,120,121,166,169,173,268,306] Again, the presence of these associated injuries indicates an extremely high-energy injury mechanism. Pneumothorax and hemothorax are more common in this situation and are likely to be a result of a more generalized chest wall injury rather than to a direct injury to the apical pleura by the fractured clavicle. Nonetheless, the importance of an evaluation for possible pneumothorax by both physical examination and close inspection of an upright film that includes the ipsilateral upper lung field should be emphasized.[56,185,312] When a clavicle fracture occurs in the setting of a high-energy traumatic injury (such as a motor vehicle accident or a fall from a height), evaluation of life-threatening injury takes precedence and should follow the protocol promoted by the American College of Surgeons.[5] Major vascular disruption can occur in association with fracture of the clavicle, but is extremely rare.[56,64,67–69,107,123,132,195,197,215,236,257,291] Injury to the thoracic duct has also been reported.[20,33] Death following a tear of the subclavian vein with resultant pseudoaneurysm was recorded in the famous case of the death of Sir Robert Peel.[66,164] Arterial thrombosis may occur following intimal injury.[128,168,301] Fracture of the clavicle or dislocation of either the sternoclavicular or acromioclavicular joint in association with lateral scapular translation represents a scapulothoracic dissociation, an injury often associated with severe neurovascular injury.[76,77,102,103,166,240]

Evaluation of the vascular status of the upper extremity should include an assessment of relative temperature and color as compared with the uninvolved extremity. Due to the extensive collateral blood supply to the upper extremity, these factors may appear normal in spite of the presence of a major vascular injury. A difference in peripheral pulses or blood pressure between injured and uninvolved upper extremities may be the only clue that a vascular injury is present. When the limb is threatened or there is persistent unexplained hemorrhage, angiography can help to detect and localize any vascular injury, thereby assisting with definitive management.

Compression[61,83,96,97,106,265] and even thrombosis[177,278,288] of the subclavian vein can occur in the early postinjury period. Pulmonary embolism has been reported in the setting of subclavian vein thrombosis following clavicular fracture.[278,313]

RADIOGRAPHIC EVALUATION

An anteroposterior view in the coronal plane of the clavicle will identify and localize the majority of clavicular fractures. To further gauge the degree and direction of displacement of clavicular fractures, oblique views of the clavicle will be necessary.[260,273] The film should be large enough to evaluate both the acromioclavicular and sternoclavicular joints as well as the remainder of the shoulder girdle and the upper lung fields. Quesada recommended 45-degree caudad and cephalad views, which he felt would facilitate evaluation by providing orthogonal views.[260] Medial clavicular fractures may be difficult to characterize on this view, and computed tomography is often necessary.

Evaluation of distal clavicular fracture displacement in the anteroposterior plane requires a different set of radiographs because cephalad and caudad tilted views are hindered by overlap of the bones of the shoulder, overexposure of the distal clavicle, and frequent failure to accurately depict the degree of displacement. Neer has suggested a stress view (with 10 pounds of weight in each hand) to evaluate the integrity of the coracoclavicular ligaments and 45-degree anterior and posterior oblique views to gauge displacement.[219] However, as we noted, differentiation between displaced and undisplaced fractures may mot be relevant. The position of the fracture elements changes dramatically depending on muscle tone (x-rays taken in standing or reclining positions) and the position of the arm (arm in a sling or free-hanging) (see Fig. 31-5). Instead the shape and number of fragments should be identified.

A radiograph taken with the x-ray source angled with a combination of both anteroposterior and cephalad–caudad obliquity has recently been advocated in the evaluation of midclavicular fractures.[266,305] The so-called apical oblique view (45-degree anterior [plane of the scapula], 20-degree cephalad tilt) may facilitate the diagnosis of minimally displaced fractures (e.g., birth fractures and fractures in children).[305] Ultrasound is also a very sensitive diagnostic tool in the evaluation of birth fractures (Fig. 31-6).[145]

The abduction–lordotic view, taken with the shoulder abducted above 135 degrees and the central ray angled 25-degrees cephalad, proves useful in evaluating the clavicle following internal fixation.[266] The abduction of the shoulder results in rotation of the clavicle on its longitudinal axis, causing the plate to rotate superiorly and thereby exposing the shaft of the clavicle and the fracture site under the plate (Fig. 31-7).

MANAGEMENT OF SPECIFIC INJURIES

Birth Fractures

Fracture of the clavicle is the most common traumatic birth injury. Clavicle fracture must be distinguished from less

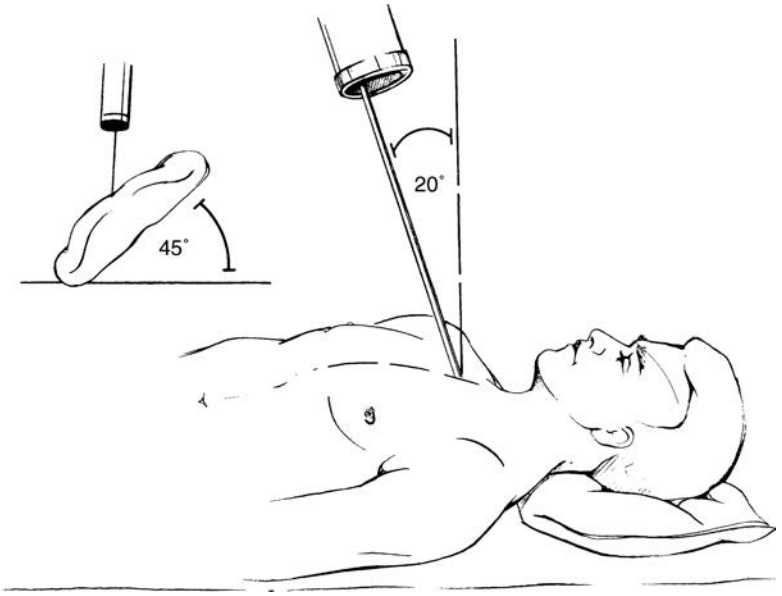


Figure 31-6 The apical oblique view is taken with the involved shoulder angled 45 degrees toward the x-ray source and the x-ray source angled 20 degrees cephalad.

common birth injuries including brachial plexus palsy, fracture–separation of the proximal humeral epiphysis, and fracture of the humeral shaft.[238] High birth weight and shoulder dystocia are clearly associated with an increased risk of all types of traumatic birth injury.[37,45,49,103,104,135,174,238] However, upwards of 75% of neonates with clavicle fractures are the product of a normal labor and delivery and weigh less than 4,000 grams.[42,45,135]

Shoulder dystocia results in a wide separation of the head and shoulder as passage of the shoulder through the birth canal is blocked by the symphysis pubis. This may result in a traction injury to the upper roots of the brachial plexus (Erb's palsy). Fracture of the clavicle may actually protect the brachial plexus by allowing passage of the fetus and decreasing tension on the upper roots.[238] Identification of a clavicle fracture in a neonate that is not moving an upper extremity can be reassuring in that the fracture may explain the findings and generally will heal without long-term sequelae. However, between 1.8% and 5.3%[174,238] of clavicle fractures will have associated brachial plexus injury, and approximately 13% of patients with birth injury to the brachial plexus also have clavicle fractures.[104] Concurrent clavicular fracture apparently does not alter the prognosis of the brachial plexus injury.[4]

The incidence of birth fracture of the clavicle is uncertain as many fractures are asymptomatic and may not be detected. Prospective investigations of consecutive births using either radiography or serial physical examinations have detected birth fracture of the clavicle at an incidence of 1.7% and 2.9%,[135] respectively. The prospective examination of neonates demonstrated that many fractures are not detectable until callus begins to form 1 to 2 weeks following the birth injury. The traditional signs of birth fracture of the clavicle—instability, motion, or crepitus at the fracture site; significant local swelling; and asymmetric Moro reflex—are commonly absent.[135]

Most birth fractures of the clavicle occur during a vertex delivery, although they may also occur with a breech presentation or cesarean section.[238] The anterior shoulder is more commonly involved in most studies. However, in some reviews, the posterior shoulder was more commonly involved [238] and bilateral fracture may also occur. This suggests that both clavicles are subject to compressive forces during delivery as the widest part of the neonate (the shoulders) passes through the birth canal and that either clavicle may fracture, occasionally both.[238]

The level of experience of the obstetrician has been implicated in some studies[49] and found to be unrelated in others.[37] Considering the difficulty making the diagnosis, the benign nature of the injury, and the lack of a clear association with level of experience, the incidence of birth fracture of the clavicle is probably a poor indicator of quality of obstetric care, although it has been used as such.[49,174] It is difficult to assess the risk of forceps delivery because it is now infrequently used and may have simply been associated with more difficult deliveries in prior investigations.[49,174] Most studies demonstrate normal Apgar scores in neonates with birth fracture of the clavicle, indicating that this injury is not associated with postnatal difficulties. On the other hand, at least one study suggests that intrapartum fetal distress may be associated with an increased risk of clavicle fracture as the second stage of labor is iatrogenically shortened by a concerned obstetrician.

Despite a great deal of investigative effort, no reliable risk factors have been determined that distinguish neonates at high risk of birth fracture of the clavicle, and no management recommendations can be made. Recent studies concur that birth fracture of the clavicle may be an

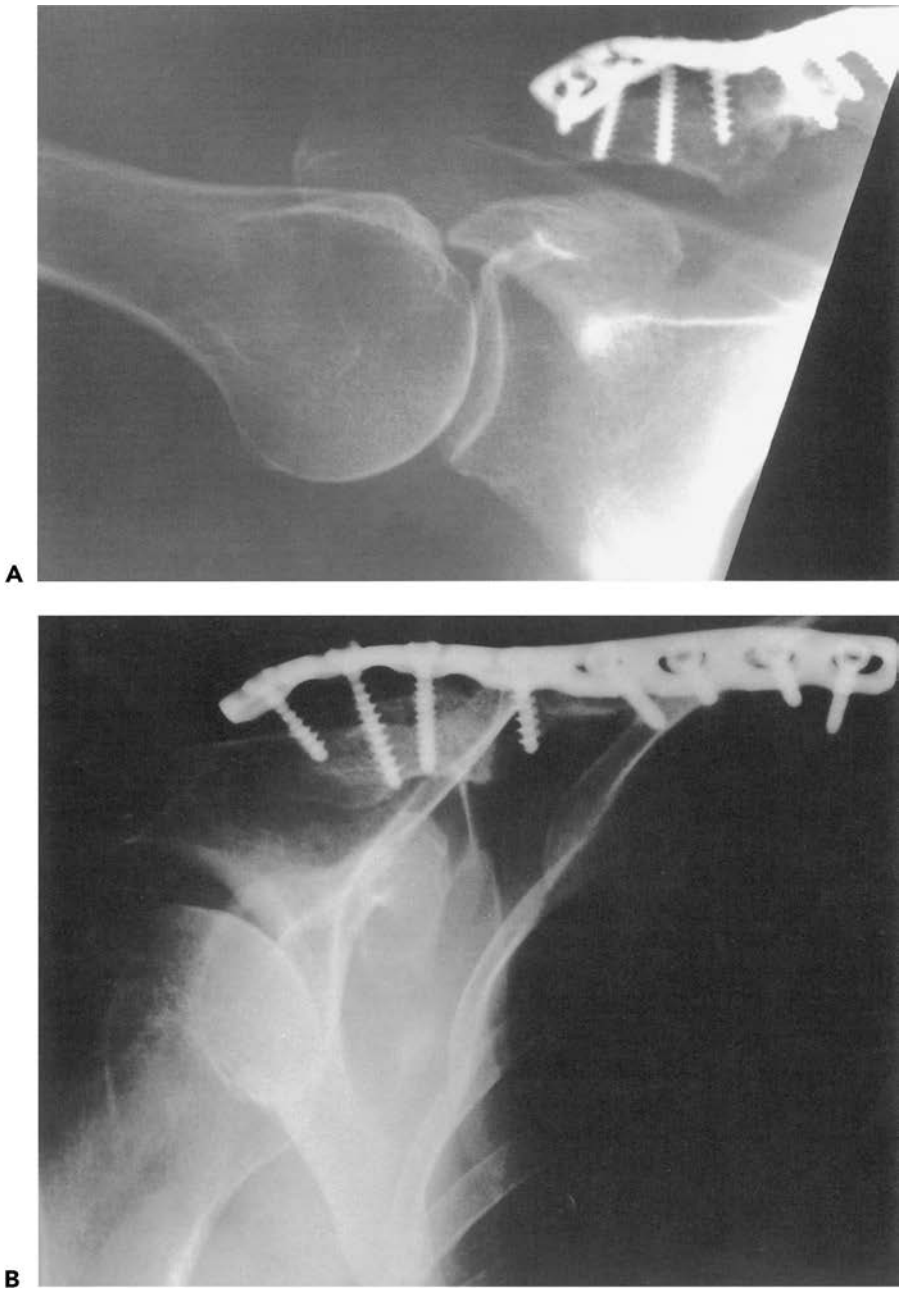


Figure 31-7 The abduction lordotic view **(A)** takes advantage of the rotational motion of the clavicle with abduction of the shoulder to provide an alternative view of the clavicle. This radiographic projection is useful for visualizing the fracture site under the plate, which is often obscured in the routine anteroposterior view **(B).** (From Browner B.D, Jupiter JG, Levine AM, Trafton PG, eds. *Skeletal trauma: fractures, dislocations, ligamentous injuries,* 2nd ed. Philadelphia: WB Saunders, 1998.)

unavoidable, and fortunately inconsequential, complication of normal birth.[45]

Isolated birth fractures of the clavicle heal quickly, remodeling any deformity. Immobilization is probably unnecessary but is commonly used for a brief period to reassure the parents. Careful handling of the infant is the most important measure for reducing discomfort and irritability. If treatment is instituted, a simple, safe method of holding the arm at the side, such as strapping the arm to the trunk with stockinet, a gauze roll, or an elastic bandage with the elbow flexed to 90 degrees and a cotton pad between the arm and trunk, should be used for about a week, after which spontaneous movement should return to the arm indicating that interval healing has increased the stability and decreased the pain associated with the fracture.

Midclavicular Fractures

Nonoperative Treatment

Surviving writings from ancient Greece and Egypt document that for over 5,000 years humans have been concerned primarily with the deformity rather than healing of fractures of the clavicle. In fact, the method of closed reduction of clavicle fractures described in the Edwin Smith papyrus differs little from methods used today.[2,32]

Since that time more than 200 bandaging/strapping techniques have been described.[10,160]

To effect a closed reduction, in most cases the distal fragment must be brought upward, outward, and backward while the medial fragment is depressed. A hematoma block (10 mL of 1% lidocaine injected into the fracture site) can provide adequate anesthesia, but in some cases conscious sedation or general anesthesia may prove necessary. The reduction technique described in the Edwin Smith papyrus,[32] and still commonly used,[261] involves placing a pillow between the shoulder blades of the recumbent patient while the shoulders are spread outward and upward.[32,53,261] Another method for achieving reduction is to bring the shoulders backwards and upwards with the patient in a sitting position while the physician's knee or clenched fist is placed between the shoulder blades to control the position of the trunk.[245,300]

Innumerable devices have been devised in an attempt to effect or maintain closed reduction and thereby minimize the deformity associated with fracture of the clavicle. The majority of these were cumbersome, painful, and even dangerous.[224] Dupuytren in 1839 and Malgaigne in 1859 argued that despite these valiant attempts, deformity of the clavicle was inevitable.[68,69,186] They emphasized the use of the simplest and most comfortable method of treatment, which for Dupuytren consisted of placing the arm on a pillow until healing occurred. It has long been suggested that excellent function can be expected despite residual deformity.[94,95,167,212,245,277] Nonetheless, devices intended for the maintenance of reduction and immobilization of clavicle fractures have remained popular and commonly take the form of either the figure-eight bandage with or without a sling and only on rare occasions a figure-eight plaster (Billington yoke[26]) or a half-shoulder spica cast.[3,245,261,273] Others have followed Dupuytren and Malgaigne in arguing that accurate reduction and immobilization of clavicular fractures is, as stated by Mullick, "neither essential nor possible."[77,78,132,172,212,277] These authors advocate the use of a simple sling for comfort, forgoing any attempts at reduction.

The advantage of the figure-eight bandage is that the arm remains free and can be used to a limited degree. Disadvantages include increased discomfort, the need for frequent readjustment and repeat office visits, and a potential for complications including axillary pressure sores and other skin problems, upper-extremity edema, venous congestion brachial plexus palsy worsening of deformity, and perhaps an increased risk of nonunion.[9,78,94,200,216,255,276,308]

Few investigations have compared treatment with a figure-eight (or reducing) bandage with the use of a simple sling (or supporting bandage).[200] While the details of patient selection and evaluation in these investigations remain unclear based on the data published, these authors found no difference with regard to shoulder function, residual deformity, or time to return to full range of motion and full activity.[9,10,172,200,287]

It is important to emphasize that although the clavicle is one of the most commonly fractured bones, very little in the way of stringent, detailed analysis of clavicular fracture data has been performed. The existing literature regarding nonoperative treatment consists of relatively few series,[52,88,172,277,294] relatively limited studies comparing treatment modalities,[9,200,287] and some technique descriptions,[26,53,115,172,245,300] anecdotal observations,[85,94,224,261] and general reviews.[3,120,121,220–273,257] That conservative management is not always uncomplicated has been reported already by many authors in the past.

Rowe pointed out that injury-related complaints in adults such as pain and injury-caused impediments in the first 3 weeks postinjury are frequently underestimated.[273] On different x-ray series, Mullick showed that the goal of reduction and immobilization in figure-eight bandage was virtually never reached, and that in a few cases displacement actually increased.[212] Petracic et al. proved that with increased tension on the figure-eight sling, venous congestion occurs even before reduction of the fracture.[252] He calls this form of therapy a "symbolic treatment, performed in order to satisfy the patient's need for a decorative bandage." Fowler pointed out in 1968 that figure-eight slings can lead to more problems than the fracture itself due to chafing and pressure in the axilla.[94] In 1982, Piterman reported a painful excoriated axillary wound caused by an overzealously applied figure-eight bandage.[255]

The rates of nonunion with conservative therapy are quoted in the literature with marked variation from 0.3% to 15%.[122,219,273,307] Neer and Rowe reported nonunion rates of under 1% with conservative management of clavicle fractures, however, without differentiating the evaluated patient cohort regarding age and fracture location. Eskola reported a 3% nonunion rate in a predominantly adult population.[85] White et al. reported a 13% nonunion rate and found a correlation with high-energy trauma.[307] Hill et al. assessed nonunion in 15% of his patients and reported a significant correlation with an initial shortening of over 2 cm.[122] Thirty-one percent of their investigated patients were dissatisfied with the outcome of their treatment. In a study of 157 patients, Matis et al. found that half of the cases with clavicular shortening of 1 cm and all cases with clavicular shortening of 2 cm exhibited a significant worsening of shoulder function.[194] A randomized, controlled study investigating whether figure-eight slings really affect healing and function of postclavicular fracture has not yet been completed.

Few reports have attempted to evaluate the relationship between residual deformity and shoulder function.[237] Eskola et al.[85] invited all 118 patients treated for fracture of the clavicle at Helsinki University Central Hospital in 1982 to return for evaluation 2 years following the injury. Among the 89 who presented for the follow-up examination, 24 (27%) had either slight pain on exercise or restricted shoulder movement, with four identified as having

major functional problems. Primary fracture displacement and shortening of the clavicle as compared radiographically with the opposite, uninvolved side at the 2 year follow-up were used as measures of deformity. Among the 15 patients with primary fracture displacement greater than 15 mm at the time of injury, eight (53%) had pain with exercise, whereas only 12 of the remaining 74 patients had pain (16%), a difference found to be statistically significant using chi-squared analysis ($p = 0.02$). Among 47 patients with demonstrable shortening at the final follow-up, 17 (36%) had pain with exercise as compared with 3 of 42 patients (7%) without shortening ($p = 0.02$ by chi-square analysis). Based on these findings, they recommend reduction of deformity associated with displaced clavicular fractures, particularly with regard to shortening. One might question the authors' conclusions based on the simple fact that the more displaced fractures were those associated with higher-energy traumatic injury and might be expected to do worse regardless of residual deformity. The question of which clavicular fractures can be expected to do poorly and why remains uncertain. While the fact that 27% of their patients returning for 2 year follow-up reported problems with pain during exercise or restricted shoulder movement suggests that there may be room for improvement in the treatment of clavicle fractures, we must await more focused and rigidly controlled investigations for data that might alter our approach to treatment (Fig. 31-8).

Operative Treatment

With the development of more rigid implants, there has been some interest in the use of primary operative treatment for clavicle fractures.[149,179,238,242,256,289,315] The good results with open reduction, internal fixation, and bone grafting of clavicular nonunion that have been documented in recent reports also support the contention that internal fixation of the clavicle, when performed properly, should not impede healing.[28,142,188] A number of authors have reported good results using plate fixation of clavicular fractures for open fractures, for fractures with severe angulation that could not be reduced closed, or in multiple traumatic injury, especially in the setting of ipsilateral upper-extremity trauma or bilateral clavicular fracture.[35,149,182,243,289] Kloen et al.[152] favored anteroinferior plating of the clavicle, but biomechanical studies revealed that plates fixed at the superior aspect of the clavicle exhibit significantly greater stability than those on the anterior aspect.[125] Whether the new locking compression plates provide advantages due to biomechanical stability is not yet clear. In particular, scapulothoracic dissociation and the so-called floating shoulder, representing a combination of displaced clavicular and glenoid neck fractures, are felt to be important indications for open reduction and plate and screw fixation of the clavicular fracture.

Khan and Lucas noted no nonunions among 19 patients treated with primary plate fixation.[149] Schwartz and Hocker[289] used 2.7-mm plates and reported nonunion in 3 of 36 patients, which they attributed to using a plate of inadequate length. Poigenfürst et al.'s extensive experience with plate fixation of fresh clavicular fractures identified nonunions in 5 of 122 patients (4.1%) treated operatively.[256] They also related these failures to technical errors including the use of a plate of inadequate length or strength or devitalization of fracture fragments during operative exposure.

External fixation has also been used for fixation of the clavicle. In a study by Schuind et. al.,[280] good results were obtained in 15 fresh midclavicular fractures and in five delayed unions. However, considering the rarity of severe soft tissue injury in this area, the role of external fixation remains unclear.[59]

Within the past few years several publications have described poor outcomes after conservative treatment of severely displaced midclavicular fractures. It has been reported that between 10% and 30% of patients had clinically, radiographically, and subjectively unsatisfactory results because of shoulder shortening,[18,75] nonunion,[49] or impaired function.[49] However, surgical procedures have also been associated with poor cosmetic results and higher incidences of nonunion and refracture when compared with conservative treatment. Therefore, surgery should be done only in specific situations.[16]

The standard treatment in surgical therapy of midclavicular fractures is plate fixation. To avoid breakage of the implant, the plate that is used must be relatively large compared with the bone size. A large incision causing additional soft tissue damage is necessary to position this implant, most commonly a small dynamic compression plate or a small reconstruction plate. Typical complications of plate fixation are infection, hypertrophic scars, implant loosening, nonunion, and refracture after hardware removal.[139,149]

From a biomechanical point of view, intramedullary positioning of the implant is ideal.[259] The diameter of the titanium (Ti) nail Jubel et al.[149] used for intramedullary fixation was small; however, no implant displacement or material breakage occurred. Because of anatomic features of the clavicle, devices for intramedullary fixation need to be flexible. Besides the need for flexibility, the implant needs to be stable enough to neutralize the potential disruptive forces acting on the fracture after stabilization. The implant also has to be small enough to enable its passage through the medullary space, which is narrow, especially in the middle third of the clavicle.[8]

Several authors have described various modifications of intramedullary stabilization.[22,23,29,30,34,38,39] Jubel et al.[136,141] performed a study using elastic stable intramedullary fixation for clavicle fractures (Fig. 31-9). In this study, patients showed significant improvement of shoulder function and

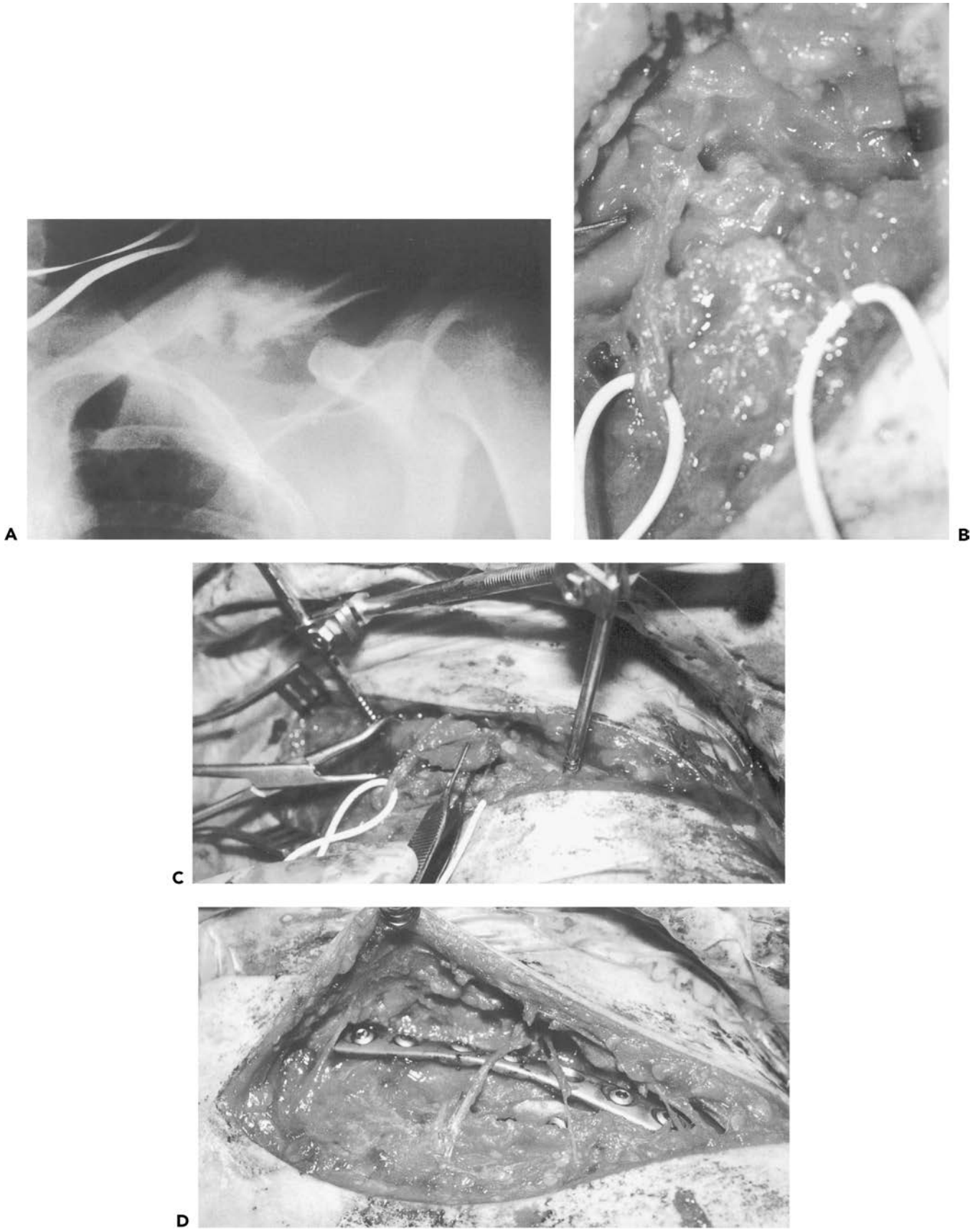
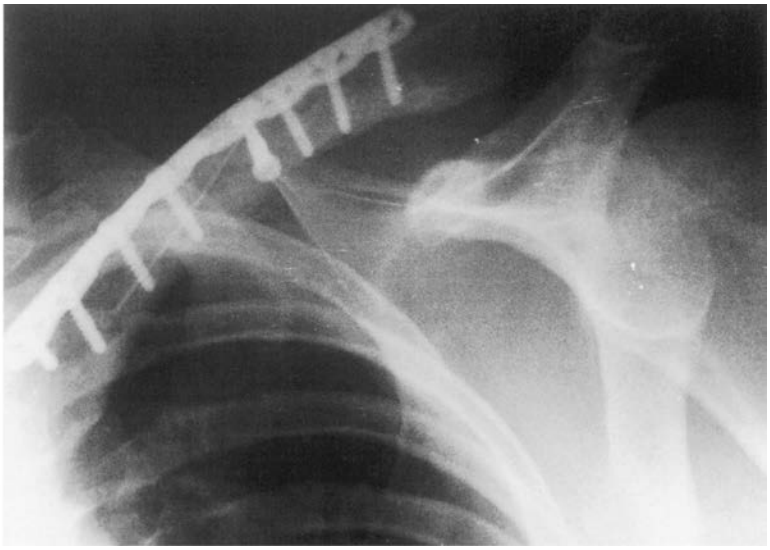


Figure 31-8 A 30-year-old male presented with an ununited fracture of the clavicle associated with excessive callus formation. He had complaints of numbness and weakness in the ipsilateral upper extremity. **(A)** Anteroposterior radiograph demonstrates the nonunion and hypertrophic callous formation. **(B)** The supraclavicular nerves are preserved during exposure of the clavicle. **(C)** A distractor was used to restore clavicular length and alignment. A sculptured tricortical iliac crest graft is placed into the resulting bony defect. **(D)** The clavicle is then stabilized with a 3.5-mm limited contact dynamic compression plate.



E

Figure 31-8 (*continued*) **(E)** Postoperative radiograph demonstrates restoration of length and alignment of the clavicle and stable plate fixation. (From Browner, Jupiter, Levine, Trafton, eds. *Skeletal trauma: fractures, dislocations, ligamentous injuries*, 2nd ed. Philadelphia: WB Saunders, 1998.)

reduction of pain on day 3 postoperatively, compared to the day prior to surgery. Even patients with bilateral fractures were able to do daily activities without assistance immediately after surgery.

Patients with additional injuries to the lower extremity were able to walk with crutches within the first week after surgery, enabling early mobilization. The original length of the clavicle was restored in all patients but one. Objectively and subjectively, good cosmetic results were achieved using this technique. All patients but one had good biologic fracture healing, so there was no refracture. Because of reduction and stabilization, excessive callus formation, typically seen after conservative treatment, was prevented and the shape of the long bone was restored. The incidence of nonunion was lower compared with the nonunion rate of similar displaced fractures after conservative treatment, plate fixation, or other intramedullary techniques.[17,32,37] These results showed that intramedullary fixation of displaced midclavicular fractures with a flexible Ti nail is a safe, minimally invasive surgical technique, producing excellent functional and cosmetic results compared with plate fixation or conservative treatment. Intramedullary nailing of displaced midclavicular fractures is an alternative treatment to conservative procedures or plate fixation in patients with markedly displaced midclavicular fractures, multiple trauma, fractures of the lower extremities, or associated shoulder girdle injuries.

Author's Preferred Treatment

Nondisplaced and minimally displaced fractures of the midclavicle require little more than symptomatic treatment. This is best achieved with a simple sling, which can be supplemented by a swathe component if necessary for added comfort early in postinjury. The majority of these nondisplaced fractures will be encountered in children who will heal quickly, and although they may not be compliant with sling wear, they usually self-regulate their activity level until healing has progressed and merely require gentler handling during the healing period. The clavicle typically heals sufficiently to discontinue immobilization within 3 to 4 weeks in young children, 4 to 6 weeks in older children, and 6 to 8 weeks in adults. Limitation of activity is usually encouraged for a minimum of 8 weeks following clinical and radiographic union to reduce the risk of refracture.

The optimal treatment of displaced and comminuted fractures of the clavicle is disputed. Wide displacement and soft tissue interposition have been implicated as risk factors for the development of nonunion,[128,142] and at least one study has suggested that residual deformity may alter function.[88] Open reduction and internal fixation of so-called irreducible fractures of the clavicle has been reported with good results.[35,144,149,242,289,315] Our opinion is that displacement should no longer be used as the most important decision criterion for or against surgery. It is an invalid parameter and very difficult radiographically to objectively report (see Diagnosis). The patients themselves should be more involved in making the decision, and should make that decision together with the physician after detailed description of the advantages (pain reduction, immediate sling-free exercise stability) and risks of operative treatment are explained. The physical and vocational activities of the patient must also be considered for this decision.

However, further work is needed to determine the following: (a) What is the percentage of displaced midclavicular fractures that will go on to nonunion, and is it high enough to consider routine operative treatment? and (b) Does the potential loss of function related to persistent deformity justify the risks of routine operative intervention?

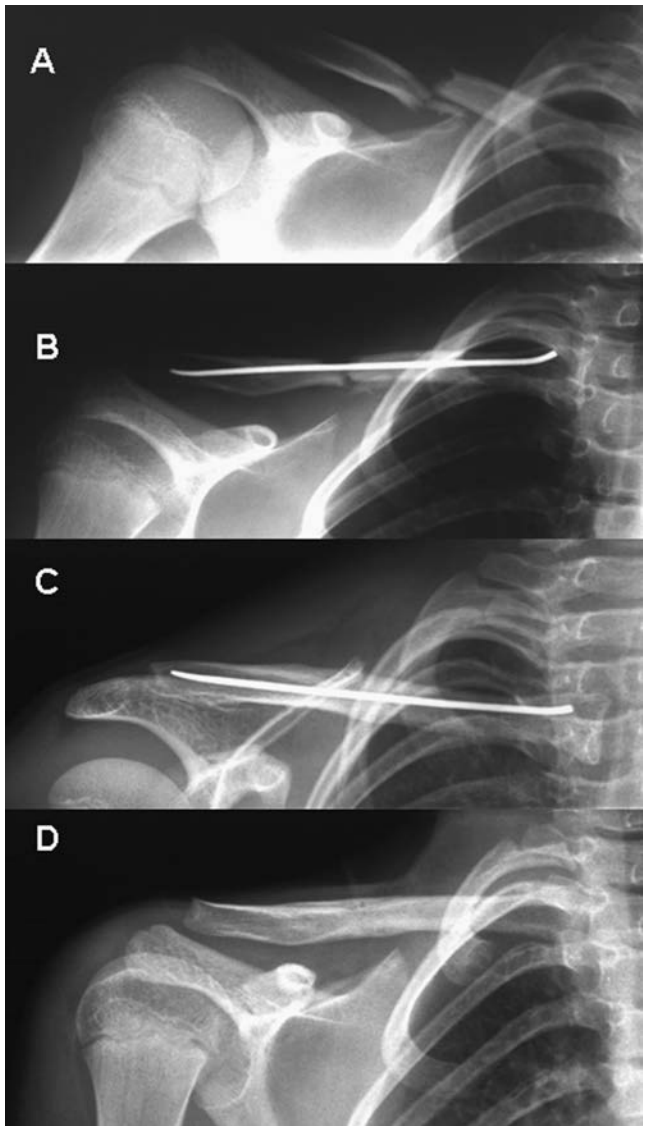


Figure 31-9 A professional 13-year-old motocross driver had a fall during a contest: **(A)** preoperative x-ray; **(B)** postoperative x-ray; **(C)** x-ray 6 weeks after the operation; and **(D)** x-ray after hardware removal. One week after the operation, he resumed the training on his motocross. In the second postoperative week, he participated again in a contest and, in the third week, became German Champion in his division. (Reproduced with permission from the BMJ Publishing Group: *Br J Sports Med* 2003;37(6):480–483.)

The definitive indications for primary surgical intervention that we would consider are uncommon and include open fracture, scapulothoracic dissociation,[76,77,166] so-called floating shoulder injuries,[102,120,121,268] and associated major vascular injury in which an open approach will be necessary for vascular repair. While isolated fractures of the sternal end or middle third of the clavicle do well with nonoperative treatment, complex clavicular injuries involving dislocation or epiphyseal separation at one end of the clavicle in combination with a fracture of the middle third would probably benefit from open reduction and internal fixation.[81,112,165,170,297,311] Likewise, the rehabilitation of associated ipsilateral upper-extremity trauma may be facilitated by operative fixation of the clavicle. In the absence of these indications, nonoperative treatment remains our preference.

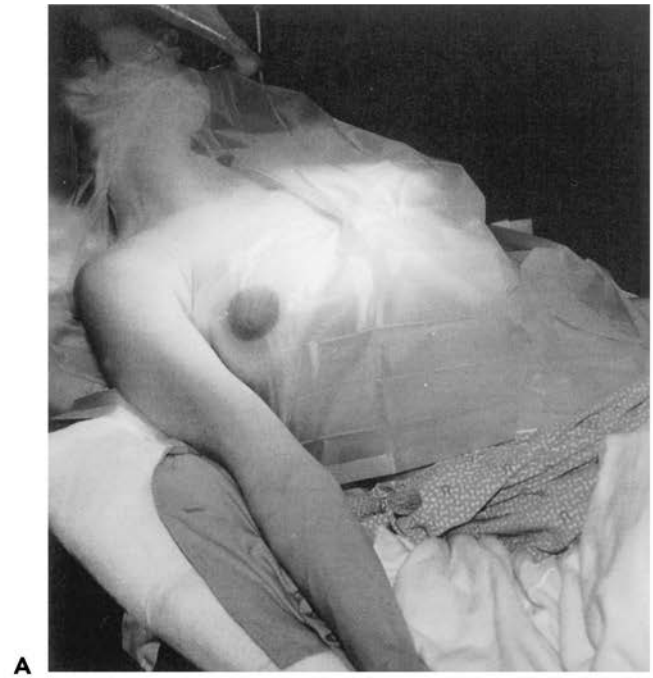
It is often stated that when the skin is threatened by pressure from a prominent clavicular fracture fragment, then closed reduction and internal fixation should be considered. Actually, it is extremely rare that the skin will be perforated from within.[263] However, in the head-injured patient, operative stabilization may be required.

The merits of open or closed reduction and operative fixation in the setting of neurovascular compromise are also unclear. Certainly, when an open approach for vascular repair is required, internal fixation of the clavicle should be performed, but fortunately acute neurovascular injury is rare in association with clavicular fracture.[96] The most frequent vascular disturbance encountered is venous congestion of the arm, which, in the absence of deep venous thrombosis, aneurysm, or pseudoaneurysm, can be treated expectantly.

Acute injury to the brachial plexus is extremely uncommon following fracture of the clavicle, and when present, is more likely to be the result of a traction injury to the upper roots of the brachial plexus. What may prove to be an indication for operative intervention is a plexopathy, which develops in relation to abundant callus in a malaligned fracture presenting at a time remote from the injury. In these instances, open realignment, reduction of callus bulk, and internal fixation of the fracture should be considered (Fig. 31-10).[302]

When performing closed or open reduction and internal fixation of the clavicle, we prefer minimally invasive intramedullary nailing with a 2.0- to 3.5-mm titanium nail (Prevot nail) for closed clavicular fractures (CCF) type A and B, and fixation with plate and screws for CCF type C fractures. Given the fact that intramedullary fixation of the clavicle is technically difficult owing to the curvature, high density, and small intramedullary canal of the bone, it is remarkable that good results with this technique have been observed.[28,41,136,137,139–141]

Our technique for internal intramedullary fixation of the clavicle is as follows: Patients are positioned on a radiolucent operating table in the supine position. A skin incision of 1 to 2 cm is made just above the sternal end of the clavicle. Approximately 1 cm distal to the sternoclavicular joint, a hole is drilled into the ventral cortex of the medial end of the clavicle with a 2.5-mm drill and widened using an awl. The Ti nail, 2.0 to 3.5 mm, is fixed in a universal chuck with a T-handle. With oscillating movements of the surgeon's hand the unreamed Ti nail is advanced until it reaches the fracture site. To ensure correct placement of the nail, fluoroscopic control is used for reduction and insertion of the nail into the lateral fragment. If closed reduction fails, an accessory incision of 3 to 4 cm is made above



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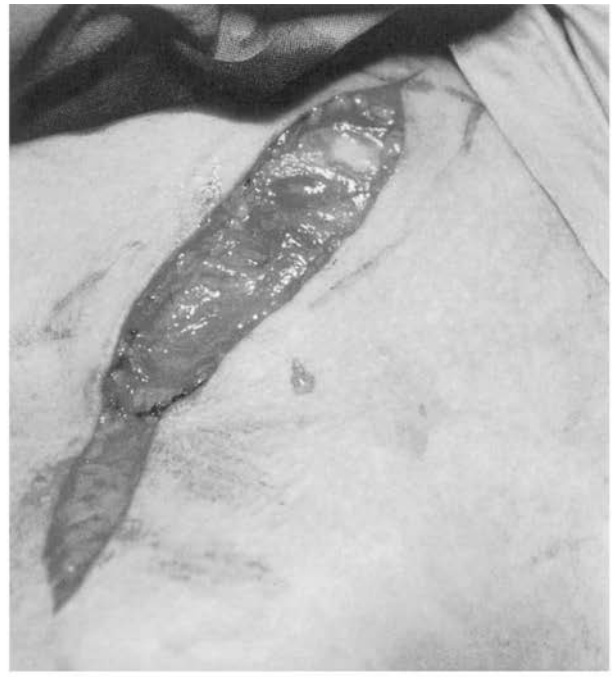

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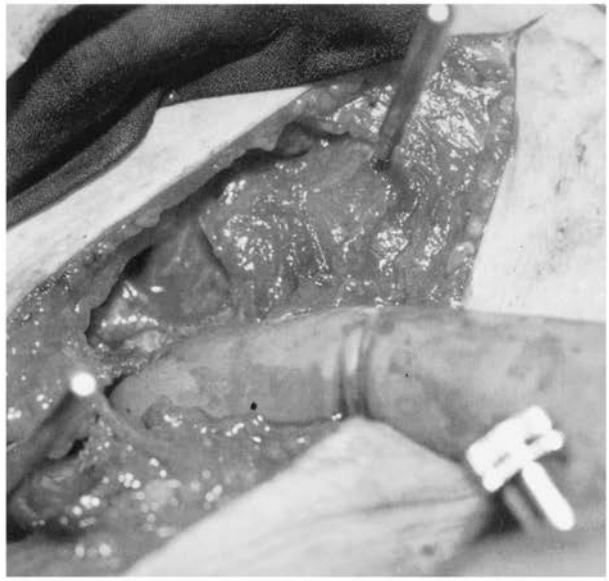

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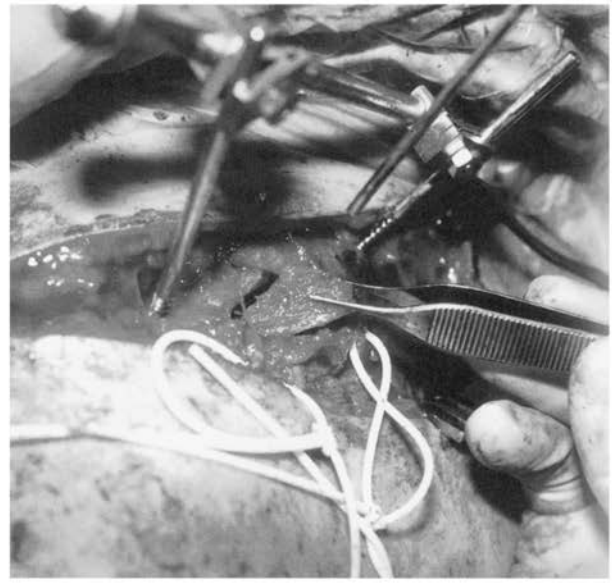

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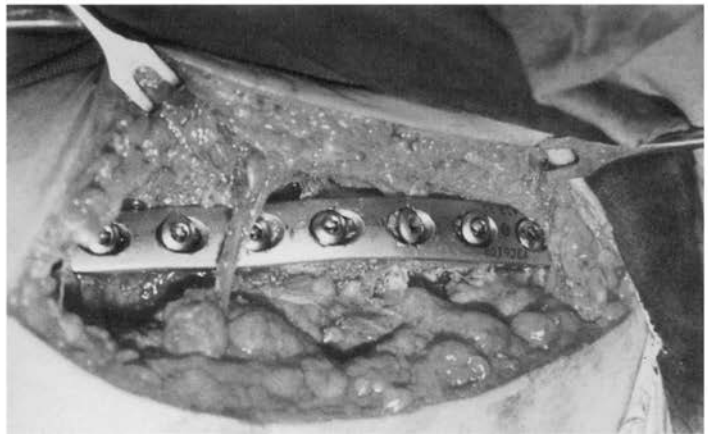
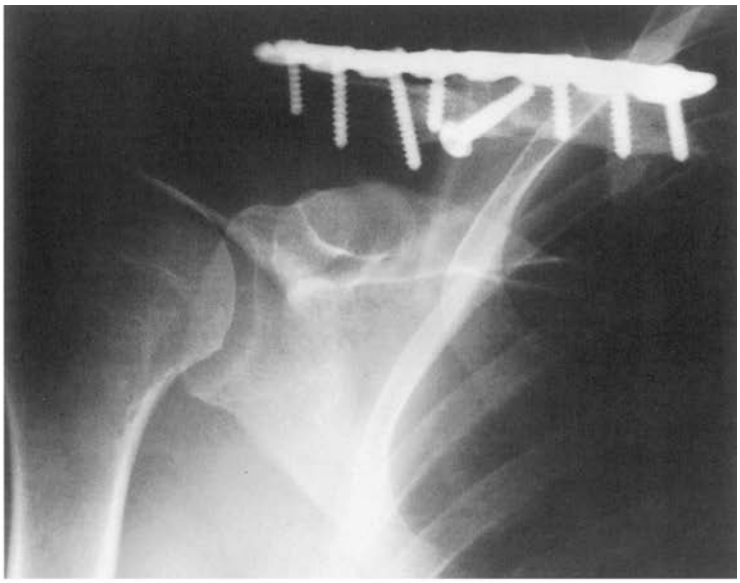


E

Figure 31-10 Operative technique for plate fixation of the clavicle. **(A)** The patient is positioned in the beach-chair position. **(B)** Incision of the skin is chosen to respect the relaxed skin tension lines. **(C)** The supraclavicular nerves are identified under loupe magnification and protected. Schantz screws are placed medial and lateral to the fracture site. **(D)** A small distractor is used to restore the length and alignment of the clavicle. When comminution results in a bony defect after anatomic reduction, an autogenous iliac crest autograft is applied. **(E)** The clavicle is stabilized using a 3.5-mm limited contact dynamic compression plate.



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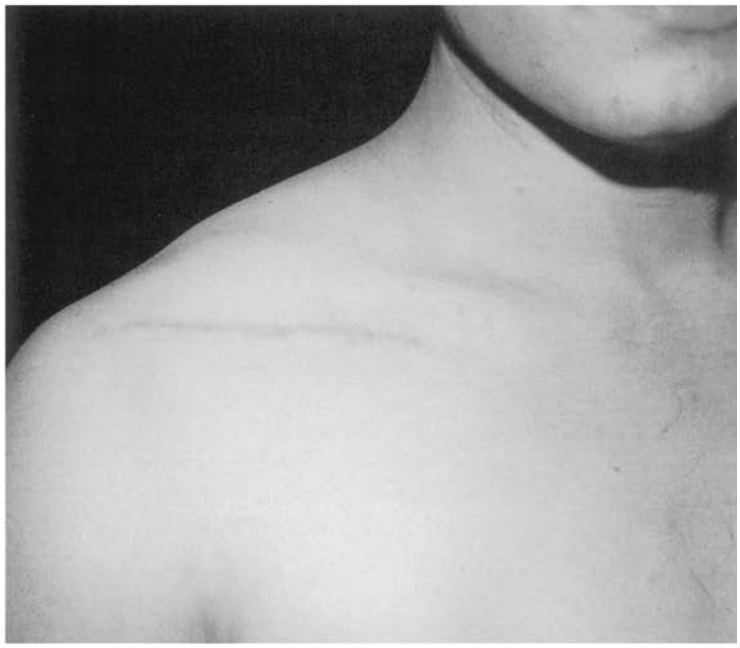


G

Figure 31-10 (*continued*) **(F)** When the fracture pattern allows, an interfragmentary lag screw is used to obtain compression between the fracture fragments. **(G)** Hypertrophy of the scar is uncommon. (From Browner, Jupiter, Levine, Trafton, eds. *Skeletal trauma: fractures, dislocations, ligamentous injuries,* 2nd ed. Philadelphia: WB Saunders, 1998.)

the fracture site to enable direct manipulation of the fragments. Additional fragments are maintained in their positions, maintaining soft tissue connections. The protruding end of the nail is cut off at the site of its insertion.

For postoperative treatment, patients are instructed to mobilize the affected extremity. Postoperative immobilization is not performed. Physical therapy is prescribed for patients with multiple trauma or accessory injuries.

Our technique for internal plate fixation of the clavicle is as follows:[142,178] We apply a 3.5-mm limited contact dynamic compression plate (LCDC Plate, Synthes, Paoli, Pa.) or the locking compression plate (LC Plate, Synthes, Paoli, Pa.) to the anterior or superior aspect of the clavicle. A minimum of three screws should be placed in each major fragment. When the fracture pattern allows, an interfragmentary screw will greatly enhance the stability of the construct. In the presence of fracture comminution or gaps in the cortex opposite the plate, we recommend the addition of a small amount of autogenous iliac crest cancellous bone graft.

We prefer to close the wound over a suction drain, ensuring meticulous hemostasis. If the skin condition allows, wound closure is accomplished in an atraumatic fashion with a subcuticular suture. These measures will reduce the incidence of wound hematoma and promote cosmetic wound healing.

Our current practice, provided we are confident with the security of fixation, is to use a sling for patient comfort during the initial 7 to 10 postoperative days. The sling may be removed for short periods of passive shoulder pendulum and overhead elbow-flexion range-of-motion exercises without resistance, which are continued until fracture union has been demonstrated, which usually occurs between 6 and 8 weeks postoperatively. Thereafter, progressive strengthening exercises are permitted, and full overhead activities are gradually resumed. A return to all occupational duties and recreational pursuits is usually possible by 3 months after operative treatment.

In most cases, plate removal is unnecessary; on occasion, however, hardware may cause skin problems due to its prominence. In those instances, we remove the plate, provided a minimum of 12 to 18 months have elapsed since the injury and the cortex under the plate has reconstituted as viewed from an apical lordotic projection.

Distal Clavicular Fractures

Fractures of the distal clavicle with little or no displacement are treated symptomatically with a sling. While some cases of nonunion following such fractures have been reported,[227,263] the chance of this occurrence is extremely low.

Displaced distal clavicular fractures, on the other hand, are recognized as the only general type of clavicle fracture for which routine primary operative treatment should be considered. This is based on the work of Neer[216,218] and others[79,228] who have found that between 22% and 33% of these fractures will fail to unite following nonoperative treatment. An additional 45% to 67% will require more than 3 months to heal the fracture.

A number of surgeons have reported healing in 100% of operatively treated displaced lateral clavicle fractures within 6 to 10 weeks after surgery, with few associated complications. The period of disability in these cases was shortened, with a relatively rapid return to full shoulder mobility and function.[79,100,218,228] Other authors have reported acceptable results with nonoperative treatment,[204,205,269] stating that those few nonunions that become symptomatic can be treated with a reconstructive procedure at a time remote from the injury if necessary.

Neer recommended stabilization of displaced distal clavicular fractures using two Kirschner wires to control rotation. The wires are first passed into the distal fragment from proximal to distal through the fracture site, crossing the acromioclavicular joint and exiting the acromion and the skin on the lateral aspect of the shoulder. The protruding part of the wires is then engaged and advanced proximally across the fracture site and into the medial fragment. The wires are then bent to decrease the risk of migration, and are cut beneath the skin. Shoulder motion must be restricted to prevent pin breakage and migration. Others have used a single wire,[119,247,273] threaded wires,[119] or screws,[220,222,223] and some have made a point of avoiding the acromioclavicular joint. Caution was urged in a recent report by Kona et al., who noted high rates of both nonunion and infection with transacromial wire techniques.[158]

Alternative techniques for operative fixation of distal clavicular fractures include coracoclavicular screw fixation[11] or transfer of the coracoid to the clavicle.[43,110] A combination of coracoclavicular ligament reconstruction and wire fixation of the fracture fragments,[44,175] a transacromial Knowles pin fixation,[91] or a hook plate fixation[74,93,105,207,273] have also been described. The AO/ASIF group has recommended using a tension band wire construct with two Kirschner wires, which enter on the superior aspect of the clavicle, avoiding the acromioclavicular joint.[118,281] In addition, they also suggest consideration of a small plate, either a small T-shaped plate or a hook plate, and occasionally direct one of the screws into the coracoid as described by Bosworth.[11,29] A specially designed plate that is contoured so that its distal limit curves under the acromion through the acromioclavicular joint has also been utilized.[116,274]

We prefer to use a tension band wire technique. The distal clavicle and acromion are exposed through an incision in the relaxed tension lines of the skin with the development of thick flaps. Provisional fracture reduction can be held with a transacromial Kirschner wire. Definitive fixation consists of two stout smooth Kirschner wires passed through the outer edge of the acromion and crossing obliquely across the acromioclavicular joint and fracture to purchase in the solid cortex of the dorsal clavicle medial to the fracture. An 18-gauge wire is then looped through a drill hole medial to the fracture and around the tips of the wires, which are bent 180 degrees, turned downward, and impacted into the acromion (Fig. 31-11)

If a tear in either the trapezoid or conoid ligaments is identified, an attempt is made to perform a suture repair. The wound is closed over a suction drain. The postoperative management differs from that for midclavicular fractures in that patients are maintained in a sling for a minimum of 4 to 6 weeks.

Medial Clavicular Fractures

Little exists in the literature about fractures of the medial clavicle. These fractures are very uncommon and most surgeons have limited experience with them. The literature offers little more than case reports, the majority of which describe medial physeal separation injuries. While some authors recommend open reduction and internal fixation, the majority advocate nonoperative treatment initially with resection of the medial clavicle if symptoms persist.[3,136,143,176,258] Considering the risks attendant with implant insertion and migration in this region, we rarely consider operative treatment. The generally good results of nonoperative treatment are related to the

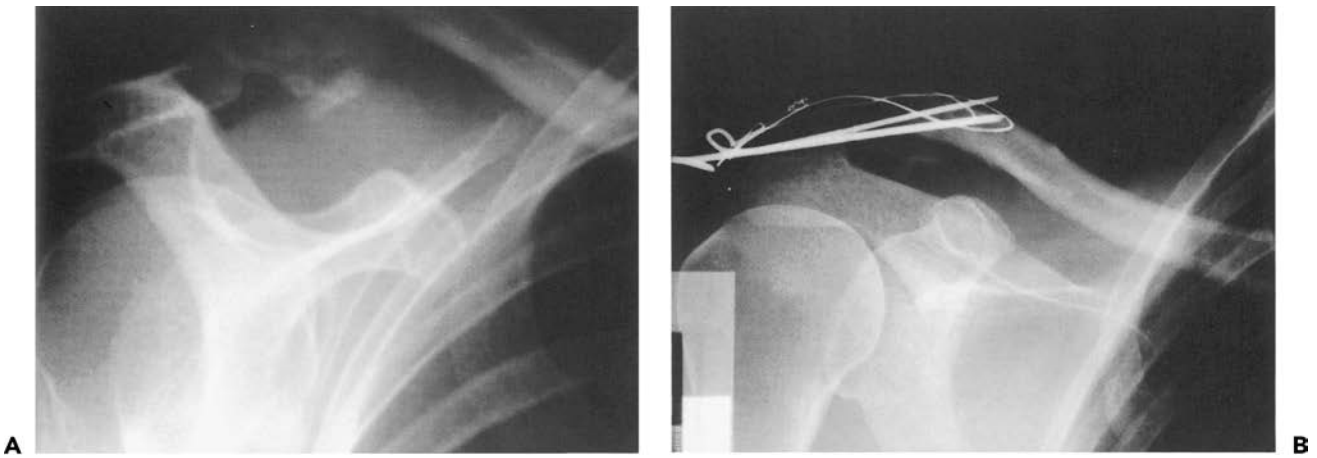


Figure 31-11 **(A)** A 55-year-old female sustained a comminuted type II fracture of the distal clavicle in a motor vehicle accident. **(B)** Fixation was achieved using two transacromial Kirschner wires exiting through the thick dorsal cortex of the medial fragment in combination with a tension band wire. (From Browner, Jupiter, Levine, Trafton, eds. *Skeletal trauma: fractures, dislocations, ligamentous injuries,* 2nd ed. Philadelphia: WB Saunders, 1998.)

fact that fractures of the sternal end of the clavicle are often physeal separations that heal with stability and may even remodel somewhat. Displaced fractures must be evaluated with computed tomography scanning to be certain that posterior displacement of the fragments does not present a threat to neurovascular structures at the base of the neck.

Floating Shoulder

Floating shoulder is an unstable combination of fractures that involves the scapular neck and the ipsilateral midclavicle, and it requires surgical treatment. According to Herscovici et al.,[120] floating shoulder is a rare injury with an incidence of approximately 0.1% in all patients with fractures. They also reported that functional disorders sometimes remain when only the fracture of the clavicle is diagnosed, the fracture of the scapula is missed, and conservative treatment is performed. Floating shoulder is an unstable injury that is likely to displace as a result of muscle strength and upper-limb weight, and for which surgical treatment is considered to be appropriate. If the patient is treated conservatively, where sufficient reduction and maintenance of reduction are difficult to assess, nonunion and/or malunion will occur, breaking down the suspension mechanism of the clavicle and leading to a drooping shoulder. Leung et al.[173] reported that open reduction and plate fixation procedures are necessary for both fractures of the scapula and the clavicle. On the other hand, Herscovici et al.[120,127] reported that, if the clavicle is fixed by open reduction, an unstable fracture becomes stable, resulting in bony union for both fractures; therefore, plate fixation of only the clavicle is sufficient to correct a floating shoulder.[114]

COMPLICATIONS

Nonunion and Malunion

The percentage of nonoperatively treated clavicular fractures that fail to heal within 6 months of injury has been reported variously as 0% (of 342 patients),[277] 0.1%,[216] 0.47%,[120] 0.8%,[239] 0.9%,[144] and 2.2%.[84,88] White et al. reported that they had encountered eight nonunions (8%) and 18 delayed unions (16%) among 112 adult fractures of the clavicle proximal to the coracoid, the majority occurring as a result of a high-energy traumatic injury.[307] Proposed risk factors for nonunion based on series of patients presenting with nonunion include the severity of the initial trauma,[24,95,134,276,309] fracture comminution, and refracture. Jupiter and Leffert,[142] as well as others,[188,202] found that the degree of fracture fragment displacement was the most important risk factor for nonunion. These risk factors often are interrelated and reflect more severe soft tissue injury, decreased stability, and limited apposition of fracture fragments. The role of soft tissue interposition remains unclear.[129,188] The fact that midclavicular nonunion is far more common than distal clavicular nonunion has been ascribed to the fact that midclavicular fractures are far more common overall. Primary operative treatment of fractures of the clavicle has been associated with a risk of nonunion (3.7% according to Rowe[273] and 4.6% according to Neer[219]). While contemporary series report high union rates with internal fixation of fresh clavicular fractures,[35,79,315] they identify improper technique including utilization of too small or too short a plate and excessive soft tissue stripping as reasons for operative failures[256,281] (Fig. 31-12).

On occasion a clavicular nonunion may be asymptomatic and discovered incidentally on a chest radiograph.[134]

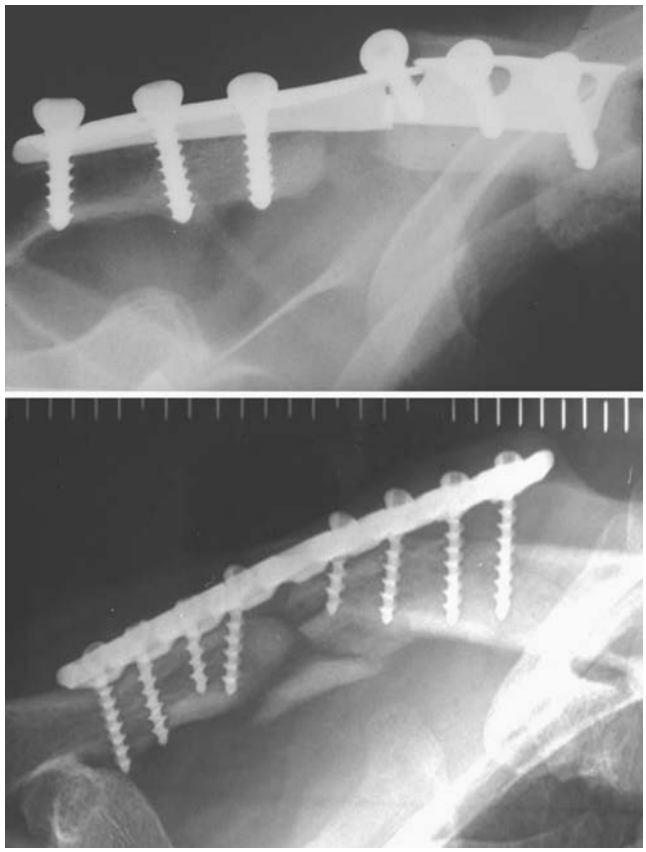


Figure 31-12 Implant loosening and nonunion are typically related to inadequate plate size and length. (With kind permission from Prof. Klaus Emil Rehm and Dr. Axel Jubel.)

Prior to more reliable means of internal fixation, some authors[134,309] recommended against operative intervention. In fact, patients presenting with an ununited clavicle are more likely than not to have specific complaints regarding increasing deformity, consisting of adduction, shortening, and protraction of the shoulder girdle. Altered shoulder function occurs as a result of the deformity, pain, or local compression of the underlying brachial plexus or vascular structures.[142,285] Occasionally patients will present decades after the original injury requesting treatment.[142,309] Unfortunately, this may be partly due to the fact that these patients had been advised previously that nothing operative could or ought to be done (Fig. 31-13).

The neurovascular problems that may accompany clavicular nonunion include thoracic outlet syndrome,[13,19,31,51,89,210,254,289] subclavian artery or vein compression[62,159] or thrombosis,[62,106,312,313] and brachial plexus palsy.[40,65,142] The prevalence of neurovascular dysfunction in patients presenting with clavicular nonunion has varied widely in reported series from as few as 6% to as many as 52% of patients.[134,142,157,309]

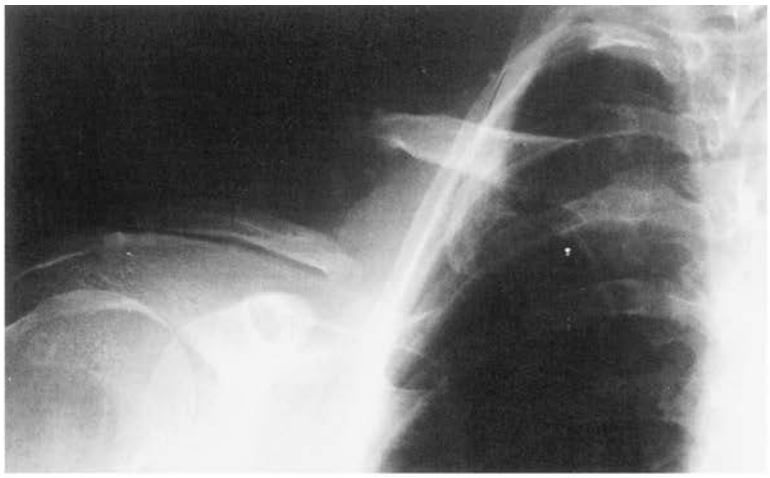
In the treatment of clavicular nonunions, we prefer to distinguish between reconstructive procedures in which the goals of both relief of pain and neurovascular compression as well as enhanced function are sought via restoration of the length, alignment, and continuity of the clavicle; and salvage procedures in which the clavicle is either resected, contoured, or avoided altogether (i.e., first rib resection[61]) with the limited goal of relieving symptoms. Although treatment of clavicular nonunion with electrical stimulation has been attempted,[34,50,51,63] there are few indications for its use. Symptomatic clavicular nonunion typically has elements of both shoulder deformity and dysfunction as well as neurovascular compromise that are not addressed by electrical treatment,[213] and the union rates do not approach those obtained by open reduction and internal fixation with provision of an autogenous bone graft.

With the advent of improved techniques of stable fixation, the results of reconstructive procedures have improved to the point that salvage operations are now largely of historical interest.[202,285] The only situation in which we would consider partial resection of the clavicle is in a chronically infected clavicle in a medically compromised patient or in a very distal clavicular nonunion. A small distal clavicular fragment can be resected and the coracoclavicular ligaments securely attached to the outer end of the medial fragment.[285]

The treatment of clavicular nonunion has evolved from the screw fixation of tibial or iliac crest bone grafts used by early authors,[16,98,276] to intramedullary fixation,[111,134,181] which seems to be a good alternative technique to plating,[28,41] to plate and screw fixation.[105,142,157,192] One of us treated 29 hypertrophic malunions of the clavicle with intramedullary nailing (as described above; study is currently being published). Twenty-five cases required an additional incision over the fracture to cannulate the medullary canal with a 3.5-mm drill. The bone shavings produced from debulking the callus, with or without additional cancellous bone graft, facilitated bony healing. In all cases, however, the patient was additionally informed and consented for plate fixation with cancellous bone grafting, since it is often decided intraoperatively that conventional plate fixation is necessary. Our preference for plate fixation and our operative technique and rehabilitation protocol are described previously under Author's Preferred Treatment. A few points regarding the treatment of midclavicular nonunions deserve further discussion.

In hypertrophic nonunions, the exuberant callus can be resected and saved for use as bone graft, making harvest of an iliac crest graft unnecessary in some cases. The nonunion site does not require débridement, as the fibrocartilage will progress to union following stable internal fixation. If the fracture line is oblique, it is sometimes possible to secure the fragments using an interfragmentary screw in addition to the superiorly placed plate.

Atrophic nonunions present sclerotic ends with interposed fibrous tissue while pseudoarthroses will have a false synovial-lined cavity. Resection of the ends of the



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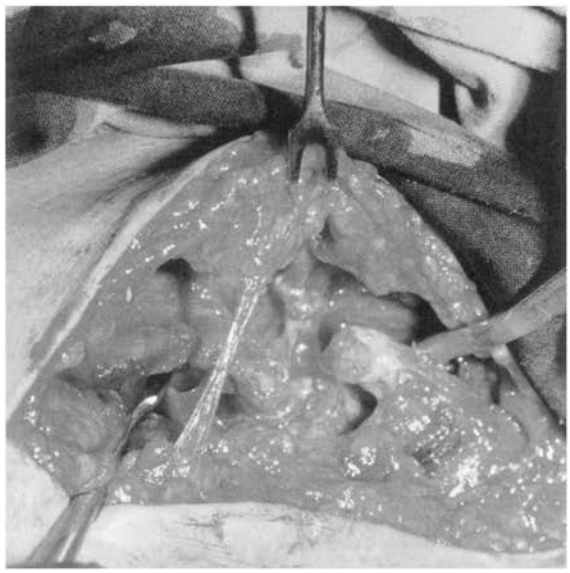

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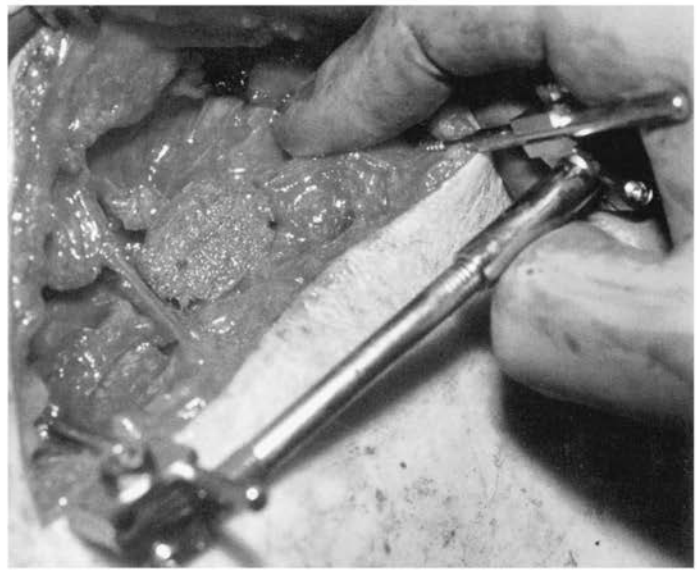

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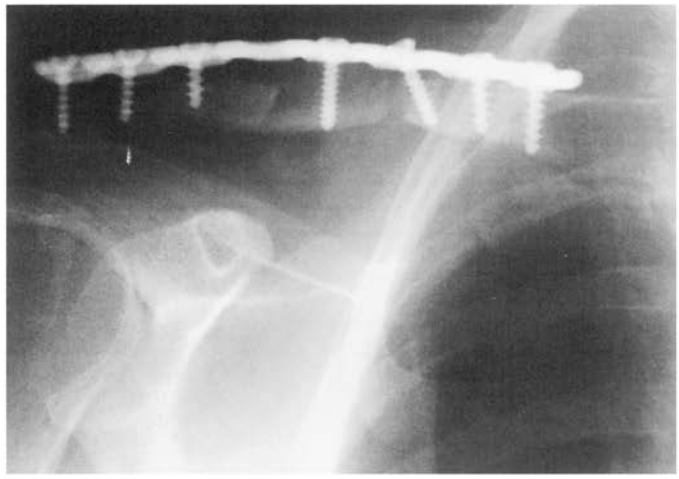


D

Figure 31-13 A 56-year-old woman presented with complaints of increasing shoulder pain, stiffness, and deformity after injuring her clavicle at age 14. **(A)** An anteroposterior radiograph demonstrated an ununited fracture of the clavicle with widely displaced, atrophic fragments. **(B)** The supraclavicular nerves were identified under loupe magnification and protected throughout the operative procedure. **(C)** A distractor was used to restore the anatomic length and alignment of the clavicle and a tricortical bone graft from the iliac crest was used to bridge the residual bony defect. **(D)** One of the screws through the plate transfixes the bone graft. (From Browner, Jupiter, Levine, Trafton, eds. *Skeletal trauma: fractures, dislocations, ligamentous injuries,* 2nd ed. Philadelphia: WB Saunders, 1998.)

fracture fragments and the intervening tissue is required in both situations. In this situation a small distractor often proves invaluable in helping to control the fragments as well as to attain the desired length and alignment. A sculptured tricortical iliac-crest bone graft will be useful to ensure restoration of length and alignment and to promote healing.

We harvest the iliac graft from the crest through an oblique incision along the midpoint of the ilium. The crest is exposed subperiosteally and a tricortical section, measuring one and one-half times the anticipated size of the final graft, is removed with either osteotomes or a small oscillating saw. The graft is then sculptured to create large cancellous pegs at each end, which plug into the medullary canals of the clavicular fragments (Fig. 31-14).

This interdigitation increases the stability of the construct and facilitates plate fixation. The graft is positioned so that the dorsal cortical margin of the iliac crest lies on the inferior surface of the clavicle, affording the advantages of better purchase of a screw as well as more resistance to bending forces at the nonunion site. Additional cancellous graft from the iliac crest is compacted into the medullary canals of each fragment prior to the final impaction of the corticocancellous segmental graft.

A 3.5-mm limited contact dynamic compression plate (Synthes, Paoli, Pa.) is then applied with a minimum of three screws in each major fragment and a single screw transfixing the graft. Compression is applied to both surfaces of the graft to enhance its incorporation. The wound is closed with a subcuticular suture over suction drainage.

Malunion has traditionally been considered primarily a cosmetic concern.[224] However, it is not surprising that some reports exist of difficulties in shoulder function in patients with overriding of clavicular fragments.[84,85,88] In addition, compression of underlying neurovascular structures has been reported in association with malaligned clavicle fractures due to narrowing of the costoclavicular space and compression of the brachial plexus and subclavian artery or vein. Malunited fractures typically may give rise to neurovascular symptoms weeks or months following the injury due to proliferative callus.[13,50,51,89,264]

The senior author (JBJ) has treated four patients with malunited clavicular fractures for deformity associated with ipsilateral glenohumeral dysfunction either alone or in combination with scapulothoracic dysfunction (unpublished series). The malunion was osteotomized through the plane of deformity, realigned using a small distractor, and secured with a plate and screws. In each case, function was improved and the outcome deemed satisfactory (Fig. 31-15).

Some patients are unhappy with the appearance of their healed clavicular fractures due to a prominent bump at the apex of the deformity. These patients should be advised that an operation to smooth the contour of the deformity would essentially trade a bump for a scar. There is a risk that the scar could be hypertrophic and more unsightly than the bump. Furthermore, the removal of bone places the clavicle at an increased risk for refracture.

Neurovascular Complications

Acute neurovascular complications are rare and typically occur in association with scapulothoracic dissociation[56,123,215] or are unrelated to the clavicular fracture (e.g., brachial plexus traction injury).[123,169] Neurovascular dysfunction as a result of narrowing of the thoracic outlet can occur within the first 2 months of injury when the fracture is malaligned or many months or even years later as a result of hypertrophic callus in the setting of nonunion.

Further mention should be made here of thrombosis and pseudoaneurysm of the subclavian or axillary artery or subclavian vein presenting at a time remote from the injury. Cases of axillary or subclavian artery thrombosis presenting late with symptoms of atrophy and cold intolerance of the involved upper extremity most likely represent missed acute intimal injury,[123,128,283] but may also result from compression in a narrowed costoclavicular space.[295] Cerebral embolism has been reported following subclavian artery thrombosis in this setting.[312]

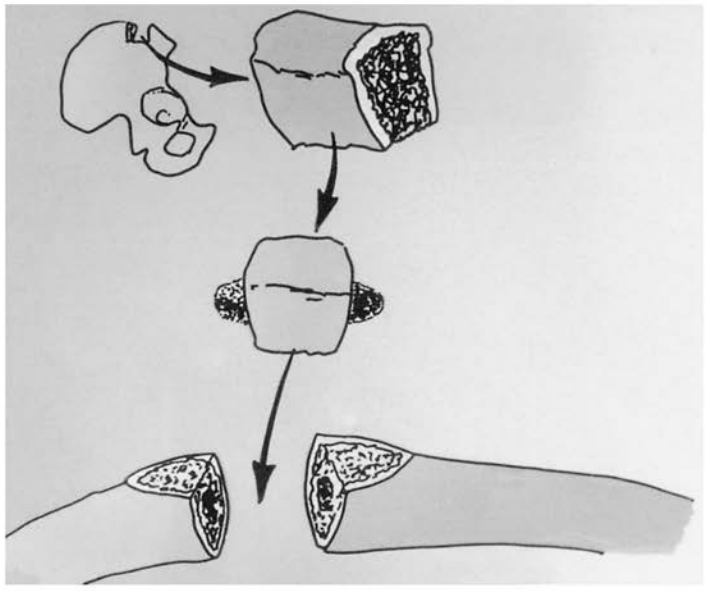
True aneurysms of the subclavian artery may occur as poststenotic aneurysms when the costoclavicular space is narrowed.[64,395] Displaced clavicular fracture fragments may very rarely cause a small perforation injury of the subclavian artery. Occasionally a pseudoaneurysm develops that may present months to years later with brachial plexus dysfunction due to compression.

Neurovascular symptoms related to compression by hypertrophic nonunion have been mistaken for sympathetically maintained pain (shoulder–hand syndrome) in the past. Damage to the supraclavicular nerves can cause anterior chest wall pain.[259]

Complications of Operative Treatment

Despite the proximity of important anatomic structures beneath the clavicle, intraoperative complications are rare. Eskola et al. reported tearing of the subclavian vein, pneumothorax, air embolism, and brachial plexus palsy all in a single patient during dissection of a clavicular nonunion.[84,151] On the other hand, wires and pins show a remarkable ability to migrate once inserted[182] and may ultimately be found in the abdominal aorta,[213] ascending aorta,[225] and pericardium causing fatal tamponade[46]; the pulmonary artery[174]; the mediastinum[38]; the heart[248]; the lung[199,207,267] (sometimes the opposite lung[267]); or the spinal canal.[232] A patient presenting to Kremens and Glauser[161] brought in a Steinman pin that he reported having expectorated 1 month following fixation of his medial clavicular fracture.

A



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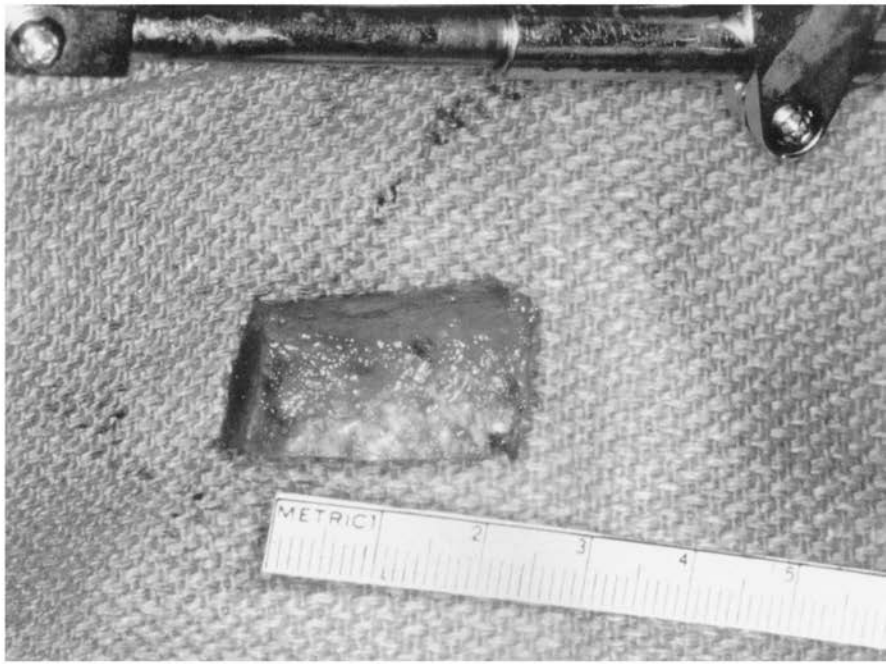
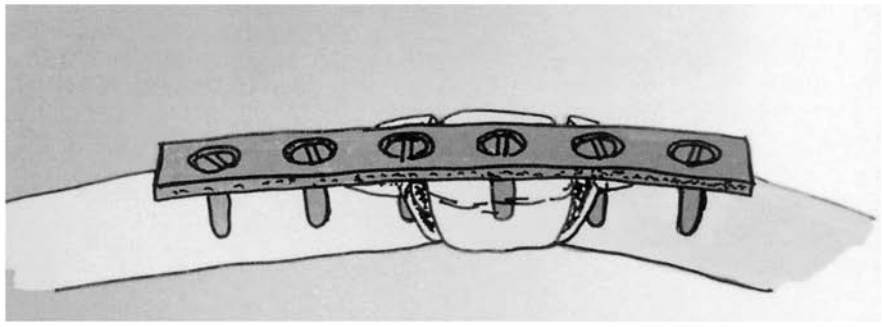


C

Figure 31-14 Sculpturing of the tricortical iliac crest autograft allows interdigitation of the graft with the fracture fragments increasing the stability of the construct and enhancing healing. **(A)** Following resection of the nonunion back to viable bone and distraction of the clavicle to restore length and alignment, a substantial bony defect is common. **(B)** A tricortical graft is harvested from the iliac crest and cancellous pegs are sculpted at the medial and lateral aspects. The medullary canals of the fracture fragments are opened with a drill and the pegs interdigitate with the fragments. **(C)** The harvested iliac crest graft prior to sculpturing.

D



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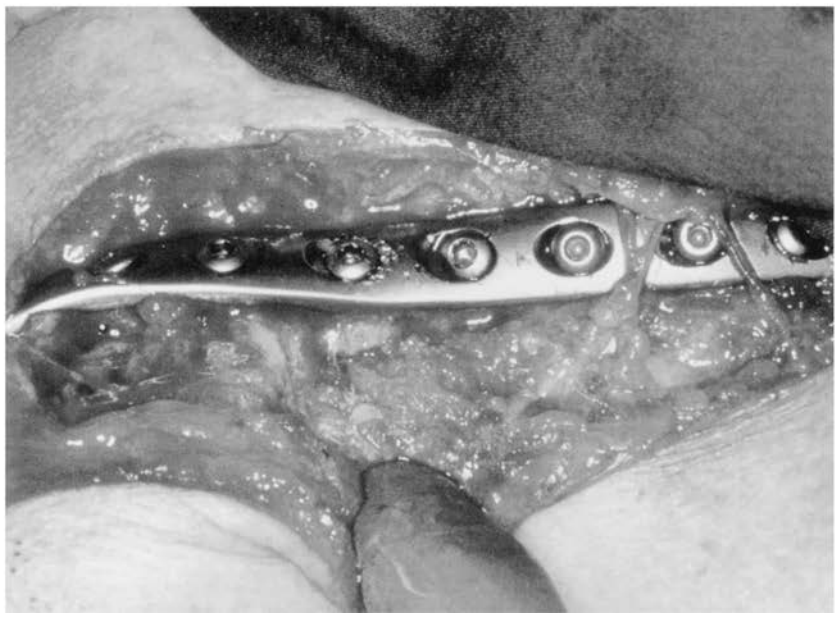

F

Figure 31-14 (*continued*) **(D)** The graft following sculpturing. **(E)** The interdigitation of the graft with the fracture fragments provides a measure of stability, facilitating plate fixation. One of the screws transfixes the plate. **(F)** Intraoperative photograph demonstrating plate fixation following graft interposition. (From Browner, Jupiter, Levine, Trafton, eds. *Skeletal trauma: fractures, dislocations, ligamentous injuries*, 2nd ed. Philadelphia: WB Saunders, 1998.)

Poigenfürst et al. documented superficial infections in 9 (7.4%) and deep infections in 2 (1.6%) of 122 patients undergoing plate fixation of fresh clavicular fractures,[256] but other authors have had little trouble with infections following plate fixation.[35,141,142,188,242] Many authors cite hypertrophic scar formation as one of the potential complications of operative treatment of clavicular fractures,[147] particularly the proponents of intramedullary fixation who advocate a more oblique incision in alignment with Langer's lines.[220,222,223] We have had no particular problem with cosmetically displeasing scars.

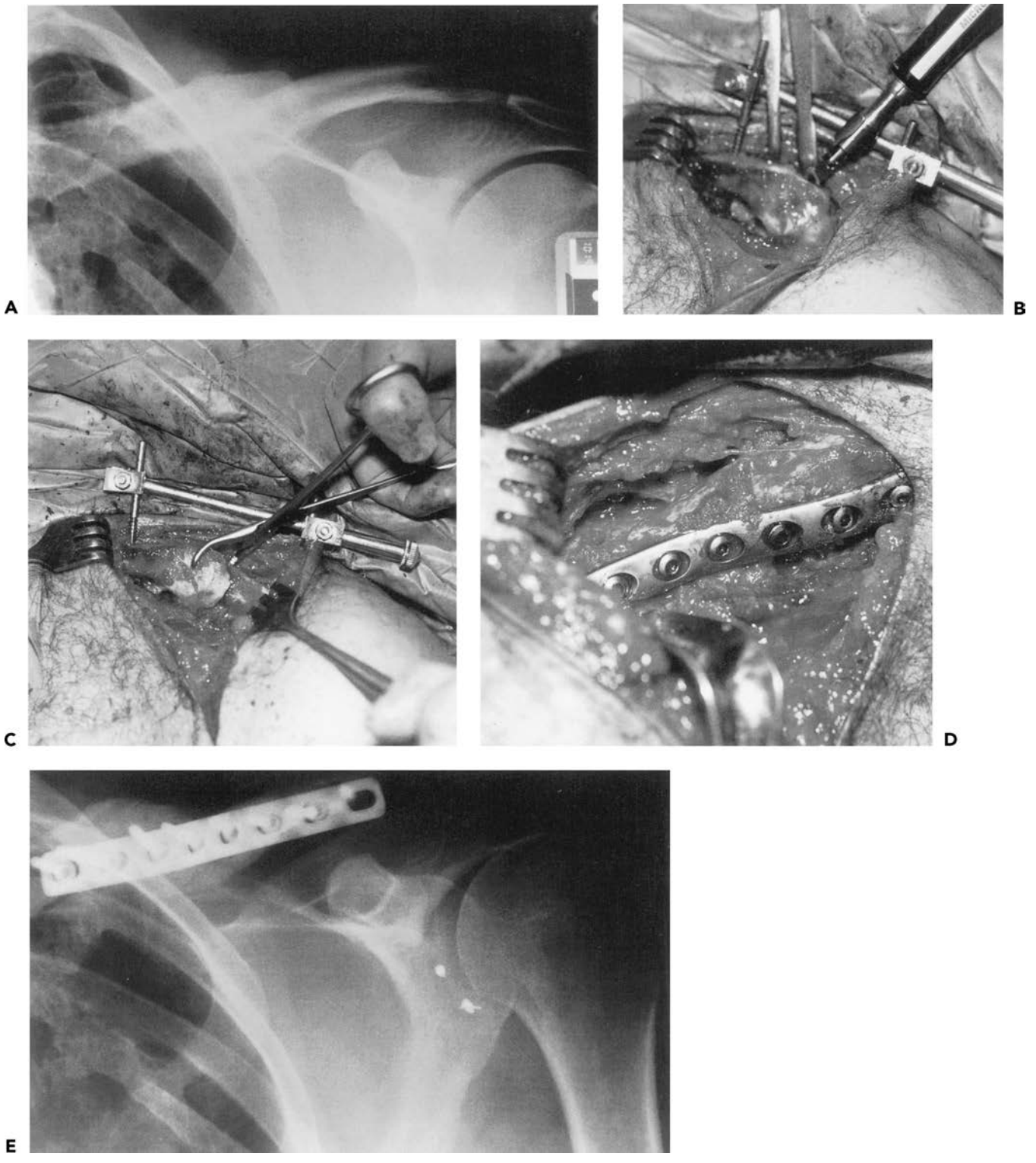

Figure 31-15 A 35-year-old male presented with complaints of restricted shoulder motion, weakness, and discomfort after healing of his fractured clavicle with 2 cm of shortening. **(A)** Anteroposterior radiograph demonstrating malunion of the clavicle. **(B)** An oscillating saw is used to create a long oblique osteotomy of the clavicle. **(C)** A distractor is used to restore length and alignment. **(D)** Fracture reduction forceps help maintain alignment while an anterior plate incorporating an interfragmentary screw is applied. **(E)** Radiograph demonstrates stable plate fixation with restoration of clavicular length. (From Browner, Jupiter, Levine, Trafton, eds. *Skeletal trauma: fractures, dislocations, ligamentous injuries*, 2nd ed. Philadelphia: WB Saunders, 1998.)

Refracture

Repeat fracture of the clavicle usually occurs upon premature resumption of full activity, in particular contact sports activity. The typically vigorous healing response of the clavicle results in a rapid decrease in pain and return of shoulder function so that overenthusiastic patients will often ignore their physician's admonition to avoid contact sports for at least 2 to 3 months or longer following healing of the fracture. Refracture following plate removal is unusual if the plate remains in place a minimum of 12 to 18 months following healing of the fracture.[256,285]

Failed Internal Fixation

The standard method of fixation for the clavicle is plate fixation. The particular problem with this technique is that the tension-held side of the clavicle, and thus the optimal position for the plate, changes with each direction of stress and rotation of the arm. Tensile forces against the implant cannot be prevented using plate fixation. To avoid plate breakage, a very large plate in proportion to the size of the bone must be chosen. However, using a typical narrow 3.5-mm LCDC plate or 3.5-mm reconstruction plate, the desired screw anchoring is not always possible. Typical complications after plate fixation include implant fracture, implant loosening, malunion, and refracture after removing the hardware. In cases of implant breakage or loosening, normally refixation is indicated if the fracture is not yet consolidated and the soft tissues allow for another procedure.

Fractures are not expected to heal independently when implants break or loosen. The goals of refixation are to achieve more proportionate stability of the fracture to avoid nonunion and to restore the clavicular length.

For refixation, a 3.5- mm LCDC plate, a 3.5-mm LC plate, or a corresponding reconstruction plate should be chosen; a one-third tubular plate is not appropriate. At least six, and preferably eight, cortical screws should be inserted into each major fragment. Particularly in cases of screw loosening, the fixed-angle plates should be used since in such cases (as well as in cases of poor bone quality), better bony anchoring can be achieved. However, if an adequate position cannot be found using a straight plate despite bending, the molded reconstruction plate is better. Because of the osteoporosis present in refixation procedures of the clavicle, titanium implants should be used exclusively (due to biocompatibility). Intramedullary support in the form of elastic titanium nails is an alternative to refixation with a plate for simple (two-fragment) fractures. After refixation procedures, elevation and abduction of the affected arm should be limited to 90 degrees for a period of 6 weeks to limit the rotation of the clavicle on the longitudinal axis.

In cases of implant fracture or loosening in combination with soft tissue infection or wound-healing disturbances, therapeutic measures should be taken in stepwise fashion. The first procedure should be simple implant removal. Once the soft tissues have revitalized, bony refixation is indicated. However, in such cases it should be explained to the patient that operative refixation may be delayed posthardware removal and postinfection recovery, in anticipation of spontaneous healing. In the case of bony consolidation, potential outcomes should be estimated as for nonoperative management. If bony healing does not occur spontaneously, the nonunion can always be treated operatively.

The development of nonunion after plate fixation, when the plate remains intact, is always the expression of a biologic (cellular), as opposed to mechanical, problem. Operative therapy for such cases requires placement of a corticocancellous bone strut graft from the iliac crest to be anchored ventrally across the fracture site with minifragment screws. (See section on Nonunion and Malunion.) This should be performed after the nonunion tissue has been débrided. If the plate is found to be loosened intraoperatively, the refixation should be performed according to the principles described previously with a 3.5-mm plate.

In cases of loose implants with radiographic evidence of delayed union or nonunion, we believe ultrasonic therapy is indicated prior to performing further operative measures.

Implant fractures, implant loosening, and nonunion postlateral clavicle fractures are usually evidence of a combination of mechanical and cellular problems. The local cellular problem is solved by débriding the nonunion tissue and attaching a corticocancellous iliac crest strut graft that can be fixed to the medial and lateral fragments with mini- or small-fragment screws. (See Nonunion and Malunion.) If there is sufficient bony anchoring for the lateral fragment, the mechanical problem can be solved by using a fixed-angle small fragment plate for refixation. The medial fracture fragment should additionally be fixed to the coracoid with a 1.5-mm Polydioxanonacid (PDS) cord figure-eight suture. If the lateral fragment is too short for good anchoring, we stabilize the fracture using a titanium nail introduced over the acromion, which enters the medullary canal of the clavicle from lateral (Fig. 31-16) or using two plates fixed from the dorsal and anterior aspect (Fig. 31-17).

As a last resort for chronic pain secondary to lateral malunion, there remains the option to resect the lateral third of the clavicle. The problem with sternal-end clavicular fractures is normally the short medial fragment. In these cases, implant loosening and subsequent fracture can occur as a result of insufficient anchoring. For refixation, we choose a fixed-angle 2.7-mm minifragment plate in L- or T-form. With such a plate, it is normally possible to anchor two to three screws in the medial fragment. Additionally, the lateral fragment is fixed near the sternum to the second or third rib with a 1.5-mm PDS-cord figure-eight suture.

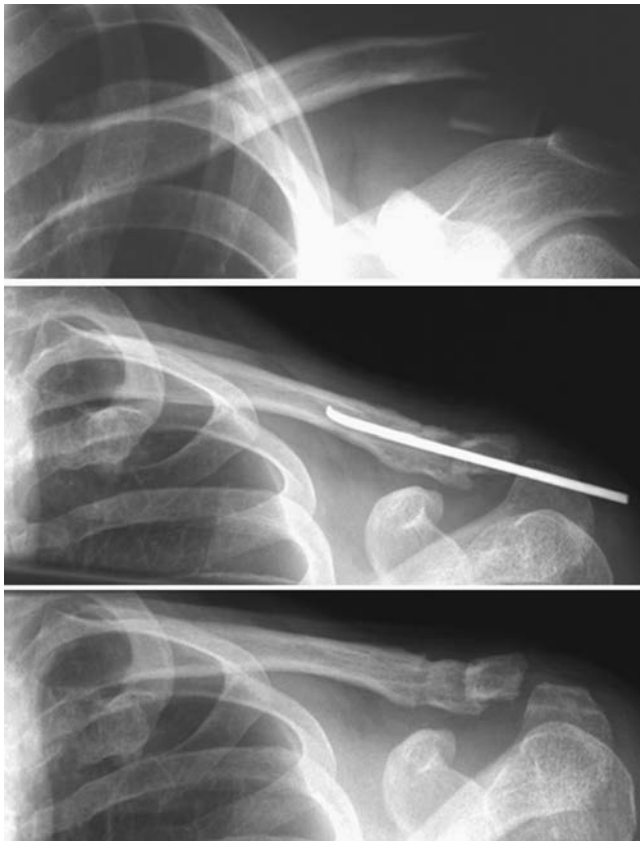

Figure 31-16 If the lateral fragment is too short for good anchoring, we stabilize the fracture using a titanium nail introduced over the acromion. The medial fracture fragment should additionally be fixed to the coracoid with a 1.5-mm PDS-cord figure-eight suture. (With kind permission from Prof. Klaus Emil Rehm and Dr. Axel Jubel.)

When refracture of the clavicle occurs after implant removal, indications for refixation depend on the complaints of the patient and the conditions of the local soft tissue. Again, this appears to come from a (preoperatively present) cellular problem. It can therefore, in principle, be treated like the case of a nonunion. Alternatively, with suitable fracture forms, intramedullary stabilization can be performed. Intramedullary fixation is performed with the goal of avoiding further compromise of the periclavicular soft tissue.

In fractures with a large wedge fragment or a comminuted zone (CCF type C), intramedullary fixation is not indicated. Possible complications postintramedullary stabilization of the clavicle are lateral perforation, painful protrusion of the medial nail end, implant displacement, and nonunion. In cases of painful, moderately prominent medial nail ends, a so-called pseudobursa can develop in a period of 4 to 6 weeks postfixation. Only in such a case, where a pseudobursa develops and the patient continues to complain of pain, or in the case of a very long nail end with impending skin perforation should a nail-shortening procedure (under local anesthesia) be performed.

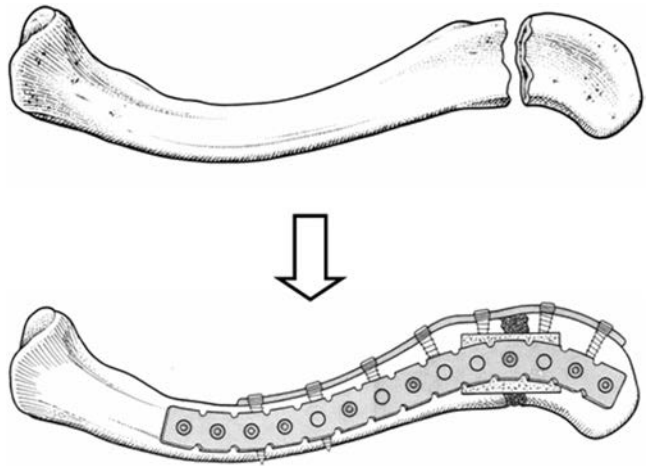

Figure 31-17 Another option in the case of a small lateral fragment is the use of two limited contact plates in combination with a bone graft.

Lateral perforation alone is not a problem, and does not require a corrective procedure. Only cases where lateral displacement of the implant occurs during the course of healing require therapy. Premature removal of the implant should be performed from the lateral side. Indications for refixation should depend on the further course and complaints of the patient; however, only seldom is reoperation necessary, since healing results are similar to those after conservative management. When indications for refixation exist such as increased displacement, absence of bony healing, and/or increased subjective complaints from the patient, it should be performed as described previously with a plate.

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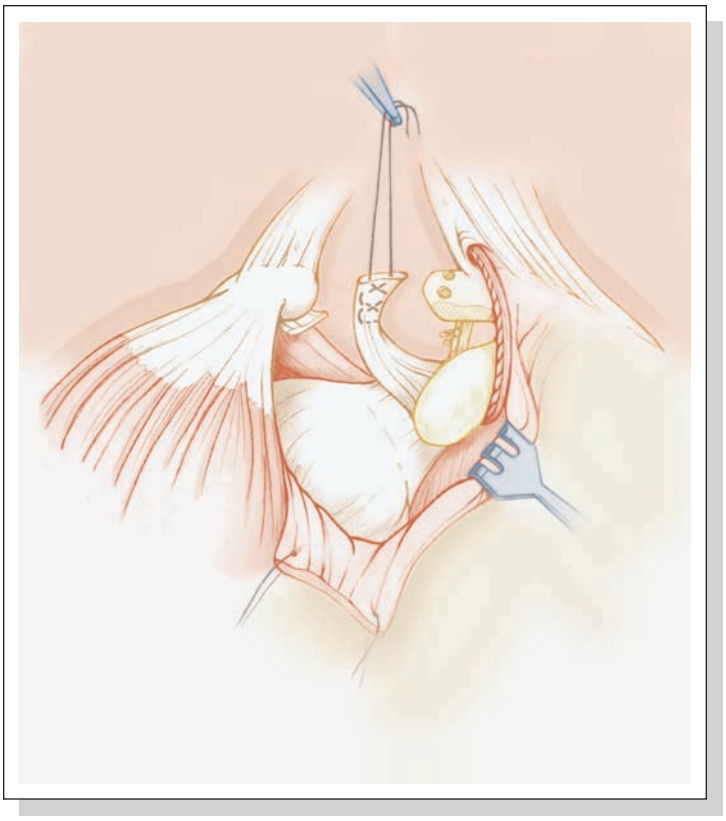
Acromioclavicular and Sternoclavicular Joints

VI



32 Disorders of the Acromioclavicular Joint: Pathophysiology, Diagnosis, and Management

Gordon W. Nuber *Laurent Lafosse*

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INTRODUCTION

The acromioclavicular joint is a diarthrodial joint formed by the distal clavicle and the medial facet of the acromion. This joint stabilizes the scapula and upper extremity to the clavicle via a complex of ligaments and the fascia of two muscles. As orthopedic surgeons we are frequently presented with patients suffering traumatic or degenerative conditions of this joint. Traumatic injuries of the acromioclavicular joint frequently occur in persons participating in contact and collision sports such as football, hockey, and rugby or those suffering a fall onto the shoulder.[28,34,96,114] Degenerative conditions evolve as a consequence of daily life but can also be provoked by repetitive use in sports such as baseball and weightlifting. Treatment of injuries to the acromioclavicular joint continues to be a topic of active discussion. Injuries that may be inconsequential to

the average person may restrict the activity of the competing athlete. A complete understanding of the relevant anatomy, biomechanics, and diagnosis of injuries to the acromioclavicular joint helps suggest a logical approach to treatment and restoration of shoulder function. This chapter should provide the reader with a working knowledge of the pertinent anatomy and biomechanics of this joint, along with recognition of injury, classification, nonoperative management, and current standards of operative treatment.

BONE AND LIGAMENT ANATOMY

An understanding of the anatomy and biomechanics of the acromioclavicular joint is essential to treating patients with acromioclavicular pathology (Fig. 32-1). Knowledge of the exact locations of insertion of the acromioclavicular ligaments along with the conoid and trapezoid ligament is important prior to operative resection of the distal clavicle. Comprehensive knowledge of the bony and ligamentous anatomy of this joint will also give us a better understanding of newer and better operative techniques to recreate anatomy of the acromioclavicular joint than are currently in our armamentarium.

The acromioclavicular joint is a diarthrodial joint involving the medial facet of the acromion and the distal clavicle. Its articular surfaces are covered with hyaline cartilage until age 17 when it acquires the characteristics of fibrocartilage on its acromial surface and at age 23 on its clavicular surface.[111] DePalma noted that before the age of 3 to 5 years only a fibrocartilaginous bridge exists between the acromion and the clavicle and not a true joint cavity.[32] Along with the sternoclavicular joint, the acromioclavicular joint provides a bony link of the shoulder to the axial skeleton. The inclination of the joint is variable in both the sagittal and coronal planes.[92] The most common type of inclination is with the clavicle overriding the acromion. The least common inclination is with the clavicle underriding the acromion.[112] Pitchford and Cahill felt that acromioclavicular joints with a more vertical orientation were more susceptible to osteolysis because higher forces were concentrated across the clavicle.[89] A fibrocartilaginous disc of varying size and shape is present in the joint.[33] Degenerative changes of the disc increase in frequency with age.

The presence of a coracoclavicular joint is uncommon but varies from the presence of a well-developed diarthrodial articulation to the presence of articular facets and synovial structures within the coracoclavicular ligaments.[52] A bony bar may form in place of the coracoclavicular ligaments, the

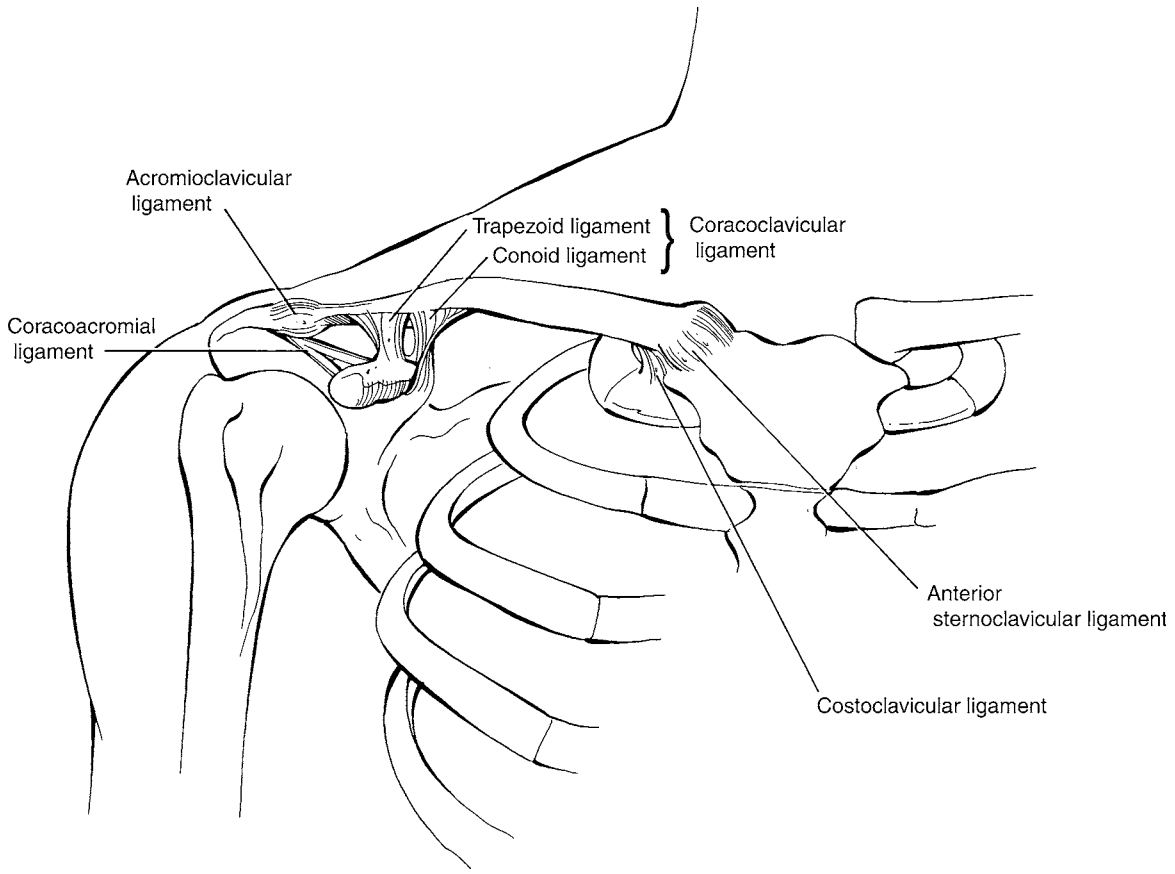


Figure 32-1 Ligamentous anatomy of the acromioclavicular joint.

so-called "coracoclavicular bar."[98] This connection may be complete or incomplete. Coracoclavicular articulations have also been linked to compression of the subclavian artery and brachial plexus, along with early degenerative changes of the acromioclavicular and sternoclavicular joints.[98,120]

The primary stabilizers of the acromioclavicular joints are the ligaments that surround the joint. A thin capsule surrounds the joint and is stabilized by anterior and posterior as well as superior and inferior ligaments. The superior acromioclavicular ligament is the most robust of the ligaments and is reinforced by fascial attachments of the deltoid and trapezial muscles.[97] The inferior ligament in many cases is nonexistant.[91] The insertion points of the superior acromioclavicular ligament on the acromion and clavicle were determined by Wright and led to the conclusion that resection of bone as little as 2.6 mm of the distal clavicle in men and 2.3 mm in women performed arthroscopically could violate the ligament.

Vertical stability (superoinferior) is provided by the coracoclavicular ligaments (conoid and trapezoid). These ligaments pass from the inferior surface of the clavicle to the base of the coracoid process of the scapula. The trapezoid ligament is the most lateral of the two. It is a broad, thin, quadrilateral-shaped ligament running obliquely from the superior surface of the coracoid process to the oblique ridge on the inferior surface of the clavicle. The conoid ligament is more conical in form, with its base directed superiorly. Its apex attaches to a rough impression at the base of the coracoid process medial to the trapezoid ligament. It attaches by its base to the coracoid tuberosity on the inferior surface of the clavicle and a line proceeding medial for 1 cm on this bone. These ligaments are the primary stabilizers of the acromioclavicular joint and constitute the primary supports by which the scapula is suspended from the clavicle. Division of the acromioclavicular ligaments and joint capsule, along with detachment of the trapezius and deltoid, allows less than 50% subluxation superiorly. Posterior dislocation, though, is possible.[112] Complete superior dislocation of the acromioclavicular joint will occur only after complete division of the coracoclavicular ligaments has also occurred.

The conoid ligament is the primary stabilizer with superior displacements. Salter found that there was considerable variation in the length and width of the conoid and trapezoid ligaments.[97] He also found that a bursa can exist between the ligaments. Recent literature also found variation in the insertional distances (from the acromioclavicular joint) of these ligaments. There was found to be a significant gender-related difference in the insertional distances of either the trapezoid or conoid ligaments. Resection of less than 11 mm should not violate the trapezoid ligament and resection less than 24 mm should not violate the conoid ligament in either gender.

In children, the clavicle is surrounded by a thick periosteal tube that extends all the way to the acromioclavicular joint. A secondary ossification center is present at the distal clavicle and fuses with the clavicle at about the age of 19.[26] Because of the variation in anatomy compared with adults, children are more prone to fracture and pseudodislocations than true dislocation of the acromioclavicular joint.[39,85]

An ossification center appears in the body of the coracoid at approximately 1 year of age. Another combined ossification center for the base of the coracoid and upper forth of the glenoid occurs close to age 10.[26] These centers fuse to the scapula at about age 15. Near puberty, the acromion forms two to five ossification centers that fuse at about age 22. Failure to fuse the acromial ossification center leads to an os acromiale.

BIOMECHANICS

Acromioclavicular joint motion occurs as the arm abducts and forward flexes. While the clavicle rotates upward 50 degrees with overhead motion, only 5 to 8 degrees occurs at the acromioclavicular joint due to synchronous scapuloclavicular motion. Observations by Inman et al. indicated that the clavicle rotates as the arm is elevated.[54] This was always felt to imply that motion (rotation) must be present at the acromioclavicular joint for overhead motion to occur. This finding was contradicted by observations by other authors, who found little motion restriction after acromioclavicular joint fusions or fixations.[42,93] Rockwood, in a classic study in which percutaneous pins were implanted in volunteers, found little motion between the acromion and clavicle.[93] His findings led him to conclude that there was synchronous movement of the scapula and clavicle. Thus, while the clavicle rotates the scapula rotates with it, and therefore, minimal rotation (5 to 8 degrees) is required between the two. Hence, most scapulothoracic motion must occur at the sternoclavicular articulation. Clinically, fusion of the acromioclavicular joint will lead to minimal dysfunction.

Stability of the acromioclavicular joint is rendered by the ligamentous complex. Testing of strength to failure at the Biomechanics lab at the Mayo Clinic demonstrated the acromioclavicular capsule/ligament complex to be the strongest and stiffest, followed by the conoid ligament and finally the trapezoid ligament.[60] Fukuda et al. used 12 fresh frozen specimens and studied ligamentous contribution to joint constraint by performing load displacement tests, along with sequential cutting of the ligaments.[44] The contributions of the different ligaments to constraint changed not only with direction of loading, but also with amount of loading and displacement. With small amounts of load and induced displacement, the acromioclavicular ligament contributed more restraining force to superior displacement. With larger loads and displacement, the coracoclavicular (conoid) ligaments were major restraints. The acromioclavicular ligaments (superior and posterior) acted as the primary constraint to posterior clavicular displacement and posterior axial rotation at all loads. For axial compression

loading of the acromioclavicular joint, the trapezoid muscle was the major restraint. Debski et al. confirmed these observations by noting 100% displacement in the anterior and posterior direction after resection of the acromioclavicular ligaments.[30] Other authors have shown that the inferior acromioclavicular ligament only contributes about 50% of the restraint to force as the superior ligament.[18,31,61] This work implies that, to achieve normal physiologic function of this joint, preservation or reconstruction of both stabilizing groups of ligaments is important. Surgical procedures that excise the distal clavicle may not make this possible. Posterior abutment of the resected distal clavicle against the base of the acromion may be a cause of failure after open distal clavicle resection.[14,24,42] Traditionally, large amounts (1 to 2 cm) of resection of the clavicle were recommended. Acromioclavicular resection of even 1 cm would potentially compromise anteroposterior stability of the distal clavicle by loss of capsular insertion. Resection of less bone (5 mm), along with preservation of part of the acromioclavicular ligament, either superior or posterior or inferior, will ensure that no bone-to-bone abutment occurs. This can be accomplished either through an open or arthroscopic approach.[7,18,43]

The impact of the deltoid and trapezius muscle and fascia on stabilizing the acromioclavicular joint is unknown. The fascia of these muscles blends with the superior acromioclavicular ligament and adds stability to the joint, but the contribution has not been elucidated.[11] Most surgeons recognize the importance of this fascia and imbricate the fascia over the acromioclavicular joint to reinforce its stability during reconstruction.[17,94] These muscles may also contribute to generation of forces across the acromioclavicular joint and the genesis of osteolysis during weightlifting activities.[20]

CLASSIFICATION OF ACROMIOCLAVICULAR JOINT DISLOCATIONS

Injuries to the acromioclavicular joint can be classified according to findings on physical examination and anteroposterior (AP) and axillary radiographs. The amount of damage to the acromioclavicular and coracoclavicular ligaments, as well as the deltoid and trapezius attachments, determines the current classification.

Injuries of the acromioclavicular joint most commonly occur as a result of direct trauma. Typically, a fall onto the superior aspect of the shoulder with the arm in adduction occurs, as with a fall off a bike or a horse. Another mechanism of injury is a laterally based force directed to the shoulder, as in a hockey player hitting the boards. If the upper extremity is not used to absorb the force, the area of impact is distributed across the acromioclavicular joint. Because of significant stability of the sternoclavicular joint, the energy of impact is distributed to the acromioclavicular and coracoclavicular ligaments or the clavicle itself.[10,82] The direction and magnitude of the forces determine the severity of injury and the structures injured. With an acromioclavicular injury, the force is initially distributed to the acromioclavicular ligaments. If the magnitude of force is severe, injury progresses to the coracoclavicular ligaments along with the deltoid and trapezial fascia. Fractures of the distal one-third of the clavicle occur rarely, but should be recognized. These fractures may or may not be associated with coracoclavicular ligament disruption.

Injuries to the acromioclavicular joint by indirect injury have also been described. Force is typically directed superiorly by the humeral head upward into the acromion. This may occur with a fall on an elbow or outstretched arm. With this mechanism, injury is primarily to the acromioclavicular joint as the coracoclavicular ligaments are relaxed with upward movement of the scapula relative to the clavicle.

The mechanism of injury for the most severe dislocation, type VI, is felt to be a severe force directed onto the superior surface of the distal clavicle along with abduction of the arm and retraction of the clavicle.[48,72,87]

Classically, injuries to the acromioclavicular joint were classified as either types I, II, or III.[3,110] Rockwood added three other types of injury to the acromioclavicular joint and expanded the classification to the current six types. Recent magnetic resonance imaging of the coracoclavicular ligaments after injury and in a cadaver has questioned the validity of the Rockwood classification system to accurately determine pathoanatomy.[6] Keeping with convention and until another classification defining the pathology is validated, we will use the Rockwood classification, as described below:

Rockwood Classification

Type I injury (Fig. 32-2)
Sprain of the acromioclavicular ligaments alone

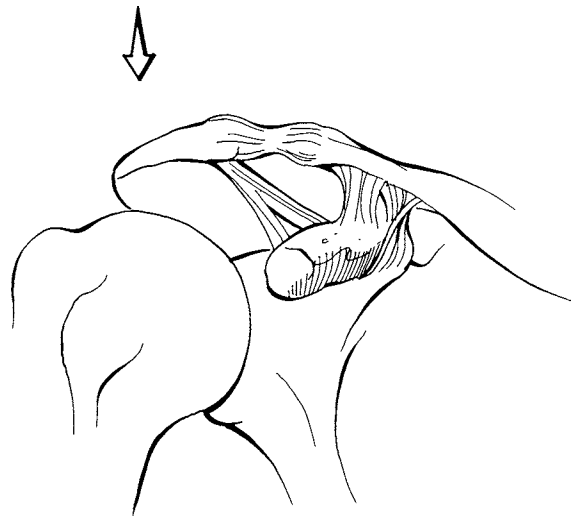


Figure 32-2 Type I Injury.

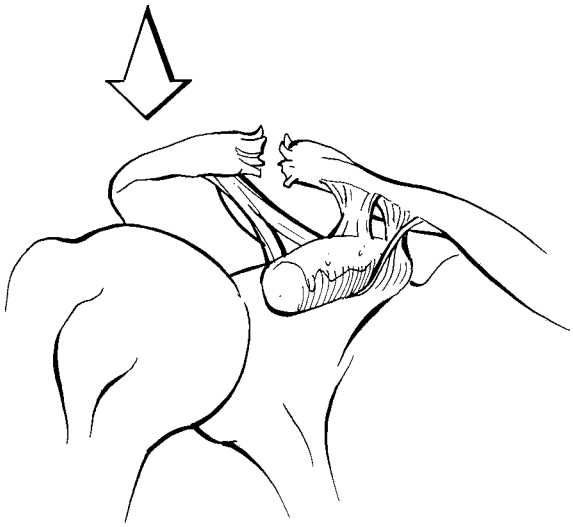


Figure 32-3 Type II Injury.

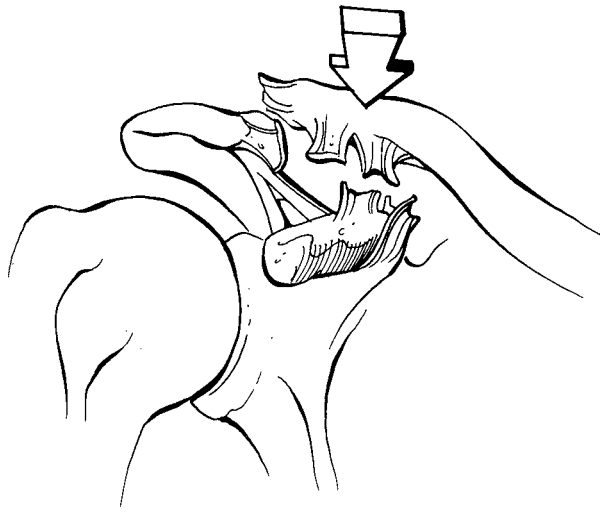


Figure 32-5 Type IV Injury.

Type II injury (Fig. 32-3)
- Acromioclavicular ligament and joint capsule disrupted
- Coracoclavicular ligaments intact
- Up to 50% vertical subluxation of the clavicle

Type III injury (Fig. 32-4)
- Acromioclavicular ligament and capsule disrupted
- Coracoclavicular ligaments disrupted
- Dislocation of acromioclavicular joint, with the clavicle displaced superiorly, and loss of contact between the clavicle and acromion

Type IV injury (Fig. 32-5)
- Acromioclavicular ligament and capsule disrupted
- Coracoclavicular ligaments disrupted
- Acromioclavicular joint dislocation with clavicle displaced posteriorly into or through the trapezius muscle (posterior displacement confirmed by axillary radiograph)

Type V injury (Fig. 32-6)
- Acromioclavicular ligament and capsule disrupted
- Coracoclavicular ligament disrupted
- Complete detachment of deltoid and trapezius fascia from the distal clavicle
- Acromioclavicular joint dislocated with extreme superior elevation of the clavicle (100% to 300% of normal)

Type VI injury (Fig. 32-7)
- Acromioclavicular ligament and capsule disrupted
- Coracoclavicular ligaments disrupted
- Acromioclavicular joint disrupted with the clavicle displaced inferior to the acromion or coracoid process

Various type III equivalent injuries have been described, primarily in children. Children younger than 15 years typically do not sustain true dislocations of the acromioclavicular

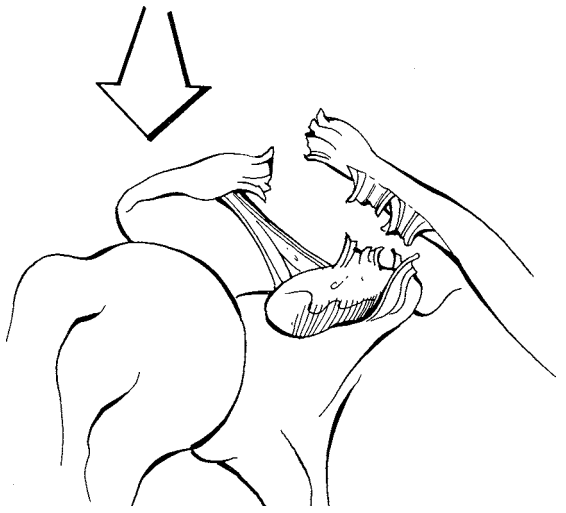


Figure 32-4 Type III Injury.

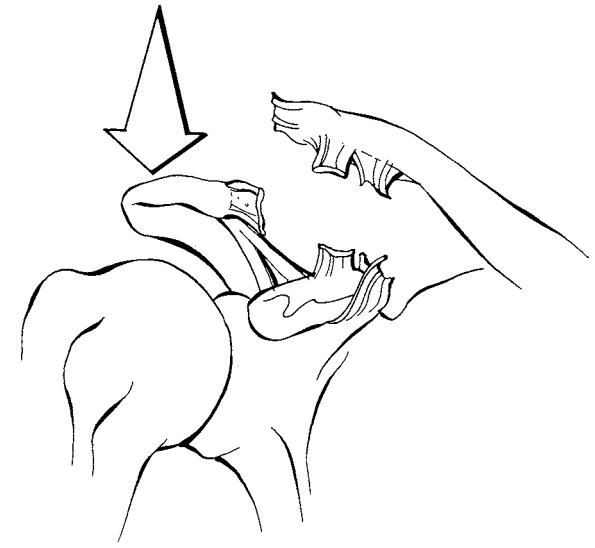


Figure 32-6 Type V Injury.

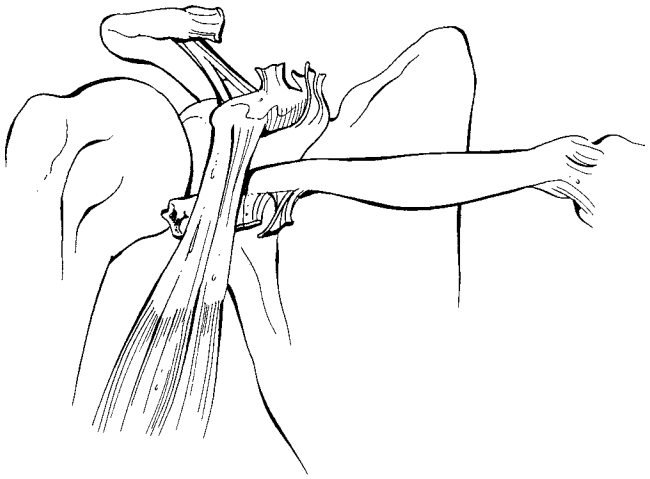


Figure 32-7 Type VI Injury.

joint, but rather, fractures of the clavicle.[26] Disruption of the periosteal sleeve of the clavicle allows the bone to displace superiorly without disruption of the coracoclavicular ligaments (Fig. 32-8). Thus, in children the classification of acromioclavicular injuries is based on the position of the clavicle with injury to the periosteal sleeve with intact ligaments.

Classification of Acromioclavicular Joint Injuries in Children

Type I injury
- Sprain of acromioclavicular ligaments
- Periosteal sleeve intact

Type II injury
- Partial disruption of periosteal sleeve
- Slight widening of acromioclavicular joint

Type III injury
- Periosteal tube disrupted with instability of the distal clavicle
- Superior displacement 25% to 100% of distal clavicle on AP radiograph

Type IV injury
- Periosteal tube disrupted
- Deltoid and trapezial detachment
- Clavicle displaced posteriorly through or into trapezius viewed on axillary radiograph

Type V injury
- Periosteal tube disrupted
- Deltoid and trapezial detachment
- Clavicle displaced subcutaneously greater than 100% of normal

Type VI
- Inferior displacement of clavicle beneath the coracoid process

Fractures of the coracoid, if through the common growth plate with the upper glenoid fossa, may mimic an acromioclavicular injury, but the coracoclavicular interspace remains intact.[63] Fracture of the base or tip of the coracoid combined with acromioclavicular ligament disruption is an atypical form of injury to this area. An axillary view will best demonstrate a fracture of the coracoid.[12,74,108] This injury should be suspected in acromioclavicular injuries in the first three decades of life.

RADIOGRAPHY

Standard anteroposterior radiography of the shoulder joint overpenetrates the acromioclavicular joint and makes

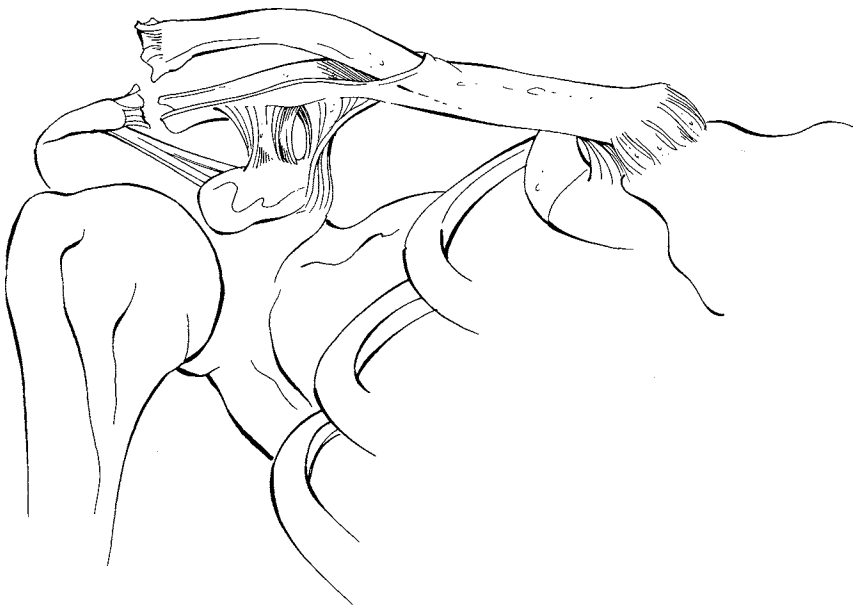


Figure 32-8 Periosteal sleeve disruption of the clavicle in a child allows super migration of the bone without disruption of the coracoclavicular ligaments.

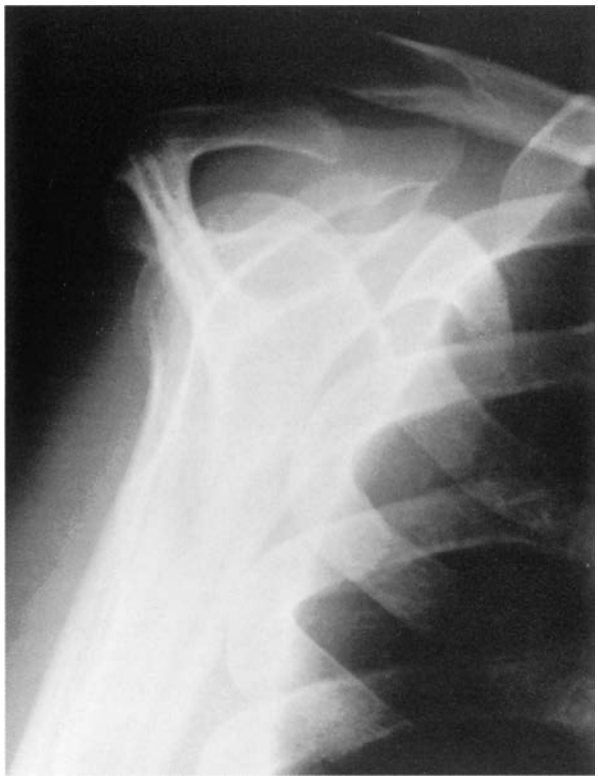


Figure 32-9 Alexander scapular lateral view with complete acromioclavicular joint dislocation.

interpretation difficult. An anteroposterior acromioclavicular joint radiograph and a 15-degree cephalic-tilt view are recommended to evaluate the acromioclavicular joint.[123] The superior tilt of the Zanca view makes sure the spine of the scapula is not superimposed on the acromioclavicular joint. Thus, subtle fractures of the distal clavicle will not be missed. Axillary lateral-view radiographs are also useful to evaluate the position of the clavicle relative to the acromion. This is particularly useful in the evaluation of type IV acromioclavicular joint injures.

Alexander described the scapular lateral radiograph as a technique to evaluate acromioclavicular joint injuries (Fig. 32-9).[1] In this view, a comparison superolateral radiograph is taken with the shoulders protracted forward. In the normal shoulder, the integrity of the acromioclavicular joint is maintained. With an acromioclavicular dislocation, the distal clavicle is displaced superiorly to the acromion.

Stress or weighted radiographs have been recommended in the past to differentiate type II from type III acromioclavicular injuries. These radiographs, with the patient seated or standing with weights (15 lb) strapped to the wrists, will accentuate the difference between the normal and injured shoulder (Fig. 32-10). Typically, a large 14 × 17-in. cassette is used to visualize both shoulders on the single plate. A comparison of the coracoclavicular space between the normal and injured shoulder is made. In large individuals, two separate plates may be required. A difference of 25% to 40% increase in space is felt to be significant for full disruption of the coracoclavicular ligaments.[9,93] It is important to have the patient relaxed or muscle forces may lift the arm superiorly and reduce the injured joint, distorting the interpretation. The same may occur if the patient is asked to hold the weights in his or her hands. The importance of these views is dependent on their use as a prognostic factor in treatment of these injuries. With the tendency to nonsurgical treatment of type III injuries, stress radiographic differentiation between type II and III injuries becomes clinically irrelevant. Others have noted low clinical yield as a reason to discourage use of stress radiographs.[15]

Our current recommendation for routine assessment of the acromioclavicular joint radiographically is an anteroposterior or Zanca view, along with an axillary view.

Computed Tomography

Computed tomography may be the best test to evaluate arthritic changes of the acromioclavicular joint including joint space narrowing, erosions, and subchondral cysts.[38] It is also ideal for demonstration of clavicular fractures, particularly those involving the distal clavicle (type II) and

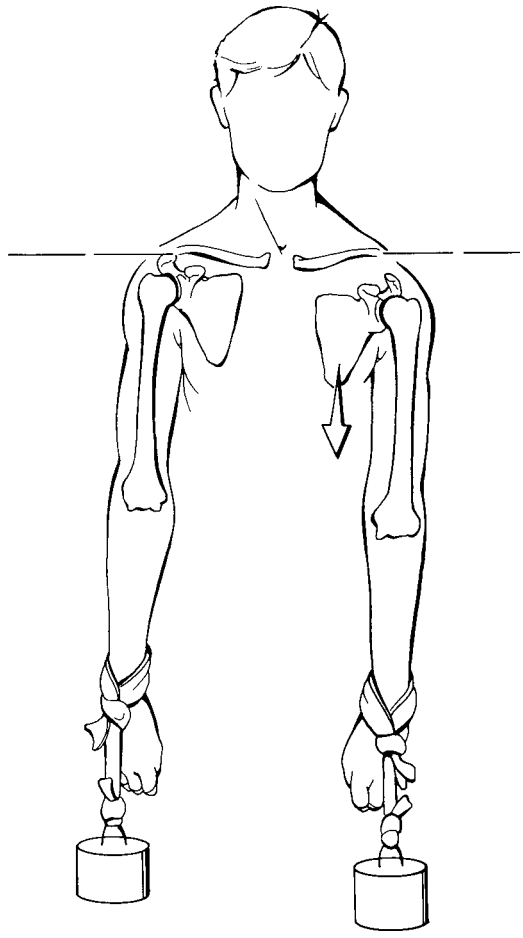


Figure 32-10 Weighted X-Ray technique.

Figure 32-11 Type IIA distal clavicle fracture.

acromioclavicular joint (Figs. 32-11 and 32-12). A benefit is its ability to be well tolerated by the multiply injured trauma patient. Its shortcoming is in its ability to show soft tissue changes including ligamentous, capsular, and synovial abnormalities. While it needs to be kept in our armamentarium, its price may make it an impractical examination for most acromioclavicular pathology.

Magnetic Resonance Imaging

Magnetic resonance imaging (MRI) is an important modality for assessment of soft tissue and bony abnormalities about the shoulder. The coronal plane best demonstrates the acromioclavicular joint.[100] Osteophyte formation and its secondary impact on the rotator cuff, along with bone edema of the distal clavicle and acromion, may be best evaluated by MRI. Stress-induced changes of osteolysis may be best seen on MRI as well as soft tissue swelling, prominence of the joint capsule, joint space widening, cortical thinning or irregularity, subchondral cystic change, and osseous fragmentation.[29,122]

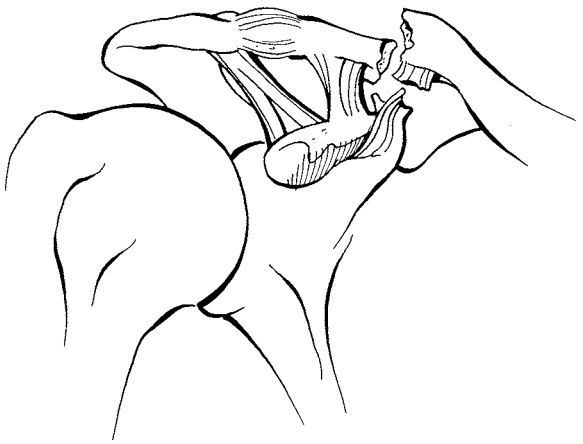


Figure 32-12 Type IIB distal clavicle fracture may mimic acromioclavicular joint disruption.

TRAUMATIC LESIONS—ACROMIOCLAVICULAR DISLOCATIONS

Injuries to the acromioclavicular joint should be suspected in anyone with trauma to the shoulder and a history demonstrating the typical mechanism of injury (Fig. 32-13). Not infrequently, the patient will present with a significant abrasion over the superior aspect of the shoulder secondary to the fall. Inspection will demonstrate swelling or prominence of the distal clavicle. It is important to remember that the prominence of the distal clavicle is actually accountable to the downward sag of the shoulder and arm, not upward displacement of the clavicle.

Initial discomfort of the injury may cause the patient to tense his or her shoulder musculature or cradle his or her arm and make the prominence of the clavicle less noticeable. As the initial swelling subsides, the deformity and prominence of the clavicle become more noticeable. Palpation will typically reveal local tenderness. In type I injuries, the only finding may be localized tenderness. Direction of instability will be difficult to discern initially because of the patient's discomfort. With time, the swelling and pain will subside and make detection of the instability easier. As

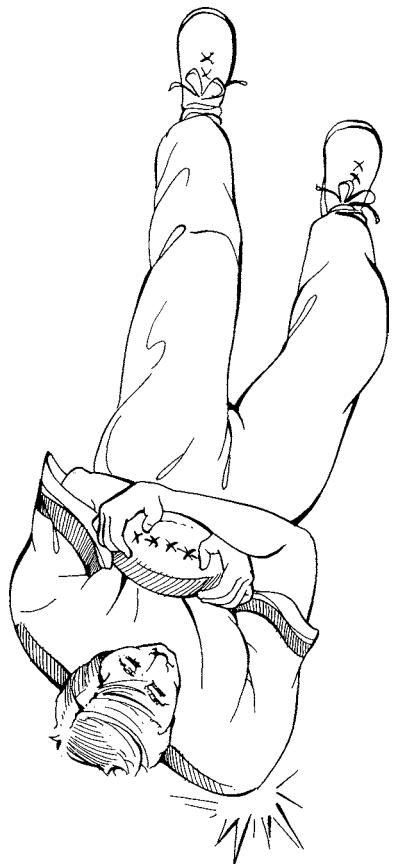


Figure 32-13 Acromioclavicular joint injuries typically are the result of a fall on the superior aspect of the shoulder.

the patient becomes more comfortable, anteroposterior and superoinferior translation of the distal clavicle can be assessed. Depending on the severity of the injury, motion of the shoulder joint is typically restricted most in abduction or cross-body adduction. With lesser injuries (type I), pain may be produced only with resisted abduction or cross-body adduction and direct palpation of the acromioclavicular joint. Rotation of the humerus with the arm at the side is generally comfortable with these injuries and can be used to detect associated injuries of the glenohumeral joint.

Type I Injury—Assessment

Because type I injury is the least severe of the injuries, generally there is mild to moderate pain and swelling. Athletes will frequently continue to participate in the athletic activity that caused the injury. Arm movements typically are comfortable except for cross-body adduction. The only obvious abnormality is tenderness to palpation.

Typically, these radiographs are essentially normal when compared with the involved shoulder. Radiographs years later may display degenerative changes at the acromioclavicular joint.

Type II Injury—Assessment

The patient will display moderate to severe pain over the acromioclavicular joint. If seen before swelling occurs, slight prominence of the clavicle is noted, best indicated by running a finger over the clavicle to the acromioclavicular joint. Anteroposterior motion of the distal clavicle can be noted when grasping the clavicle and stabilizing the shoulder. Pain may be present over the costoclavicular interspace.

Radiographic findings display slight widening of the acromioclavicular joint when compared with the normal side. Even with stress radiographs, the coracoclavicular space is maintained.

Type III Injury—Assessment

In type III injury, moderate to significant pain is usually present. Initially the prominence of the distal clavicle may be diminished by the patient cradling the arm upward to relieve pressure and discomfort on the injured joint (Fig. 32-14). Abduction of the arm or cross-body adduction increases the discomfort. With palpation, the distal clavicle feels unstable both anteroposteriorly and superoinferiorly. Typically, the patient is tender at the acromioclavicular joint as well as the coracoclavicular interspace.

The joint is totally displaced on the anteroposterior radiograph compared with normal. The axillary view fails to demonstrate anteroposterior displacement of the distal clavicle. If total acromioclavicular displacement is noted on the anteroposterior radiograph but the coracoclavicular interspace is maintained, one should suspect a fracture of the coracoid process. This can best be demonstrated by a Stryker notch view.[51,93] With this view, the patient lies supine with the cassette under the involved shoulder. The hand is placed on top of the head. The radiographic beam is tilted 10 degrees cephalad and centered over the coracoid process.

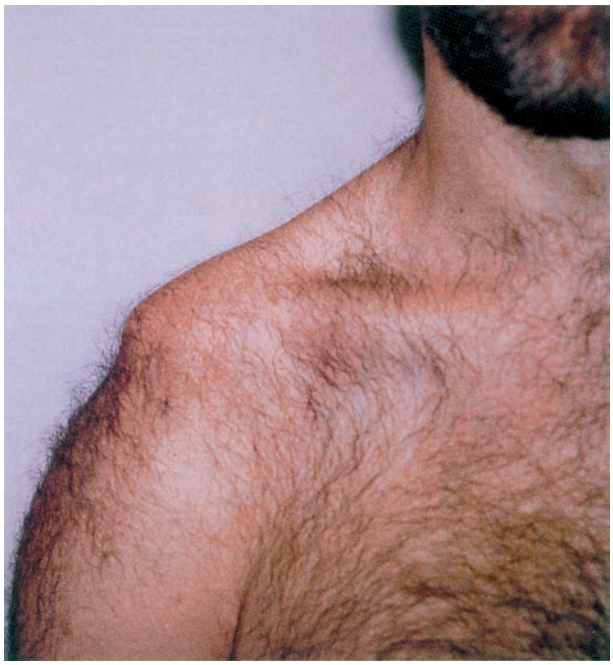


Figure 32-14 Type III injury. Prominence of the distal clavicle accountable to the downward sag of the shoulder.

Type IV Injury—Assessment

Typically, the patient is in more pain than with the more common type III injury. Palpation along the clavicle will demonstrate posterior displacement of the distal clavicle through the trapezius muscle. The clavicle does not have the "free-floating" feel of the type III injury. Once swelling subsides, observation from above and behind the patient will demonstrate posterior displacement of the clavicle in a thin patient (Fig. 32-15). Motion of the shoulder is much more painful than in the type III injury.

The anteroposterior view may show displacement of both the acromioclavicular joint and the coracoclavicular interspace. On the axillary view, posterior displacement of the distal clavicle in relation to the acromion is most pronounced. A computed tomography (CT) scan may be warranted in heavy individuals or in those with multiple injuries that make the axillary lateral radiograph difficult. The CT scan may also be used in persons for whom there is a suspicion of associated glenoid fractures.

Type V Injury—Assessment

This injury presents with findings very similar to the type III, but with more displacement of the clavicle. This generally

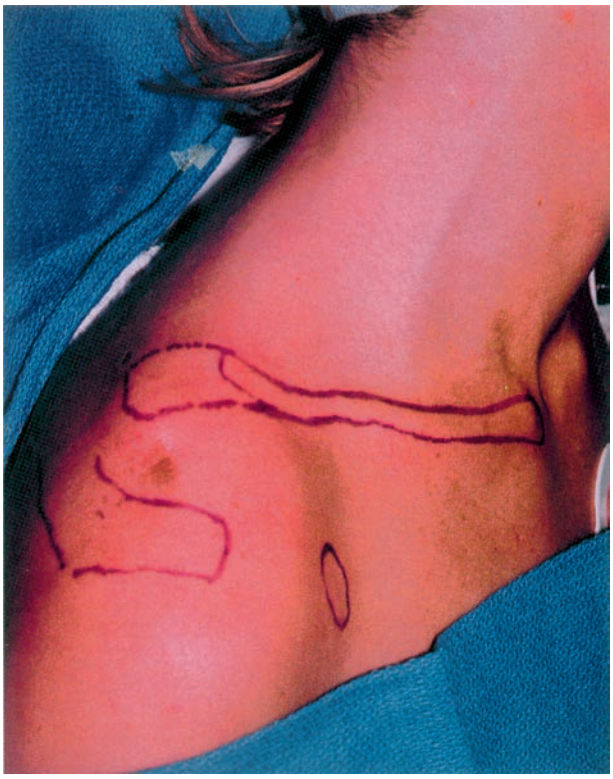


Figure 32-15 Type IV injury. Clavicle and acromion outlined with distal clavicle buttonholed through trapezius.

has more of the "ear tickler" presentation, owing to the more extensive injury to the deltoid and trapezial fascia (Fig. 32-16). Typically, the patient is in more pain than with a type III injury. The skin is severely tented and it appears that the clavicle may protrude through it.

These injuries display gross displacement of the acromioclavicular and coracoclavicular space on radiographs. The coracoclavicular space may be two to three times wider than normal.

Type VI Injury—Assessment

With this injury, the superior aspect of the shoulder appears flatter because of the downward displacement of the clavicle. The acromion is more prominent and palpable with a stepdown or depression over the distal clavicle. This rare injury may also present with neurologic changes because of proximity to the upper roots of the brachial plexus. Because of the severity of the injury, associated injuries should be suspected, including fractures of the ribs and pneumothorax, and sternoclavicular injuries.

On the anteroposterior view, the clavicle will be displaced inferior to the acromion or coracoid. Because of the magnitude of force required to produce this injury, associated fractures of the ribs or scapula should be sought.

Treatment

Although the treatment of type III acromioclavicular joint injuries remains controversial, prospective studies comparing nonoperative and surgical treatment of these injuries have yielded similar results.[5,22,99,107] Thus, the pendulum of care has swung to the initial nonoperative care of these patients. Not all patients, however, do well with conservative care. Guidelines for care at this time are not fixed and are frequently dictated by the patient's desires and goals as well as the surgeon's experience. Some authors who generally favor the conservative treatment of these injuries may recommend surgical repair in patients involved in heavy labor or overhead sports.[5,22,35,107,115,117] One study, though, demonstrated little effect on athletic strength or performance with acromioclavicular dislocation.[109] Questions still remain whether complete dislocation of the acromioclavicular joint affects performance of the overhead laborer or throwing athlete, or whether surgical procedures can fully restore normal anatomy and function.

Treatment Options and Indications

Type I—Acute

Type I sprains of the acromioclavicular joint are treated nonoperatively by all. Analgesic medication and a sling are used per patient's comfort. Cryotherapy, for periods of no longer than 15 to 20 minutes, is used to reduce swelling. Local wound care may be necessary to treat the abrasions that are frequently seen with these injuries. Early active motion and exercise to promote return to activity are recommended. Most active individuals, along with athletes, will return to their desired activity in 2 to 14 days. Athletes involved in contact sports, such as football, can add cutout pads or modify their shoulder pads to reduce contact to the joint.

Type I—Chronic

Degenerative changes at the acromioclavicular joint in athletes are frequently the result of old type I and II acromioclavicular joint sprains. Damage to the meniscus and articular cartilage with an acute injury can lead to degenerative changes later in life (Figs. 32-17 and 32-18). Pain caused by degenerative changes at the acromioclavicular joint secondary to type I sprains can be treated with mild analgesics, nonsteroidal antiinflammatory medications, or intraarticular injections of corticosteroid preparations. Those who do not respond to conservative care may require resection of the distal clavicle to provide relief of symptoms. This can be performed with the use of an open or, today, more commonly an arthroscopic approach.[42,46,47,50,76,106] The technique used depends on the surgeon's preference and experience. A potential advantage to the arthroscopic technique is the requirement for less resection of the distal clavicle (5 to 7 mm) because of the preservation of the capsular ligament.[42] Return to activity and athletic competition after

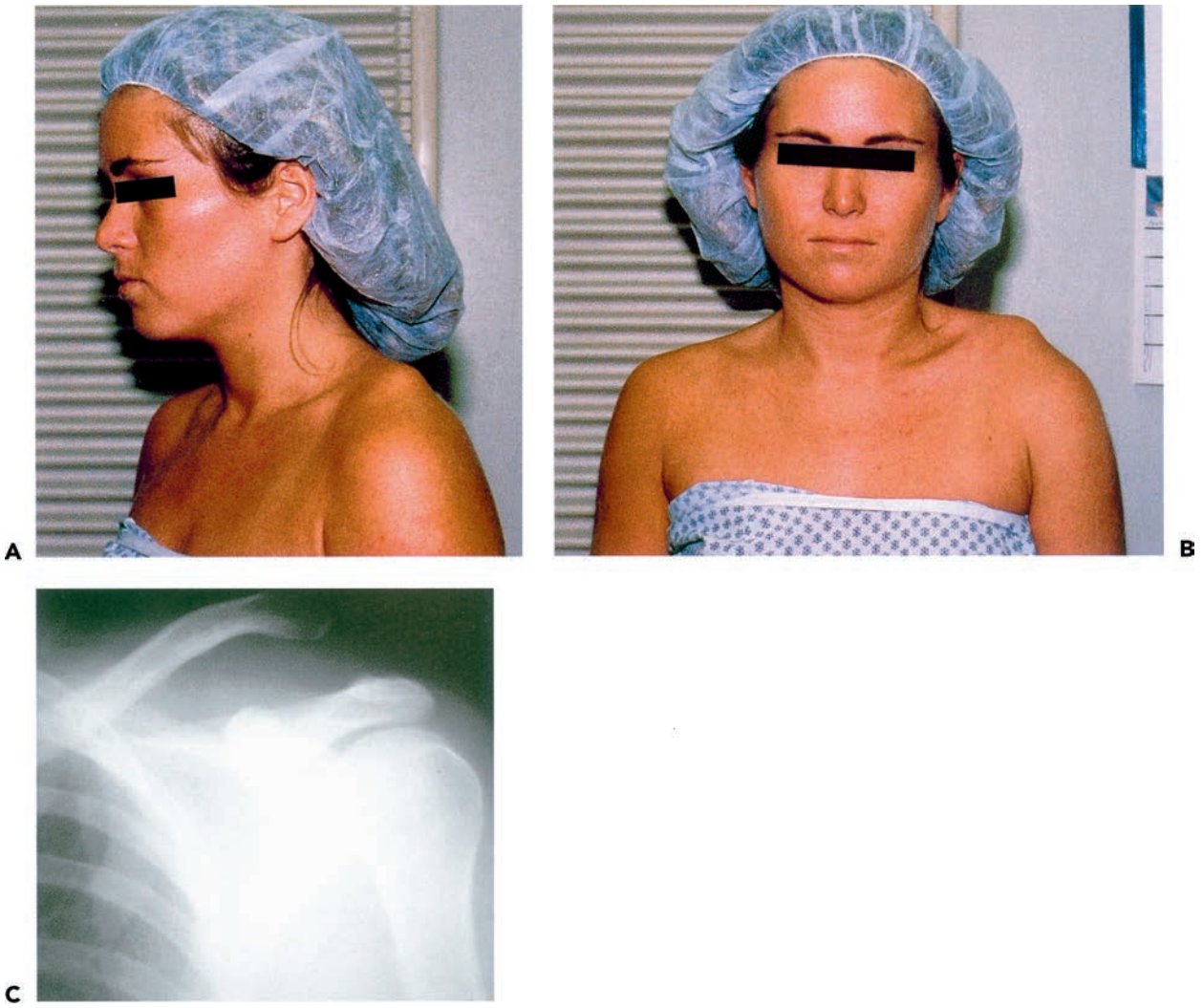


Figure 32-16 **(A,B)** Clinical photograph, and **(C)** AP nonstress radiograph of the shoulder demonstrates the typical appearance of a Type V acromioclavicular separation.

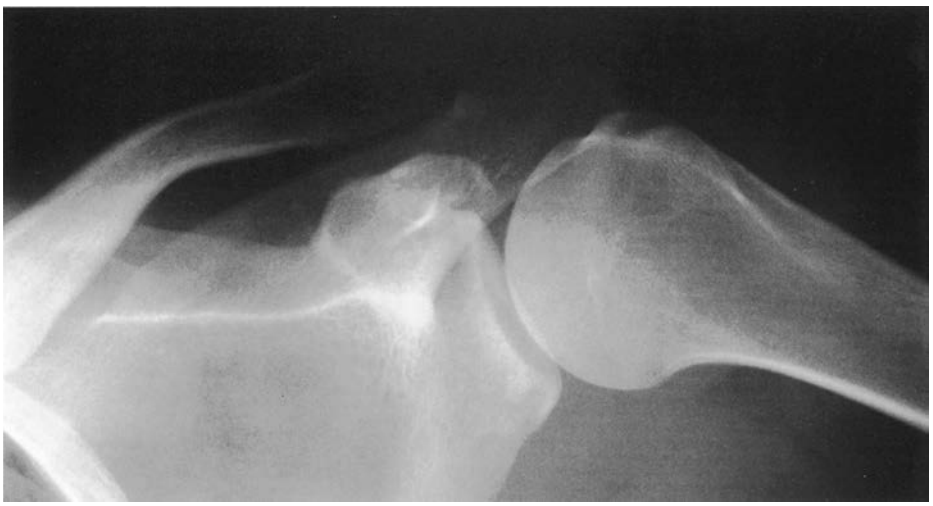


Figure 32-17 Radiograph of acromioclavicular joint at time of Type I injury.

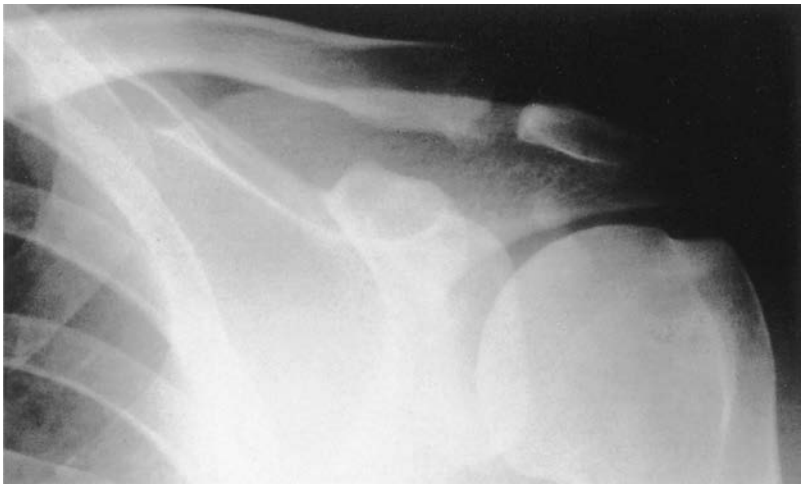


Figure 32-18 Radiograph of acromioclavicular joint 10 years after Type I injury. Note irregularity of distal clavicle.

clavicle resection is dictated by wound healing and restoration of motion, strength, and comfort.

Type II—Acute

Acute type II injuries are treated in a manner similar to the type I acute sprains: a sling, analgesic use, and icing per the patient's comfort. The potential for intraarticular damage and degenerative damage may be enhanced by the increase in sagittal plane motion of the acromioclavicular joint. Some physicians may use a sling or harness for longer periods to preclude conversion to a type III injury. Return to activity is predicted by restoration of motion, comfort, and strength. For athletes, Gladstone and colleagues recommend a four-phase rehabilitation program, as described below[34,49]:

Phase 1: Use ice, immobilization, and analgesics. Begin active assisted range-of-motion exercises, particularly internal and external shoulder rotation at low levels of abduction and forward flexion (30 to 40 degrees). Advance to phase 2 when the patient has 75% of full motion, minimal pain and tenderness on palpation of the acromioclavicular joint, and manual muscle grade of 4 or 5 of anterior and middle deltoid and upper trapezius.

Phase 2: The goals of this phase are to restore full and painless range of motion and increased strength. Rotation is continued but at greater degrees of abduction and forward flexion. Strengthening is continued for the deltoid, trapezius, and rotator cuff. Bench and military press is restricted. Advance to phase 3 when the patient has nonpainful range of motion, is nontender to palpation, and has strength 75% of normal side.

Phase 3: The goal of this phase is to restore strength to the entire shoulder girdle. Exercises emphasized include shoulder flexion, abduction, shrugs, and bench press. Progress to phase 4 when patient has full range of motion, painless motion, and nontender, normal clinical examination and isokinetic test data with close to 100% strength.

Phase 4: Progress with sports-specific exercises and throwing.

Type II—Chronic

In patients with pain and degenerative change of the acromioclavicular joint, the treatment recommended is similar to type I injuries. Analgesic medication, nonsteroidal antiinflammatory medications, and injection with corticosteroid medications seldom offer long-term relief. An evaluation of patients after isolated resection of the distal clavicle has noted some poor results attributed to abutment of the posterior clavicle on the acromion. Symptomatic type II acromioclavicular joint injuries can be treated as type III injuries, with resection of the distal clavicle along with reconstruction of the ligamentous structures. Typically reconstruction involves transferring the coracoid ligament to the resected clavicle.[113] This enhances superior–inferior stability of the joint, but provides little anteroposterior stability. Recently, techniques that recreate both the acromioclavicular and coracoclavicular ligaments with either autogenous graft (semitendinosis) or allograft have been suggested.[31,34,56] Work by Lee et al. in a cadaver model showed that shoulder extension produced forces across the coracoclavicular ligaments that exceeded the ultimate tensile strength of a Weaver-Dunn reconstruction.[67]

Type III Injuries (Fig. 32-19)

The trend in the treatment of these injuries remains a more conservative approach. A distinct advantage of surgical treatment over conservative care has never been clearly demonstrated. When comparing operative and nonoperative techniques pain, range of motion and persistent symptoms are very similar.[34,66,88] When considering a surgical approach to treatment, the surgeon must ask himself or herself whether today's surgical options truly restore the anatomy and function of the acromioclavicular joint. The

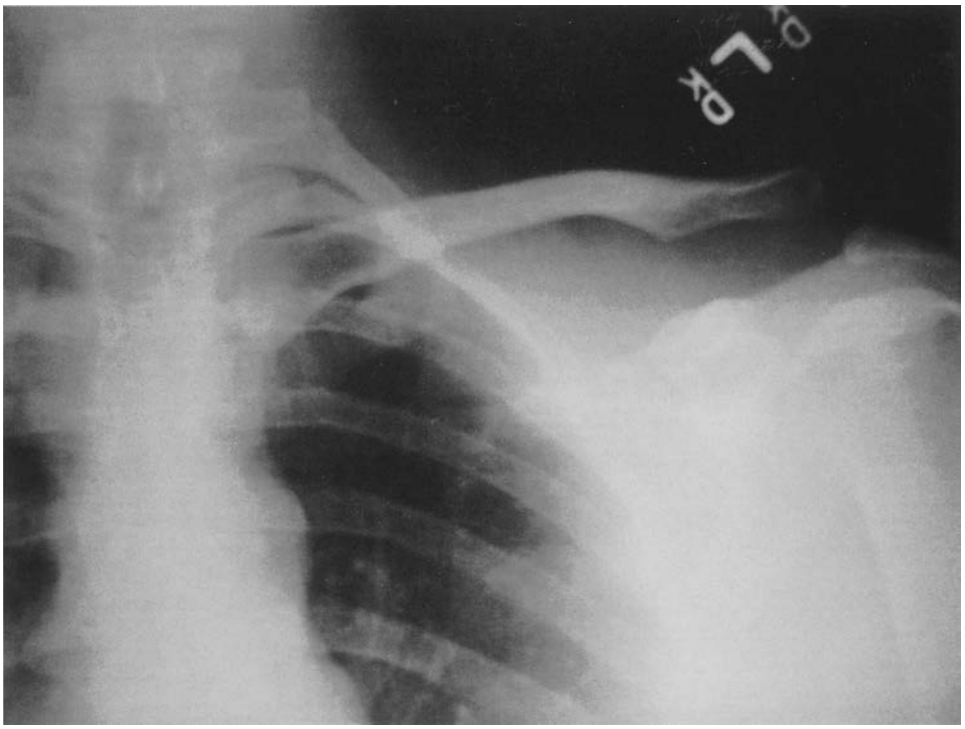


Figure 32-19 Complete displacement of the acromioclavicular joint.

authors have conducted an informal survey of physicians involved in the care of professional athletes involved in the sports of football, hockey, and baseball and found that the majority favor a nonoperative approach to the management of these injuries. However, these physicians would consider operative reduction for the throwing athlete's dominant extremity, particularly the baseball pitcher. Quarterbacks throw with a less specific motion and generally do not fall into the same consideration. A recent prospective study by Schlegel found little difference in the outcomes of type III injuries, compared to normals, treated only using a sling for comfort and active range of motion without formal physical therapy when looking at eventual strength and comfort.[99] This study did not look specifically at athletes.

For those electing a surgical reconstruction, the timing of the repair may be an issue. One consideration is to delay the operation to see if the patient remains symptomatic. Even the newer surgical reconstructions can be performed as a delayed procedure. Weinstein et al., however, noted a trend toward better results when the surgery was performed in the first 3 weeks after injury.[115] Others have found no significant difference between early and late ligamentous reconstruction.[35] Whether the surgeon's preference is operative or nonoperative, there are certainly those patients that will require surgical reconstructions after the failure of conservative care.

Nonoperative care of type III acromioclavicular joint injuries is similar to that for type I and II sprains. Analgesia, icing, and a sling for comfort are also used. Because of the pain and deformity, it is unlikely that the individual will return to heavy labor or athletic activity as soon as after a type I or II injury. Professional football or hockey players, though, will frequently return to their sport within a few days to weeks.

Correction of the deformity by a brace or cast has fallen out of favor and become mostly of historical interest. Kenny Howard, a trainer at Auburn University, along with Dr. Jack Hughston, developed a brace for this purpose (Fig. 32-20).[3] This device, known as a Kenny Howard brace (Fig. 32-21), combines a sling with a strap over the distal clavicle that applies downward pressure on the clavicle while providing a superior force on the humerus. To be effective, the brace must be worn continuously for 6 to 8 weeks while healing occurs. Any displacement of the brace during this period will lead to loss of reduction and subsequent failure. The brace can be quite cumbersome to wear and even painful. Skin maceration and breakdown is a risk, and there is even a report of an athlete who sustained an anterior interosseous nerve compression while using the sling.[84]

Our approach to the treatment of type III acromioclavicular injuries is to treat these injuries nonoperatively, with surgical stabilization being considered only if an athlete or laborer places significant demands on the upper extremity. If conservative treatment fails, stabilization is considered later in those who continue to note symptoms with activity. Excellent results can be obtained with late reconstruction.

Various surgical options have been described and are being expanded upon. They will be detailed later in this chapter. These options include arthroscopic and open reconstructions. The open options include dynamic muscle transfers, primary acromioclavicular joint fixation,

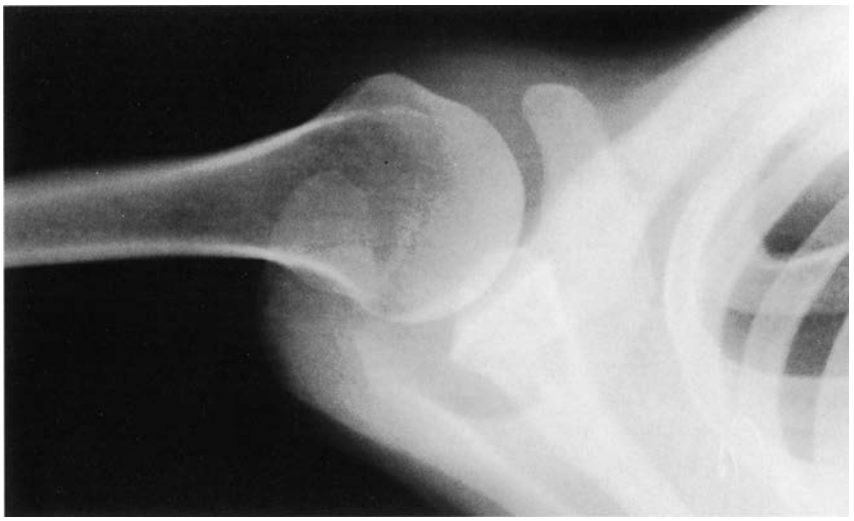


Figure 32-20 Axillary radiograph demonstrating posterior displacement of the distal clavicle in relationship to the acromion.

primary coracoclavicular ligament fixation, excision of the distal clavicle with or without coracoclavicular ligament reconstruction, and reconstruction of both the acromioclavicular and coracoclavicular ligaments with autogenous or allograft tissue. Many of these options include augmentation of the reconstructions with soft tissue grafts or artificial tapes or sutures.

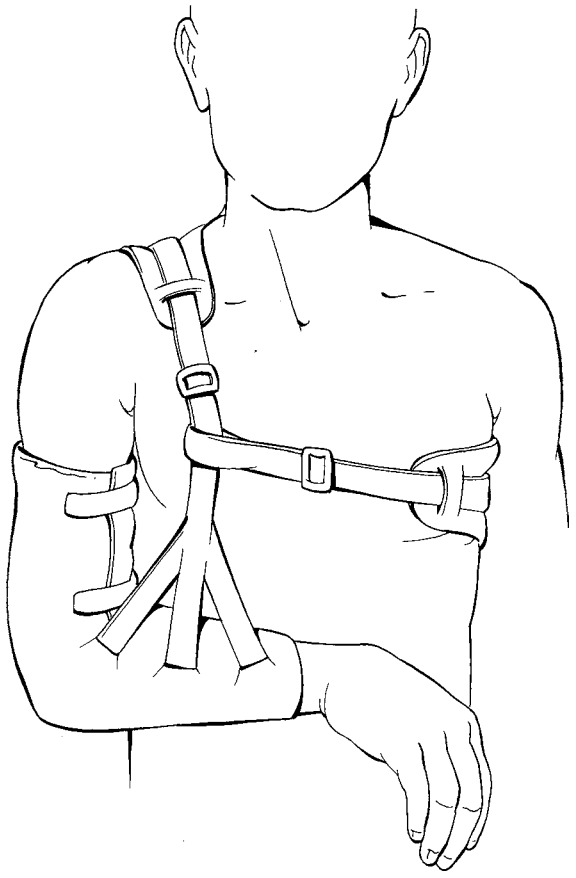


Figure 32-21 Kenny Howard brace.

Type IV Injuries

We agree with most authors that posterior displacement of the clavicle through the trapezius will lead to discomfort with motion. Thus, these injuries should be treated with the goal of reducing the deformity. Options include an attempt at closed reduction to displace the clavicle from its buttonholed position in the trapezius, and converting the injury to a type III and then treating it conservatively. The most common recommendation would be open reduction and fixation by one of the surgical options outlined with type III injuries. Our recommendation would be to reduce the joint operatively because the displacement involves significant stripping of the deltotrapezial fascia. Meticulous closing of the deltotrapezial fascia over the repair augments the procedure.

Type V Injuries (Fig. 32-22)

We feel that many type V injuries are actually treated as type III injuries and may do quite well with conservative care. Most authors feel that these injuries imply a greater amount of damage to the ligamentous structures. Type V injuries may require operative reduction because of the significant stripping of deltotrapezial fascia and potential compromise of the overlying skin. These injuries are probably best treated with a reconstruction that addresses the coracoclavicular ligament, acromioclavicular ligament, and deltotrapezial fascia disruption. We favor a procedure that excises the distal clavicle because of concern for later degenerative changes at the acromioclavicular joint and the potential for discomfort or rotator cuff impingement. Excision of the distal clavicle has not been shown to compromise muscle strength.[109]

Type VI Injuries

The potential for closed reduction of such an injury is remote. These are rare injuries that require open reduction and stabilization. Excision of the distal clavicle will aid in the reduction of the acromioclavicular joint.[48]

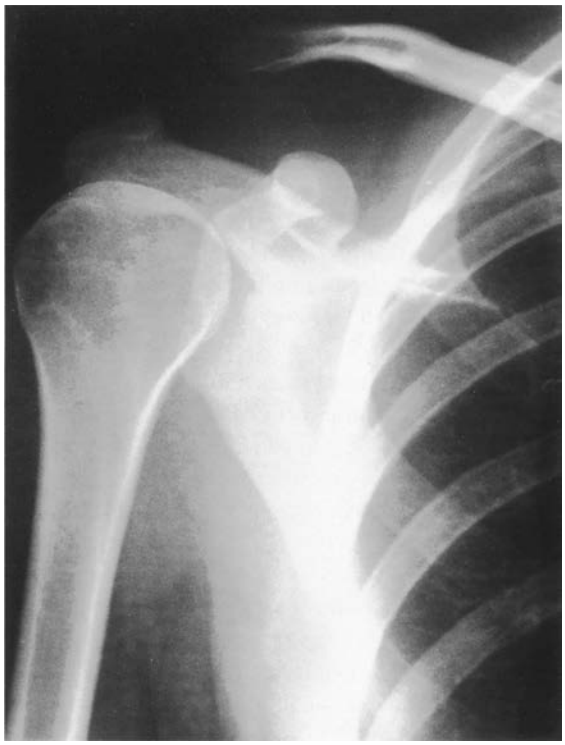


Figure 32-22 Type V injury. Note gross displacement of acromioclavicular and coracoclavicular spine.

Injuries in Children

Type I, II, and III injuries in children are treated similarly to those in adults. Use of a sling, ice, and mild analgesics are all that are required. These injuries can be expected to go on and heal without sequelae.[37,53] Markedly displaced type IV, V, and VI injuries should be treated surgically. Replacement of the clavicle into its periosteal sleeve, suturing the sleeve closed, and then fixation with a coracoclavicular lag screw or transacromial fixation are generally recommended.[26,85] After 4 to 6 weeks the fixation will have to be removed before starting rehabilitation.

Type II Equivalent Injuries

A fractured coracoid along with an acromioclavicular dislocation is an exceedingly rare injury. It is easy to overlook the coracoid fracture, particularly in light of the more obvious acromioclavicular joint disruption. Most occur in the second or third decade of life. Review of the literature would indicate that these injuries do well when treated conservatively or surgically. Thus, we would recommend a conservative approach and avoid potential complications of surgery.[12,55]

Surgical Treatment of Acute Acromioclavicular Joint Dislocation

A variety of surgical approaches have been described to manage these injuries and restore the anatomic relations of the acromioclavicular joint.[5,35,40,56,62,65,103,107,115] The question remains whether these techniques are effective at restoring normal functional anatomy and, therefore, offer a distinct advantage over a conservative approach.

Surgical Options

Many different operative procedures have been described to repair and stabilize acute acromioclavicular dislocations (types III, IV, V, and VI). Some can be performed arthroscopically or via an open surgical technique. The most popular options include dynamic muscle transfers, primary acromioclavicular joint stabilization, primary coracoclavicular and extraarticular stabilization, excision of the distal clavicle with coracoclavicular ligament reconstruction, or more recently reconstruction of both the acromioclavicular ligaments and coracoclavicular with autogenous or allograft tissue. The goal of each of these procedures is to reduce the joint and create an environment for soft tissue healing and stabilization of the distal clavicle.

Dynamic Muscle Transfers

The clavicle may be stabilized by transfer of the tip of the coracoid process and its attached tendons of the coracobrachialis and short head of the biceps to the undersurface of the clavicle.[13,41,104] Theoretically, this transferred tendon acts as a dynamic depressor of the clavicle and holds the acromioclavicular joint reduced. Ferris et al. reviewed 20 patients managed with a dynamic transfer and found nearly half with continued aching at the acromioclavicular joint.[41] These results are most likely due to the lack of distal clavicle stability and excessive motion. Possible complications of this surgical procedure include the risks of nonunion or injury to the musculocutaneous nerve.

Primary Acromioclavicular Joint Stabilization

The dislocated acromioclavicular joint can be stabilized by transfixing it with Kirschner wires or Steinmann pins or screws (Fig. 32-23). This treatment typically has been performed in conjunction with repair of the acromioclavicular or coracoclavicular ligaments.[95] Technically, transfixion of this joint is demanding because of the thin acromion and the curved nature of the clavicle. Other concerns include the necessity for hardware removal and the risk of pin breakage and migration.[71] In addition, surgical trauma to the meniscus and articular cartilage may occur and lead to the subsequent development of degenerative arthritis of the acromioclavicular joint.[107] There has been recent interest in the use of bioabsorbable materials to transfix the acromioclavicular joint. The potential advantage of these materials is that they provide fixation of the joint, without the necessity of a second procedure for hardware removal.

Recent studies have advocated the use of a specialized hook plate to stabilize the acromioclavicular joint.[40,103,119] Described in the 1980s, the plate provides stability to the

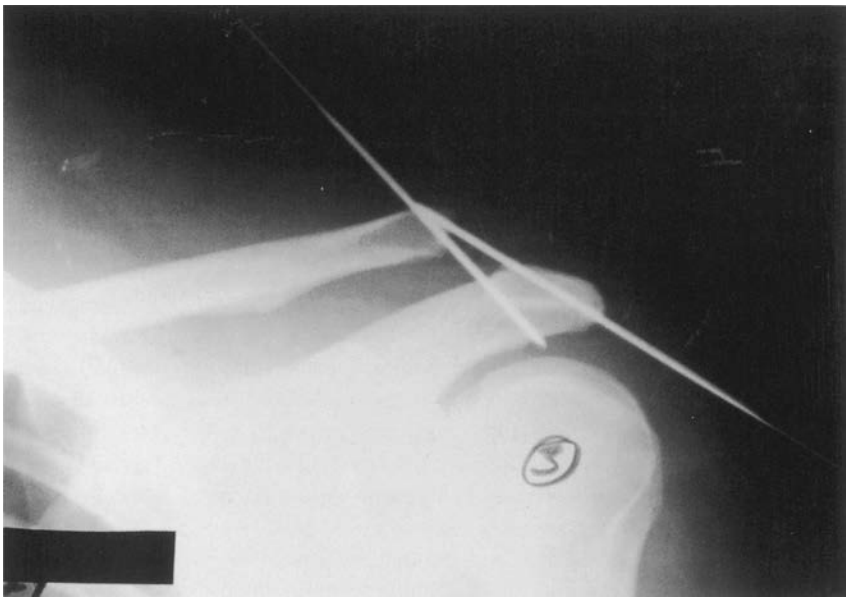


Figure 32-23 Fixation failure: Transfixion of acromioclavicular joint is demanding due to the thin acromion and curvature of the clavicle.

acromioclavicular joint but is a technically demanding procedure with a high complication rate that may make it impractical. It also requires a secondary removal at a later date. Studies of small groups of patients showed high incidence of infection and wound healing problems (up to 28%) with the plate such that common usage of this plate should not be undertaken.

Primary Coracoclavicular and Extraarticular Stabilization

Stabilization of the distal clavicle can be achieved by securing the clavicle to the coracoid process of the scapula. Bosworth described a technique in which he placed a screw through the clavicle into the base of the coracoid (Fig. 32-24).[16] Initially, the screw was placed percutaneously and the acromioclavicular joint was not explored or débrided. The Bosworth screw technique is often accompanied now by joint exploration and repair of the coracoclavicular ligaments.[58,59] Repair of the deltotrapezius fascia is also accomplished during this procedure. Screw removal is typically considered between 8 and 10 weeks postoperatively. Ossification between the coracoid and the clavicle occurs with some frequency and has not generally caused a problem with shoulder motion.[58]

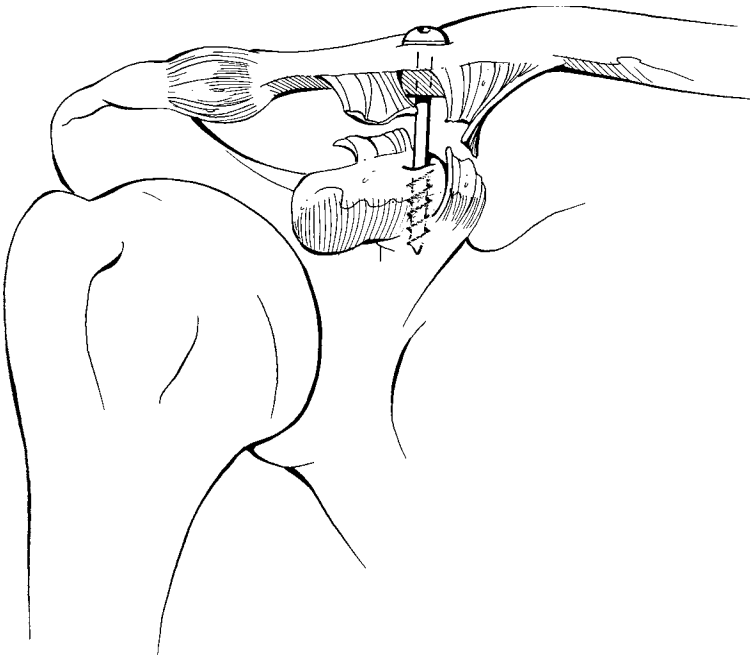


Figure 32-24 Screw fixation of clavicle to base of coracoid.

Other forms of extraarticular stabilization eliminate the need for screw removal. Cerclage fixation of the clavicle to the coracoid has been accomplished using wire,[2,9] Dacron graft, or other synthetic tape[57,78,86]; braided bioabsorbable suture; or, more recently, autologous or allograft hamstring tendon.[56] Typically, coracoclavicular fixation is accompanied by repair of the coracoclavicular ligaments, acromioclavicular joint, and deltotrapezius fascia. Use of nonabsorbable Dacron tape has been associated with erosion through the clavicle, failure of fixation,[27,45] and late infection.[79]

Accurate reduction of the acromioclavicular joint is difficult, but critical, particularly when the distal clavicle is not excised. Morrison and Lemos documented the need to accurately determine the subcoracoid and transclavicular positions of the fixation device.[75] They determined that the loop should be placed at the base of the coracoid and then inserted through a hole at the junction of the anterior and middle third of the clavicle. They felt that if the loop is posterior or goes around the clavicle, it tends to displace the clavicle anteriorly.

Excision of the Distal Clavicle and Coracoacromial Ligament Reconstruction

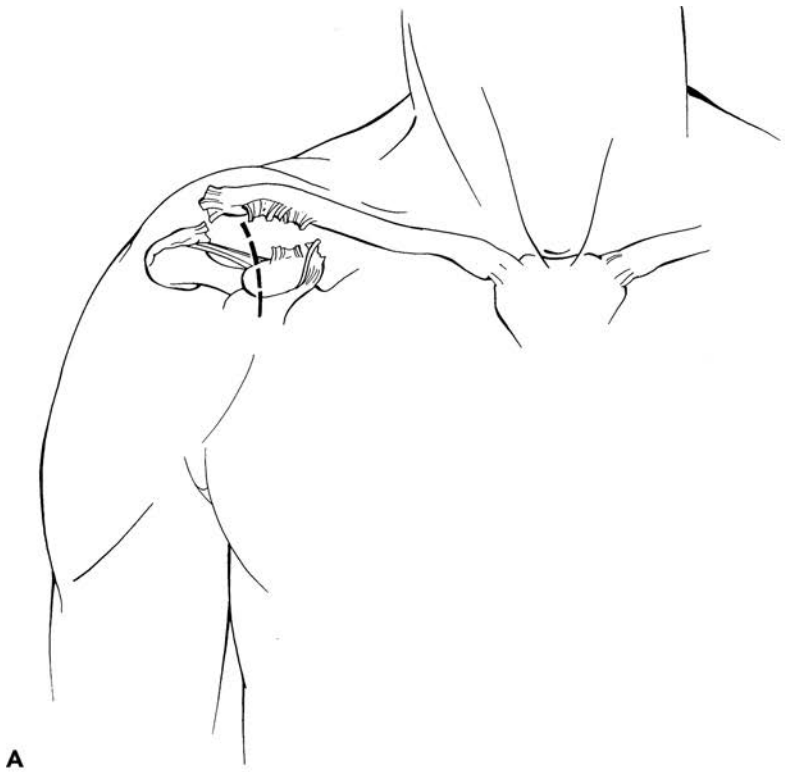
In 1972, Weaver and Dunn described a technique they used to treat acute and chronic acromioclavicular joint dislocations.[113] The surgical procedure involved excision of the distal clavicle, followed by transfer of the coracoacromial ligament to the resected end of the distal clavicle. Many variations and modifications of this procedure have been used when reconstruction of the joint is performed.[19,35,115] The decision of whether to preserve or excise the distal portion of the clavicle is based on concern over the ability to completely reduce the acromioclavicular joint and the possibility for development of degenerative arthritis. In evaluating acromioclavicular joint and coracoclavicular ligament repair, Smith and Stewart found no difference in symptoms, strength, or function, but did find a higher incidence of degenerative changes when the distal clavicle was not excised.[105] Symptomatic degeneration of the acromioclavicular joint has been effectively managed by excision of the distal clavicle.

Recent concern that this reconstruction only addresses the coracoclavicular ligaments has led to techniques that address both the coracoclavicular ligaments and their individual components (trapezoid and conoid) along with the acromioclavicular ligaments. Recently, Krishnan et al. presented their results with a technique undertaken in nine patients with type V dislocations.[62] The technique involves (a) distal clavicle resection; (b) intramedullary transfer of the coracoacromial ligament; (c) coracoclavicular reconstruction using an absorbable suture braid and ipsilateral semitendinosis graft through a drill hole in the clavicle and figure-eight around the coracoid; and (d) acromioclavicular joint superior capsular reconstruction with a remaining semitendinosis graft fixed with transosseous sutures to the acromion and posterior clavicle. All patients before the surgery demonstrated symptomatic posterior translation of the distal clavicle relative to the acromion. All patients did well at 22 months based on the American Shoulder and Elbow Surgeons (ASES) score, pain, and function. No patients demonstrated symptomatic posterior translation of the clavicle at follow-up.

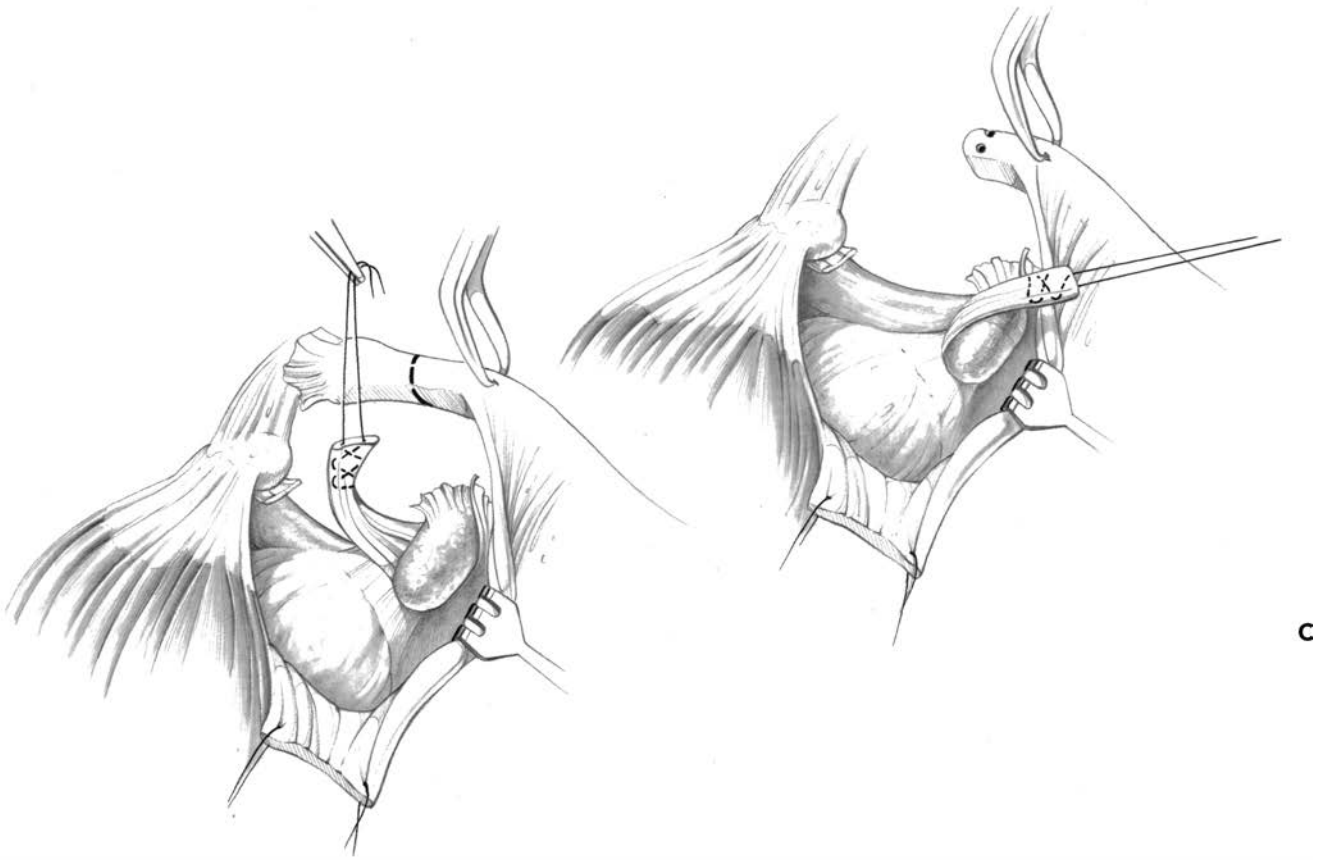
Open Technique

Our preference for reconstructing a disruption of the acromioclavicular joint is to address the pathology as completely as possible. This entails stabilizing the resected distal clavicle by transferring the coracoacromial ligament to it and supporting the repair using a braided absorbable suture along with an autogenous or allograft semitendinosis tendon. We also believe that an attempt should be made to reconstruct the acromioclavicular ligaments utilizing the semitendinosis graft. If possible, we also repair the coracoclavicular ligaments and the deltotrapezial fascia. In our opinion, the only significant surgical variable in the acute injury is deciding whether to preserve or excise the distal clavicle. We prefer its excision because of the difficulty in obtaining a perfect reduction of the acromioclavicular joint. We are concerned that the injury or incomplete reduction may lead to the development of degenerative arthritis. Pain also may occur from the degenerated joint or from inferior projecting spurs that cause mechanical impingement on the rotator cuff. No significant deficits in muscle strength were observed in athletes who have undergone excision of the distal clavicle.[109] Therefore, it may be advantageous from both a short- and long-term perspective to excise the distal clavicle as part of the reconstructive procedure.

Patients are positioned in the beach-chair position with most of the scapula and shoulder draped free. The shoulder is approached through a vertical incision from the coracoid to the posterior border of the clavicle (Fig. 32-25a). The incision passes approximately 2 cm medial to the acromioclavicular joint. The incision is planned and subcutaneous tissue undermined laterally so that the distal clavicle and anterior acromion can be reached. Next, the deltotrapezius fascia is incised along the clavicle using the Bovie electrocautery. In type IV and V acromioclavicular joint dislocations, the deltotrapezius fascia may be disrupted, and this opening should be extended as necessary. The dissection should be performed to carefully maintain a deltoid flap for later repair. As the incision in the fascia is carried laterally to the acromion, care is taken to dissect beneath the deltoid to expose the coracoacromial ligament. Once the coracoacromial ligament is completely defined, it is released from the undersurface of the acromion, with care to maximize its length. We place two independent no. 2 nonabsorbable sutures, usually Fiberwire, in the ligament to secure it to the distal clavicle.



A



B

C

Figure 32-25 **A-G:** Surgical steps as outlined in this chapter: **(A)** The vertical skin incision extends approximately three inches from the coracoid process to the posterior aspect of the clavicle. It passes about 2 cm medial to the acromioclavicular joint. **(B)** The deltoid and trapezius fasciae are carefully maintained as flaps. The coracoacromial ligament is dissected and released from the undersurface of the acromion. Two sutures are placed in the ligament in order to secure it to the clavicle. **(C)** The distal 1-1.5 cm of the clavicle is excised and the intramedullary canal is curetted open. Two drill holes are placed along the cut end of the clavicle from the superior surface into the canal.

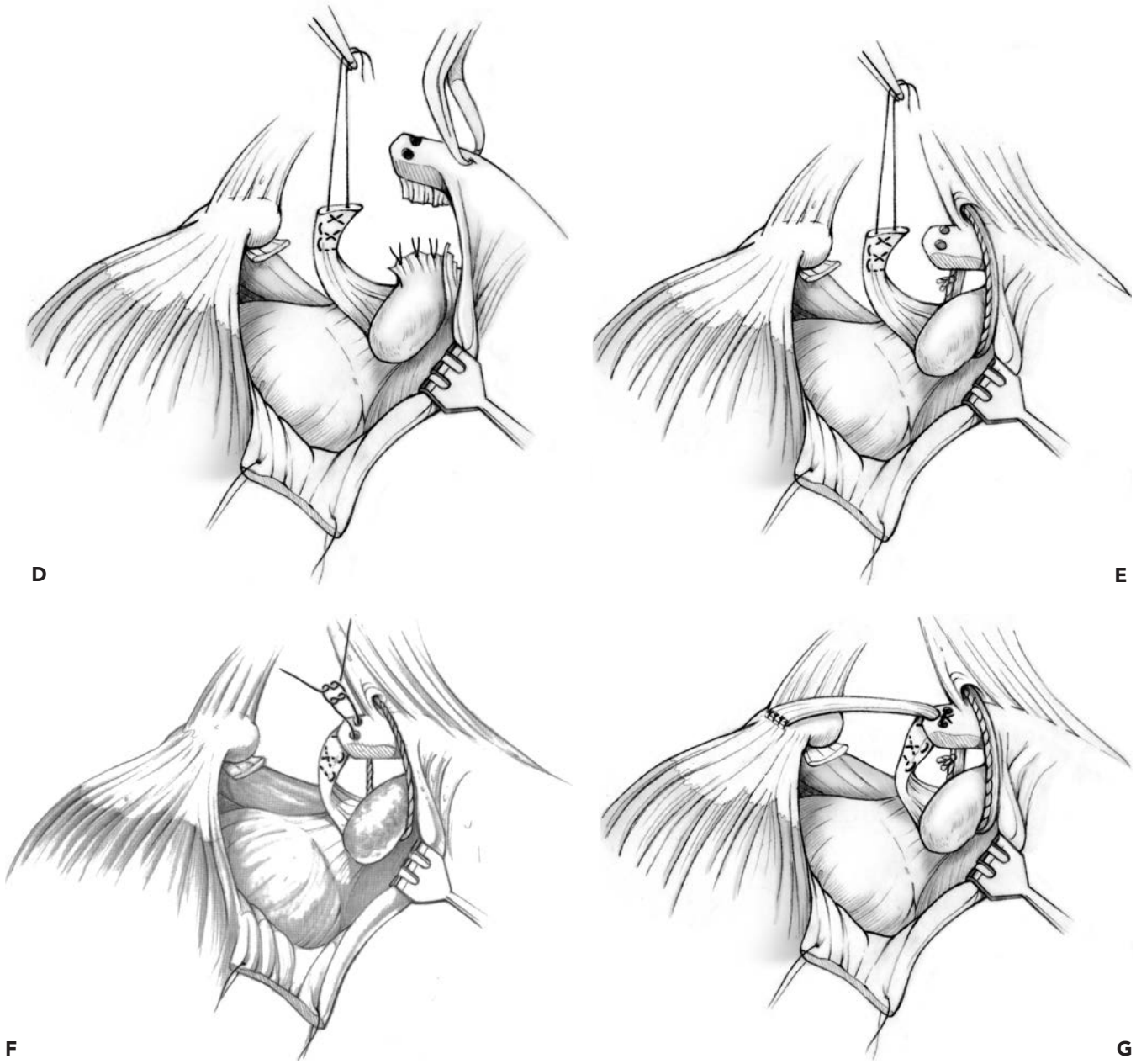


Figure 32-25 (*continued*) **(D)** The coracoclavicular ligaments are identified and tagging sutures are placed. **(E)** Braided PDS sutures are placed beneath the base of the coracoid process and through drill holes in the clavicle. Semitendiosis tendon is used along with PDS suture. Knots are tied inferior to the clavicle. **(F)** The coracoclavicular loop of suture is tied first. Second, the sutures from the coracoacromial ligament are tied. Finally, the coracoclavicular ligaments are repaired. **(G)** A piece of the semitendinosis tendon is used to reconstruct the superior acromioclavicular capsule.

Typically, one suture is placed in a Bunnell fashion and the other using a Kessler-type stitch. If the coracoacromial ligament is felt to be insufficient, we add an 8- to 10-mm strip of the conjoined tendon and turn it superiorly.

At this point, the distal clavicle is exposed subperiosteally from beneath the trapezius muscle and fascia. We excise the distal 1 to 1.5 cm of the clavicle using a sagittal saw. A curette is used to hollow out the intramedullary aspect of the clavicle. A 1.6-mm drill bit is used to create two drill holes along the superior surface of the cut end of the clavicle entering into the intramedullary canal. Looped passing sutures are then passed through each of these holes, which are used later to facilitate passage of the sutures placed in the coracoacromial ligament. Care is taken to place the holes back from the cut end of the clavicle and provide a secure bony bridge between drill holes.

After the coracoacromial ligament is prepared for transfer, we dissect the torn coracoclavicular ligaments and reconstruct them as much as possible. Although most disruption occurs in the midsubstance, avulsion from the bone is occasionally encountered. These injuries are more easily sutured and repaired back to bone. Typically, we place tagging-type sutures, as these are tied in the final step of the procedure before closing the deltotrapezial fascia.

The third stage of the procedure involves placing a loop of absorbable suture beneath the coracoid and over or through drill holes in the clavicle. We currently also augment this reinforcing suture with a strand of autogenous or allograft semitendinosis tendon placed through drill holes in the clavicle and looped in a figure-eight fashion around the coracoid. The superior surface of the coracoid is palpated and its medial and lateral aspects are defined. We use a combination of a right-angled clamp and curved Satinsky vascular to carefully dissect beneath and around the coracoid process. Once the end of the clamp is visualized, we grasp the looped end of a suture and pull it around the coracoid process. The position of the clavicle directly superior to the coracoid is now identified. This area of the clavicle is usually still covered by adherent trapezius muscle. Two drill holes wide enough to pass a loop of semitendinosus tendon are drilled in the clavicle just over the coracoid process and are made in the anterior third of the clavicle. A looped suture is passed through the holes and held with a hemostat.

The looped sutures around the coracoid and through the anterior third of the clavicle are used to pass the suture of choice along with the hamstring tendon for final fixation. We have not used nonabsorbable suture or tape, owing to concerns over risks of infection and erosion through the clavicle. If available, a 5-mm tape of polydioxanonsulfate (PDS) is used; otherwise, strands of readily available no.1 PDS are braided together. We usually braid three strands of PDS together, use two of these braids, and tie them independently. With the advent of using a hamstring tendon to secure the repair, probably only one braid of PDS is necessary. Beginning with these coracoclavicular sutures and the hamstring augmentation, the first part of the repair is secured. The knots are tied so that they end up inferior to the clavicle and do not interfere with closure of the deltotrapezial fascia. The hamstring augmentation is secured to itself with nonabsorbable sutures. Secondly, the two coracoacromial ligament sutures are passed through the drill holes in the distal clavicle and tied independently. Next, the coracoclavicular ligaments are repaired as completely as possible. The next part of the repair involves using a strip of semitendinosus tendon to reconstruct the superior acromioclavicular capsule. This reconstruction can use transosseous sutures on the acromion and the superior sutures used to tie the coracoacromial ligament into its osseous tunnel. Finally, the deltotrapezial fascia is carefully repaired over the clavicle. This not only covers the different parts of this reconstruction, but also reinforces and supports the repair.

Postoperatively, the arm is supported in a sling for 4 weeks. Motion exercises, including pendulums and gentle active elevation, are begun at 2 weeks. Progressive range-of-motion and strengthening exercises are not begun until 6 weeks after surgery. Heavy weightlifting, pushing, pulling, and other stressful activities are delayed until 3 months, and contact sports are allowed at 5 to 6 months.

Results

A careful review of the literature fails to demonstrate a clear choice from among the many different procedures, modification, and combinations that have been described. While most procedures give satisfactory results, there definitely seems to be a trend toward the more physiologic procedures that address both sets of ligaments (coracoclavicular and acromioclavicular). There are few comparison studies, as most authors report the results of a given procedure. Kennedy has reported on a modification of the Bosworth technique that includes a débridement of the acromioclavicular joint and repair of the trapezius and deltoid fascia.[58] He found that even when a bony bridge developed between the clavicle and the coracoid process, function and range of motion were maintained. Acromioclavicular and coracoclavicular fixation have been compared in several studies.[8,64,105,107] Coracoclavicular fixation has generally been favored because of its lower complication rate and overall superior results. In addition, a higher degree of late degenerative arthritis has been noted with acromioclavicular fixation.[105,107]

Weaver and Dunn described their procedure and results in 12 patients with acute type III acromioclavicular injuries.[113] Rausching et al. reported similar good results using this procedure, based on joint stability, pain, and function.[90] Browne et al. reviewed a group of patients with coracoclavicular fixation, with and without resection of the distal clavicle, and failed to demonstrate an advantage to distal clavicle excision.[19] Smith and Stewart also looked at the effect of distal clavicle excision in patients treated with acromioclavicular fixation and coracoclavicular ligament repair.[105] They observed good results except for a higher incidence of degenerative acromioclavicular joint changes when the distal clavicle was excised.

Recently, studies have indicated that the classic Weaver-Dunn procedure may not support some of the forces required during motion of the upper extremity. Lee employed a cadaveric model to demonstrate that shoulder extension produced forces across the coracoclavicular ligaments that exceeded the tensile strength of the classic Weaver-Dunn procedure. Thus, the current trend is reconstructive procedures that augment the coracoclavicular reconstruction with hamstring tendon and also address the acromioclavicular ligaments and capsule. Krishnan et al.[62] recently reported their results with the new reconstructive procedure that addresses both sets of ligaments. In nine

patients with symptomatic type V joint dislocations, the final ASES score was 96, active anterior elevation averaged 163 degrees, external rotation was 58 degrees, and internal rotation was T10. No patient demonstrated posterior translation of the clavicle asymmetric to the normal side. Pain averaged 0.4 (0 = no pain; 10 = worst pain) and satisfaction averaged 9.6 (10 = totally satisfied) in these patients.

Arthroscopic Treatment of Acute and Chronic Acromioclavicular Joint Dislocation

All arthroscopic procedures for stabilization of an acromioclavicular joint dislocation are evolving but have not yet become widely used. An arthroscopic technique permits minimally invasive access to the acromioclavicular joint without detachment of the deltoid or trapezius. The postoperative recovery is proposed to be faster, although this has not been tested in a comparative trial. Wolf[118] was the first to develop an arthroscopic technique using a drill guide to place a transcoracoid–transclavicular loop of suture material to stabilize the distal clavicle and reproduce the coracoclavicular ligaments to form an artificial ligament. This arthroscopic technique is combined with a mini-open technique for distal clavicle resection and tying the suture material.

All-Arthroscopic Acromioclavicular Joint Stabilization Technique

The procedure is performed with the patient in the beach-chair position under interscalene block with the arm maintained by an arm holder. Four portals are used: the standard posterior for the glenohumeral joint, the lateral portal as used for acromioplasty, an anterolateral at the anterior corner of the acromion, and the anterior portal for the distal clavicle resection.

After viewing the glenohumeral joint to define and treat other pathologic lesions, the arthroscope is placed into the subacromial space using the posterior portal. The subacromial bursa is removed to obtain clear visualization of all of the subacromial structures to include the coracoacromial ligament and its associated artery (acromial branch of the thoracoacromial artery), the torn coracoclavicular ligaments, and both the more lateral and anterior trapezoid and the more medial and posterior conoid ligament. The coracoacromial ligament is carefully dissected from surrounding tissue, and the artery is cauterized from the deltoid muscle.

Sutures—Inferior Attachment

The arthroscope is then placed in the lateral portal and instrumentation is used through the anterior, anterolateral, and posterior portals. The coracoacromial ligament is dissected from the deltoid and followed to the base of the coracoid. An Orthocord (Mitek), a Fiberwire (Arthrex), or an Ultrabraid (Smith and Nephew) suture is shuttled through the ligament beginning very close to its origin on the coracoid process. Alternatively, both braids are then passed several times through the coracoacromial ligament until its insertion on the acromion keeps the artery outside the loops (Fig. 32-26). Suture management is performed by PDS shuttle with a Spectrum (Linvatec) device, or direct passage by a Clever Hook (Mitek) may be used from the combined anterior, anterolateral, and posterior portal. The coracoacromial ligament is sharply dissected from the acromion undersurface. The sutures are retrieved posteriorly to keep the coracoacromial ligament close on the rotator cuff and away from the burr when a subacromial decompression is performed to facilitate a lateral view of the acromioclavicular joint and distal clavicle. A resection of the distal clavicle is performed.[18] The access to the distal clavicle can be difficult as it is dislocated superiorly and is better exposed by pushing down the clavicle while pushing up at the elbow. A resection of 5 to 7 mm of the distal clavicle is important to prevent painful bone-to-bone contact when the clavicle is reduced and held at the level of the acromion. When reduction looks difficult to maintain, one or two double-loaded suture anchors are placed through the anterior portal at the base of the coracoid where the coracoacromial ligament was attached and the sutures are retrieved posteriorly and later passed through drill holes in the distal clavicle.

Sutures—Superior Transosseous Management

Two-millimeter drill holes are made, using a percutaneous technique, in the lateral clavicle from superior to inferior about 5 mm apart, at the insertion of the native coracoacromial ligament.

Before passing the coracoacromial ligament sutures through the drill holes, the right length of the coracoacromial ligament is assessed by bringing the free end of the looped ligament with a grasper to the clavicle undersurface, while the assistant lifts up the arm to reduce the acromioclavicular joint dislocation. If the length is not perfect, additional looping of suture in the coracoacromial ligament or release of a loop is necessary. The number of holes drilled in the clavicle is based on the number of suture anchors in the coracoid. Two to three sutures can be passed through one drill hole.

A suture shuttle is placed through the drill holes in the clavicle using a 16-gauge spinal needle loaded with a looped #0 PDS suture (Fig. 32-27). Alternatively, a Hewson suture passer can be used. A grasper is used to retrieve the PDS suture loop, and the more lateral coracoacromial ligament suture is pulled out through the anterior portal. The coracoacromial ligament suture and the shuttle loop are passed through the drill hole by pulling out the needle and its attached suture at the same time. The same procedure is performed for the second coracoacromial ligament suture. Sutures on the anchors are passed using the same technique.

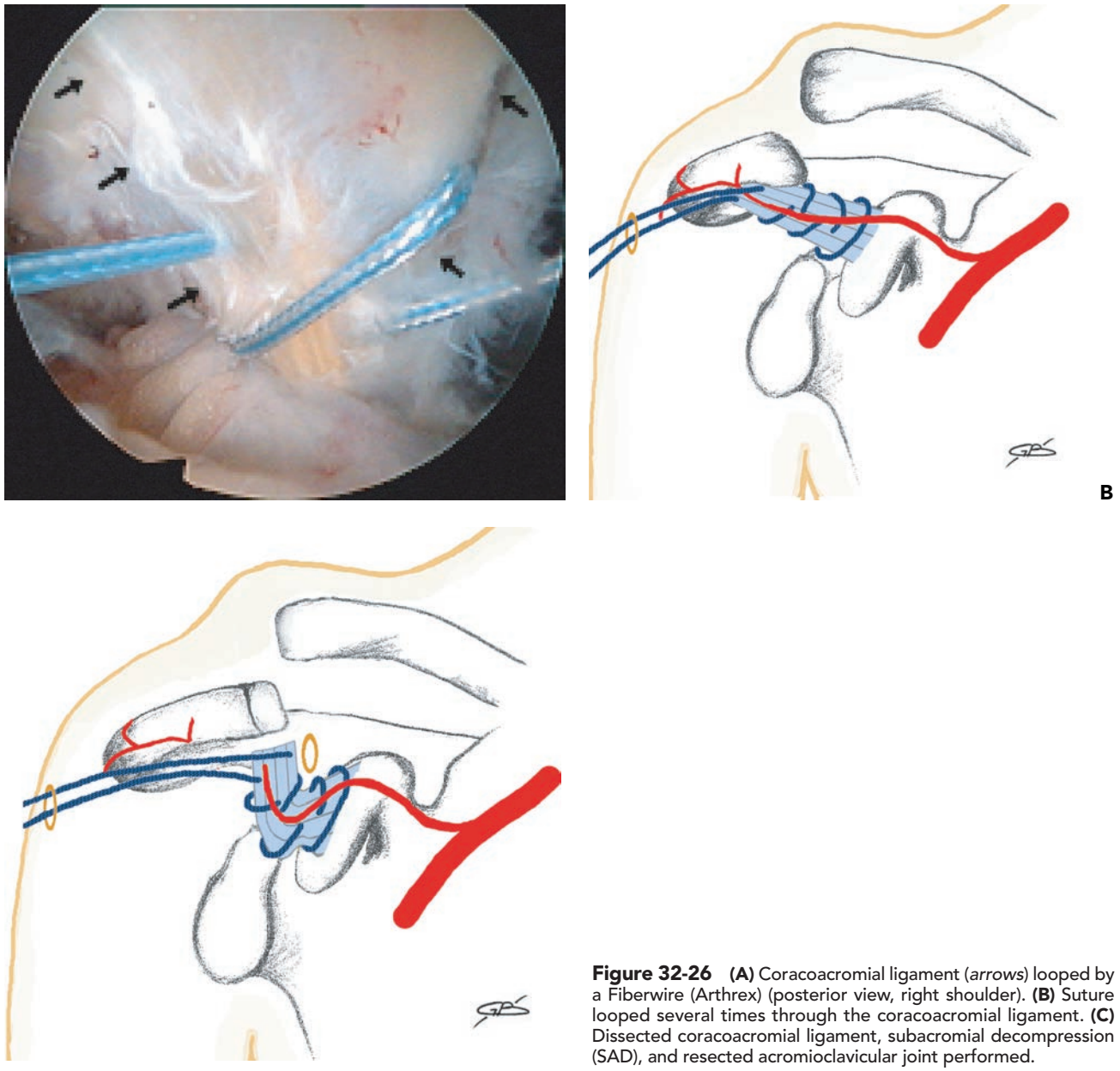


Figure 32-26 **(A)** Coracoacromial ligament (*arrows*) looped by a Fiberwire (Arthrex) (posterior view, right shoulder). **(B)** Suture looped several times through the coracoacromial ligament. **(C)** Dissected coracoacromial ligament, subacromial decompression (SAD), and resected acromioclavicular joint performed.

Final Fixation

If the final length and position of the coracoacromial ligament is good, then the knot is tied without creating another portal. Both sutures are retrieved between the clavicle and the subcutaneous tissue (Fig. 32-28). The traction on the arm is released to reduce the acromioclavicular joint by the assistant lifting the upper arm and pushing on the lateral clavicle at the same time.

The subcutaneous knot is tied blindly by successive half loops under slight overreduction of the acromioclavicular joint to compensate for the loss of reduction during the healing period. As knot tying cannot be visualized in this region, experience with knot tying is required to secure this knot. Alternately, this knot can be tied by direct visualization by making a small incision over the clavicle. Once the knot is tied, the sutures are cut with a blind cutting device and the portals are closed with absorbable sutures.

For an extra-secure reduction of the clavicle, a suture anchor in the base of the coracoid is necessary to maintain the reduced acromioclavicular joint. When this is used, the sutures are passed as described and tied first.

Postoperatively, a sling is used for 6 weeks; x-rays are performed in the recovery room and then 6 weeks after surgery. Motion is limited during the first 6 weeks with forward flexion and abduction up to 90 degrees; after 6 weeks the patients may start to move in the full range of motion, but without heavy lifting for the first 3 months.

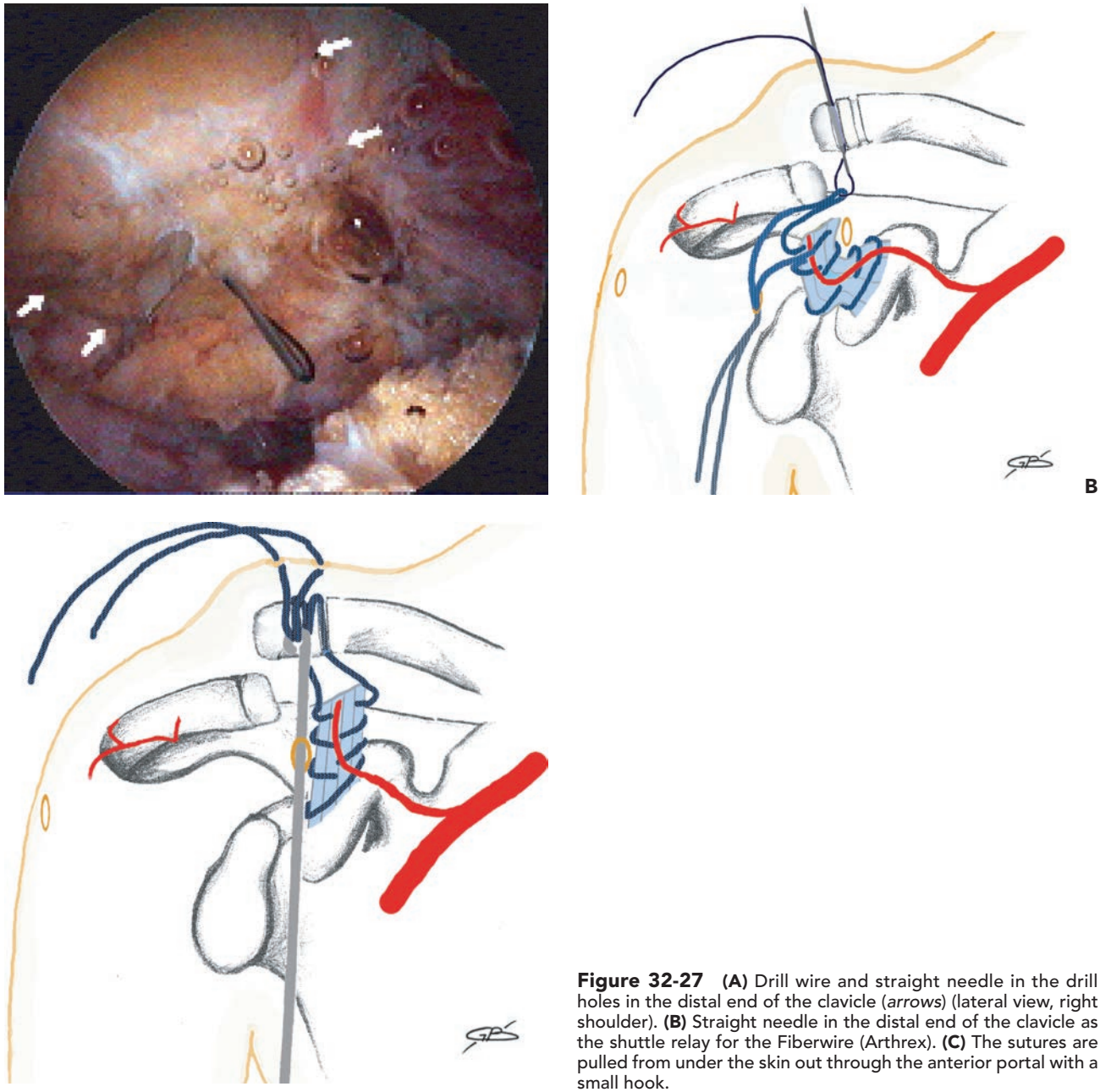


Figure 32-27 **(A)** Drill wire and straight needle in the drill holes in the distal end of the clavicle (*arrows*) (lateral view, right shoulder). **(B)** Straight needle in the distal end of the clavicle as the shuttle relay for the Fiberwire (Arthrex). **(C)** The sutures are pulled from under the skin out through the anterior portal with a small hook.

Results

An unpublished clinical experience with these techniques by one of the authors (LL) is a series of 14 cases from May 2002 to May 2003 with the technique described for transfer of the coracoacromial ligament and also temporary supplemental fixation with K-wires across the acromioclavicular joint. In only three cases was a full reduction maintained and in nine cases a partial reduction was maintained. A more recent technique of coracoacromial ligament transfer was performed in 20 cases with additional suture fixation using two double-loaded suture anchors placed in the base of the coracoid and passing the sutures between base of the coracoid and transosseous clavicle tunnels. In these cases K-wires across the acromioclavicular joint were not used. With this newer technique there was only one case of complete loss of fixation in a patient that did not follow postoperative instructions, two cases with partial loss of reduction with a good clinical result, and 17 cases without loss of reduction and an excellent clinical result. Further study and use of this technique by other surgeons will help clarify the perceived benefits over more traditional open surgical techniques, which have provided very reliable and reproducible results.

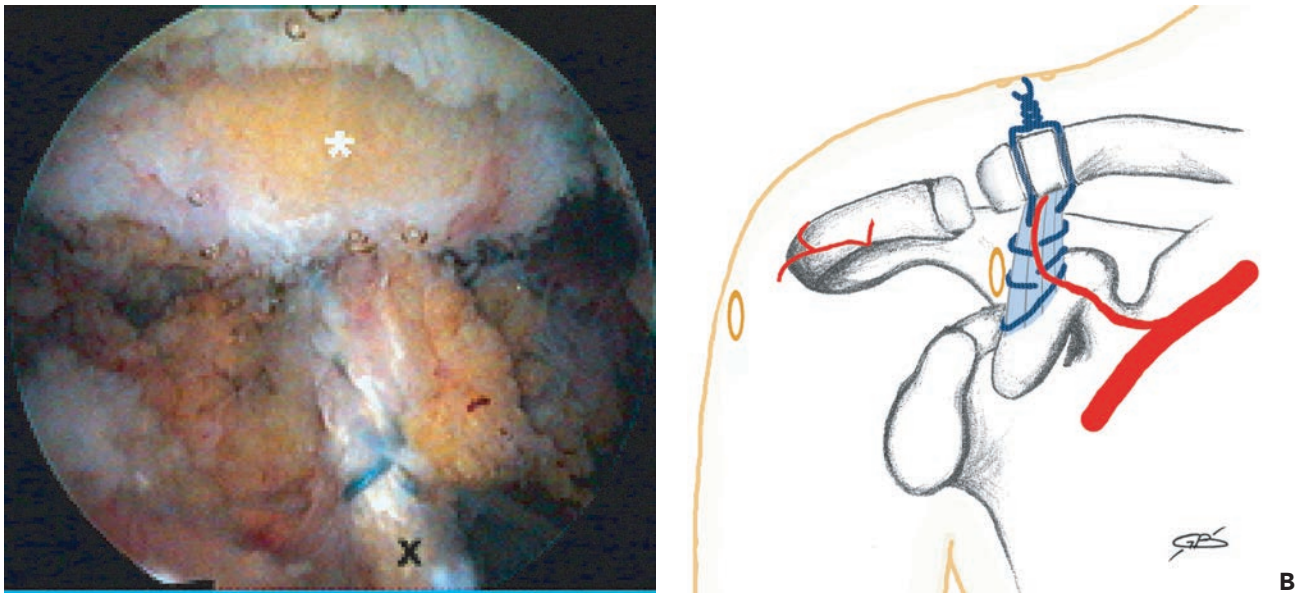


Figure 32-28 **(A)** Final arthroscopic aspect of the transferred coracoacromial ligament (x) and reduced acromioclavicular joint (*) (lateral view, right shoulder). **(B)** Final aspect of the transferred coracoacromial ligament and reduced acromioclavicular joint with the subcutaneous knot.

OSTEOLYSIS OF THE CLAVICLE

Osteolysis of the distal clavicle is felt to be related to repetitive activities such as weightlifting.[4,20,124] While an uncommon condition, it is felt to be increasing because of the popularity of weight training and the increasing awareness of physical fitness. Repetitive stress to the subchondral bone leads to subchondral fatigue fractures and a hypervascular response leading to resorption of bone. Some of the hallmarks of this condition are demineralization, osteopenia, subchondral cyst formation, and distal clavicle erosion.[77,101] The patient typically presents with pain over the acromioclavicular joint related to overhead activities. The pain may radiate to the neck and trapezius and down the arm and deltoid. Pain may be elicited by daily activities but is usually most pronounced with overhead athletic activity, particularly motions that bring the arm across the chest (adduction). Weightlifting activities such as the bench press, dips, and pushups exacerbate the pain.[20,101] Associated symptoms of popping, grinding, or catching are sometimes noted.

Physical examination of the extremity will typically find point tenderness over the acromioclavicular joint along with prominence of the distal clavicle. Maneuvers such as reaching across the body will elicit pain localized to the acromioclavicular joint. Motion is infrequently restricted. Impingement maneuvers may be positive if the rotator cuff is secondarily affected.

Radiographic evaluation of the shoulder will infrequently visualize the acromioclavicular joint adequately. The typical standard views of the shoulder overpenetrate the acromioclavicular joint and do not allow adequate assessment. For better visualization, the technique should be modified to underpenetrate one-third of joint. The Zanca view, an AP view with the beam tilted cephalad 15 degrees to better visualize the acromioclavicular joint without overlapping the spine of the clavicle, is helpful. Radiographic findings of osteolysis of the distal clavicle include distal clavicle osteopenia, cystic changes, and loss of subchondral bone detail. Later changes may include changes along the medial aspect of the acromion and widening of the acromioclavicular joint.

Occasionally, a bone scan may be helpful to further evaluate acromioclavicular joint pathology in light of relatively normal radiographs, particularly with osteolysis. Cahill believes that technetium-99m-labeled phosphate scintigraphy is the most diagnostic test in the evaluation of osteolysis.[20] Uptake of tracer is noted not only in the distal clavicle, but also in the adjacent acromion.

MRI may be a more practical test in the evaluation of the acromioclavicular joint since it allows excellent evaluation of the adjacent soft tissues including the rotator cuff, which is frequently secondarily involved. Edema in the distal clavicle on MRI has a high correlation with the presence of symptoms.[102]

Initial treatment of the acromioclavicular joint with osteolysis is nonoperative. Options include activity modification, cryotherapy, nonsteroidal and analgesic medications, occasional corticosteroid injections, and physical therapy. Most nonoperative treatments may require an extended period of time and require significant restrictions of an athletic endeavor to be effective. Many of

today's athletes will not tolerate the significant time and restriction of nonoperative treatments. Operative resection of the acromioclavicular joint may be required in patients with persistent pain and loss of function who do not respond to nonoperative modalities or in those who do not wish to modify their activities. Worcester and Green[121] found 100% success with joint resection in patients who responded to acromioclavicular joint injection.

Degenerative Lesions of the Acromioclavicular Joint

Patients who have degenerative or posttraumatic arthropathy of the acromioclavicular joint most commonly present with pain similar to osteolysis. The pain may be localized to the top of the shoulder and the area of the acromioclavicular joint, but it may also be referred to the area of the upper arm. Unlike osteolysis, typical impingement-type symptoms occur with abduction and overhead positions. Weight lifts, including bench press, incline press, and abduction exercises, frequently generate symptoms. Pain also occurs with cross-body arm positions. Patients often have night pain and pain when lying on the involved side. A history of previous injury to the shoulder and the patient's diagnosis, treatment, and recovery should be elicited. Some patients notice prominence of the acromioclavicular joint and may have clicking or catching in the area. Patients with acromioclavicular joint arthropathy may be symptomatic from motion of the arthritic joint, or outlet impingement from inferior projecting spurs may cause typical rotator cuff–type symptoms. A careful physical examination and radiographic evaluation are useful to determine the degree of acromioclavicular joint involvement and subacromial impingement.

A thorough examination is performed to evaluate the shoulder for all possible pathologies. Visual inspection may reveal asymmetry and a prominence of the acromioclavicular joint. There is asymmetrical point tenderness over the joint, which may be accentuated by horizontal cross-body adduction. This test is sensitive, but not specific, as it may elicit pain in patients with impingement from subacromial pathology. Acromioclavicular joint stability is assessed by grasping the distal clavicle between the thumb and index finger and translating the clavicle anteroposterior and superoinferior while the other hand stabilizes the acromion. Glenohumeral stability is also evaluated with typical apprehension, relocation, and translation tests. A careful neurologic examination should determine whether there is any possible brachial plexus involvement. Selective injection of lidocaine into the acromioclavicular joint frequently helps confirm that the joint is the cause of the symptoms. Response to injection of the subacromial space may also provide useful information on the presence of rotator cuff impingement.

A complete radiographic evaluation of the acromioclavicular joint includes standard AP and coracoacromial outlet ("Y") views. The outlet view is critical to accurately assess the anatomy of the anterior acromion and the degree of outlet impingement. Because the acromioclavicular joint is frequently difficult to assess on standard AP views, an underpenetrated view or Zanca view is warranted. An axillary view helps evaluate the anteroposterior position of the distal clavicle and the presence of an os acromion. MRI has supplanted use of the bone scan in determining the amount of acromioclavicular degeneration and the effect it may have impinging on the rotator cuff.

Patients who have pain primarily from a degenerated acromioclavicular joint have the best chance of responding to conservative management. Those who have symptoms that appear to be secondary to the effect of narrowing of the subacromial space from acromioclavicular joint spurring may have more refractory pain. We begin all patients who have no contraindications on a regime of nonsteroidal antiinflammatory medications. They are counseled to avoid provocative activities or arm positions and instructed in range-of-motion and stretching exercises to minimize capsular contractures. An intraarticular injection of lidocaine and steroid into the acromioclavicular joint may provide significant and sustained symptom relief. When subacromial impingement is also apparent, we will inject the subacromial space for diagnostic and therapeutic benefit. Only rarely have we used more than a total of three intraarticular acromioclavicular joint injections.

When patients have persistent pain and loss of function and activity levels despite attempts at conservative treatment, they may consider operative intervention. Excision of the distal clavicle is the procedure of choice. Similarly, patients with impingement from pathology at the acromioclavicular joint will benefit from surgical management. In these patients, excision of the distal clavicle is combined with an acromioplasty to decompress the coracoacromial outlet. Early surgical treatment may be preferred for symptomatic patients that have a complete tear of the rotator cuff.

Acromioclavicular joint resection and acromioplasty can be performed either open or by using arthroscopic techniques. The open procedure is straightforward and familiar to most surgeons. The amount of bone to be removed can be measured and predictably resected. In the absence of diagnostic errors, the results of surgery are reproducible and well documented. One disadvantage of open surgery is that it requires detachment of some portion of the deltoid from the trapezius to expose the clavicle. Access to the anterior acromion for an acromioplasty requires even greater dissection of the deltoid. This amount of soft tissue trauma accounts for the fairly significant amount of early postoperative pain that is experienced with this procedure. Historically, 1 to 2 cm of distal clavicle has been resected. Disruption of the acromioclavicular ligaments has raised some concerns about the problem of

postoperative horizontal instability of the clavicle.[14] Cosmetic issues are generally of lesser concern.

Arthroscopic excision of the distal clavicle has evolved as a popular yet technically demanding procedure. It can be used for isolated acromioclavicular joint arthrosis or in conjunction with an acromioplasty when impingement anatomy is present. Other than the difficulty in obtaining precise and reproducible bone resection, arthroscopic techniques offer advantages to open surgery. There is no significant trauma to the deltoid, less postoperative pain, and minimal scarring. In addition, no protection is necessary for the deltoid postoperatively (as opposed to open surgery). The recommended amount of distal clavicle resection has been determined to be only 5 mm to 1 cm.[42,43,81,83] Less bone resection and preservation of the acromioclavicular ligaments should diminish concerns over horizontal instability of the clavicle.

Complications of Surgery

Complications related to acromioclavicular injuries may occur as a result of the injury or related to its treatment, including surgery. Symptomatic degenerative changes occur with mild degrees of acromioclavicular injury.[25] Osteolysis of the distal clavicle may occur not only related to repetitive use, but also as a posttraumatic condition.[70,77] Ossification in the coracoclavicular interval may occur after coracoclavicular ligament injury or following surgical repair or stabilization. Its presence does not appear to affect outcome or results.[73,116] Some patients with chronic displaced acromioclavicular joint injuries complain of arm weakness, paresthesias, or other vague symptoms suggestive of traction on the brachial plexus. These thoracic outlet–type symptoms may be related to inferior position of the shoulder girdle relative to the thorax.

The surgical treatment of these injuries is associated with a significant number of possible problems. Most common may be the persistence or recurrence of a mild prominence or deformity that is often asymptomatic. Fixation failure with complete recurrence often will require a revision procedure.[68,75] The use of hardware has been associated with breakage, migration, and fixation failure.[21] Kirschner wires and Steinmann pins have migrated from the acromioclavicular joint to dangerous locations, such as the lung,[36,71] spinal cord,[80] and neck.[69] Fixation across the acromioclavicular joint may also precipitate arthritic changes of this joint. Erosion of the clavicle by wire or nonabsorbable suture used in coracoclavicular fixation has also been observed with some frequency.[57,78,86] This may lead to fracture of the clavicle or coracoid process. We have also seen infections related to the use of nonabsorbable augmentation tapes sometimes years after the initial surgery.[79,82]

Neurovascular injuries have been reported in relation to surgical treatment of these injuries, primarily because of the close proximity of the brachial plexus along with the subclavian artery and vein. Transfer of the short head of the biceps to the clavicle to provide dynamic stability has been associated with musculocutaneous injury.[23,68]

Resection of too much bone on the distal clavicle may produce instability of the clavicle in the anteroposterior plane that can be associated with symptoms.[14] Open techniques of distal clavicle resection commonly removed 2 to 3 cm of bone and has the potential to compromise the acromioclavicular ligaments, capsule, and deltotrapezial fascia attachments. Recurrent symptoms after clavicle resection were shown to correlate with the amount of anteroposterior translation of the distal clavicle.

No matter what method of treatment for these injuries is subscribed, the goals should remain the same: return to full preinjury or preoperative range of motion, with restoration of painless strength and function.

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33 Disorders of the Sternoclavicular Joint: Pathophysiology, Diagnosis, and Management

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HISTORICAL REVIEW

Rodrigues, in 1843, may have published the first case of traumatic posterior dislocation of the sternoclavicular joint in the literature, "a case of dislocation inward of the internal extremity of the clavicle."[180] The patient's left shoulder was against a wall when the right side of the chest and thorax

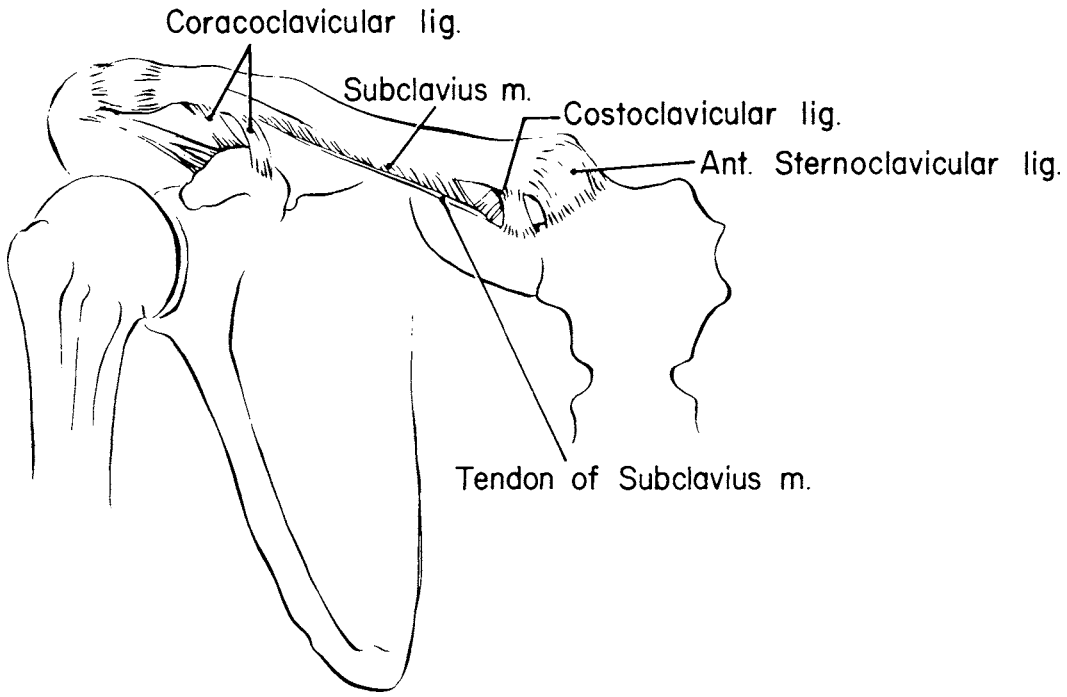


Figure 33-1 Normal anatomy around the sternoclavicular and acromioclavicular joints. Note that the tendon of the subclavius muscle arises in the vicinity of the costoclavicular ligament from the first rib and has a long tendon structure.

were compressed and rolled forward almost to the midline by a cart. Immediately, the patient experienced shortness of breath, which persisted for 3 weeks. When first seen by the physician, he appeared to be suffocating and his face was blue. The left shoulder was swollen and painful, and there was "a depression on the left side of the superior extremity of the sternum." Pressure on the depression greatly increased the sensation of suffocation. Rodrigues observed that when the outer end of the shoulder was displaced backward, the inner end of the clavicle was displaced forward, which relieved the asphyxia. Therefore, treatment consisted of binding the left shoulder backward with a cushion between the two scapulas, but only after the patient had been bled twice within the first 24 hours. Rodrigues may have seen other cases of posterior dislocation, since he stated that the patient "retained a slight depression of the internal extremity of the clavicle; such, however, is the ordinary fate of the patients who present this form of dislocation."

In the late 19th century, a number of articles appeared from England, Germany, and France; it was not until the 1930s that articles by Duggan,[51] Howard and Shafer,[90] and Lowman[131] appeared in the American literature.

SURGICAL ANATOMY AND BIOMECHANICS

The sternoclavicular joint is a diarthrodial joint and is the only true articulation between the clavicle of the upper extremity and the axial skeleton (Fig. 33-1). The articular surface of the clavicle is much larger than that of the sternum, and both are covered with fibrocartilage. The enlarged bulbous medial end of the clavicle is concave front to back and convex vertically, and therefore creates a saddle-type joint with the clavicular notch of the sternum.[6059] The clavicular notch of the sternum is curved, and the joint surfaces are not congruent. Cave[32] has demonstrated that in 2.5% of patients, there is a small facet on the inferior aspect of the medial clavicle that articulates with the superior aspect of the first rib at its synchondral junction with the sternum.

Because less than half of the medial clavicle articulates with the upper angle of the sternum, the sternoclavicular joint has the distinction of having the least amount of bony stability of the major joints of the body. As Grant[81] noted, "The two (make) an ill fit." If a finger is placed in the superior sternal notch, with motion of the upper extremity one can feel that a large part of the medial clavicle is completely above the articulation with the sternum.

LIGAMENTS OF THE STERNOCLAVICULAR JOINT

There is so much joint incongruity that the integrity has to come from its surrounding ligaments: the intraarticular disk ligament, extraarticular costoclavicular ligament (rhomboid ligament), capsular ligaments, and interclavicular ligament.

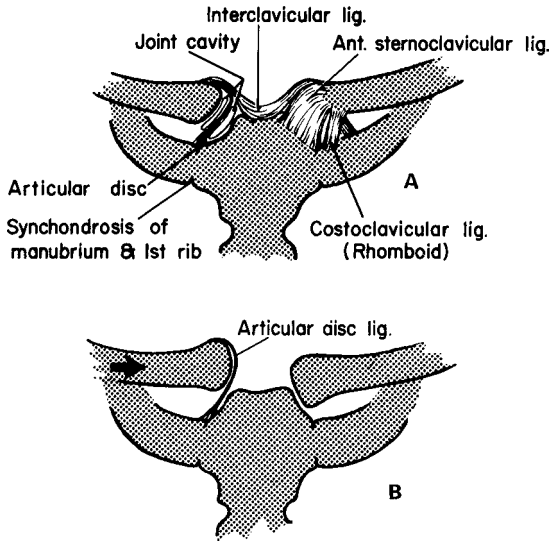


Figure 33-2 **(A)** Normal anatomy around the sternoclavicular joint. Note that the articular disk ligament divides the sternoclavicular joint cavity into two separate spaces and inserts onto the superior and posterior aspects of the medial clavicle. **(B)** The articular disk ligament acts as a checkrein for a medial displacement of the proximal clavicle. (Reprinted with permission from Rockwood CA, Matsen F III, eds. *The shoulder.* Philadelphia: WB Saunders, 1990: Fig. 13-2.)

Intraarticular Disk Ligament

The intraarticular disk ligament is a very dense, fibrous structure that arises from the synchondral junction of the first rib to the sternum and passes through the sternoclavicular joint, which divides the joint into two separate joint spaces (Fig. 33-2).[7,59,60] The upper attachment is on the superior and posterior aspects of the medial clavicle. DePalma[47] has shown that the disk is perforated only rarely; the perforation allows a free communication between the two joint compartments. Anteriorly and posteriorly, the disk blends into the fibers of the capsular ligaments. The disk acts as a checkrein against medial displacement of the inner clavicle (see Fig. 33-2).

Costoclavicular Ligament

The costoclavicular (CC) ligament, also called the rhomboid ligament, is short and strong and consists of an anterior and a posterior fasciculus (Fig. 33-3).[11,32,60] Cave[32] reported that the average length is 1.3 cm, the maximum width 1.9 cm, and the average thickness 1.3 cm. Bearn[11] has shown that there is always a bursa between the two components of the ligament. Because of the two different parts of the ligament, it has a twisted appearance. The costoclavicular ligament attaches below to the upper surface of the first rib and at the adjacent part of the synchondral junction with the sternum, and above to the margins of the impression on the inferior surface of the medial end of the clavicle, sometimes known as the rhomboid fossa. Cave[32] has shown, from a study of 153 clavicles, that the attachment of the costoclavicular ligament to the clavicle can be any of three types: a depression, the rhomboid fossa (30%); flat (60%); or an elevation (10%).

The fibers of the anterior fasciculus arise from the anteromedial surface of the first rib and are directed upward and laterally. The fibers of the posterior fasciculus are shorter and arise lateral to the anterior fibers on the rib and are directed upward and medially. The fibers of the anterior and posterior components cross and allow for stability of the joint during rotation and elevation of the clavicle. The two-part configuration of the costoclavicular ligament is similar to the coracoclavicular ligament on the outer end of the clavicle.

Bearn[11] has shown experimentally that the anterior fibers resist excessive upward rotation of the clavicle and that the posterior fibers resist excessive downward rotation. Clinically, the cruciate nature of the costoclavicular ligament

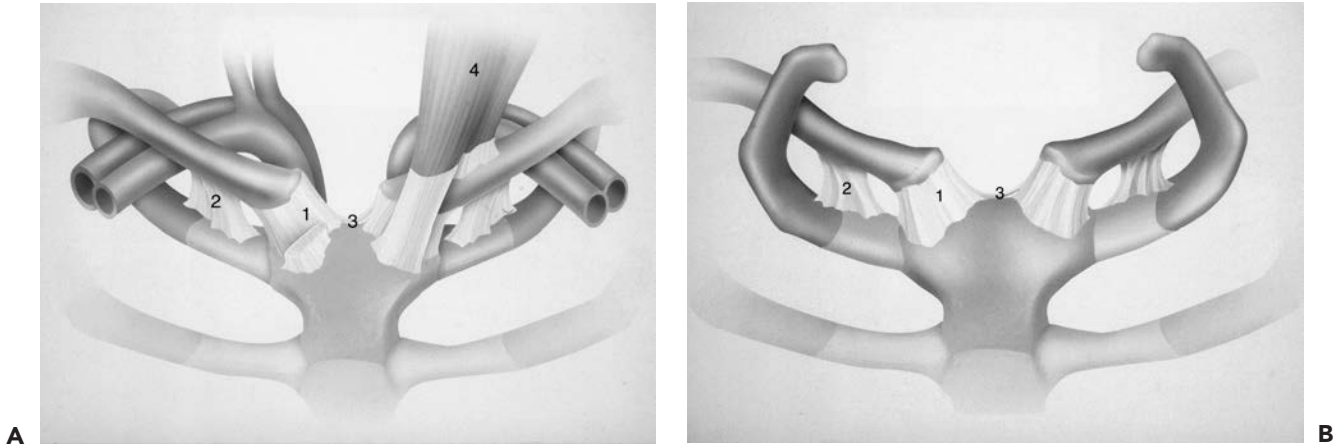


Figure 33-3 **(A)** Anterior view of SC joints. 1 = Anterior capsular ligament, 2 = Costoclavicular ligament, 3 = Interclavicular ligament, 4 = Sternocleidomastoid muscle. **(B)** Posterior view of SC joint. 1 = Posterior capsular ligament, 2 = Costoclavicular ligament, 3 = Interclavicular ligament. (Reprinted from Spencer EE, Kuhn JE, Huston LJ, Carpenter JE, Hughes RE. Ligamentous restraints to anterior and posterior translation of the sternoclavicular joint. *J Shoulder Elbow Surg* 2002;11(1):43–47.)

provides a point about which the clavicle can rotate when the shoulder is brought through full range of motion.

Interclavicular Ligament

The interclavicular ligament connects the superomedial aspects of each clavicle with the capsular ligaments and the upper sternum (see Fig. 33-3). According to Grant,[8] this band may be comparable with the wishbone of birds. This ligament helps the capsular ligaments to produce "shoulder poise," that is, to hold up the shoulder. This can be tested by putting a finger in the superior sternal notch; with elevation of the arm, the ligament is quite lax, but as soon as both arms hang at the sides, the ligament becomes tight.

Capsular Ligaments

The capsular ligaments cover the anterior and posterior aspects of the joint and represent thickenings of the joint capsule (see Fig. 33-3). The posterior capsular ligament is heavier and stronger than the anterior portion.

According to the original work of Bearn,[11] these may be the strongest ligaments of the sternoclavicular joint, and it is the first line of defense against the upward displacement of the inner clavicle caused by a downward force on the distal end of the shoulder. The clavicular attachment of the ligament is primarily onto the epiphysis of the medial clavicle, with some secondary blending of the fibers into the metaphysis. The senior author has demonstrated this, as have Poland,[162] Denham and Dingley,[45] and Brooks and Henning.[22]

Although some investigators report that the intraarticular disk ligament greatly assists the costoclavicular ligament in preventing upward displacement of the medial clavicle, Bearn[11] has shown that the capsular ligament is the most important structure in preventing upward displacement of the medial clavicle. In experimental postmortem studies, he evaluated the strength and the role of each of the ligaments at the sternoclavicular joint to see which one would prevent a downward displacement of the outer clavicle. He attributed the lateral "poise of the shoulder" (i.e., the force that holds the shoulder up) to a locking mechanism of the ligaments of the sternoclavicular joint (Fig. 33-4). To accomplish his experiments, Bearn dissected all the muscles attaching onto the clavicle, the sternum, and the first rib and left all the ligaments attached. He secured the sternum to a block in a vise. He then loaded the outer end of the clavicle with 10 to 20 lb of weight and cut the ligaments of the sternoclavicular joint, one at a time and in various combinations, to determine each ligament's effect on maintaining the clavicle poise or, thinking of it in another way, which ligament would rupture first when a force was applied to the outer end of the clavicle.

He determined, after cutting the costoclavicular, intraarticular disk, and interclavicular ligaments, that they had no effect on clavicle poise. However, the division of the capsular ligaments alone resulted in a downward depression on the distal end of the clavicle. He also noted that the intraarticular disk ligament tore under 5 lb of weight, once the capsular ligament had been cut. These data demonstrate the importance of the capsular ligaments in maintaining the poise of the clavicle.

Although poise is important, clinically, dislocations of the sternoclavicular joint are seen in the anterior or posterior direction. Therefore, a more recent study by Spencer et al. evaluated the restraints to anterior and posterior translation of the sternoclavicular joint.[203] Thirty-two cadaveric specimens were dissected leaving the ligamentous structures intact. The sternum was mounted in a supine position on a biaxial rotation table while the clavicle was translated anteriorly and posteriorly by the servohydraulic testing system. Serial ligament sectioning of the interclavicular ligament, costoclavicular ligament, and the anterior and posterior capsular ligaments was performed. It was found that the primary restraint to posterior translation was the posterior capsular ligament (Fig. 33-5). The primary restraint to anterior translation was also the posterior capsular ligament, with the anterior capsular ligament providing an important secondary stabilizer (Fig. 33-6). The interclavicular ligament and costoclavicular ligament provided very little stability to the joint. This emphasizes the importance of the posterior capsule, which is twice as thick as its anterior counterpart. This physiologically makes sense in that posterior displacement of the medial end of the clavicle can impinge upon and damage the important neurovascular structures and trachea.

Range of Motion of the Sternoclavicular Joint

The sternoclavicular joint is freely movable and functions almost like a ball-and-socket joint, in that the joint has motion in almost all planes, including rotation.[95,132,230] Inman et al. observed that within the first 90 degrees of arm elevation, the clavicle would elevate 4 degrees per 10 degrees of arm elevation. After 90 degrees of arm elevation there was negligible motion of the clavicle.[95] The clavicle, and therefore the sternoclavicular joint, in normal shoulder motion is capable of 30 to 35 degrees of upward elevation, 35 degrees of combined forward and backward movement, and 45 to 50 degrees of rotation around its long axis (Fig. 33-7). It is most likely the most frequently moved joint of the long bones in the body, because almost any motion of the upper extremity is transferred proximally to the sternoclavicular joint.

Epiphysis of the Medial Clavicle

Although the clavicle is the first long bone of the body to ossify (fifth intrauterine week), the epiphysis at the medial end of the clavicle is the last of the long bones in the body to appear and the last epiphysis to close (Fig. 33-8). The medial clavicular epiphysis does not ossify until the 18th

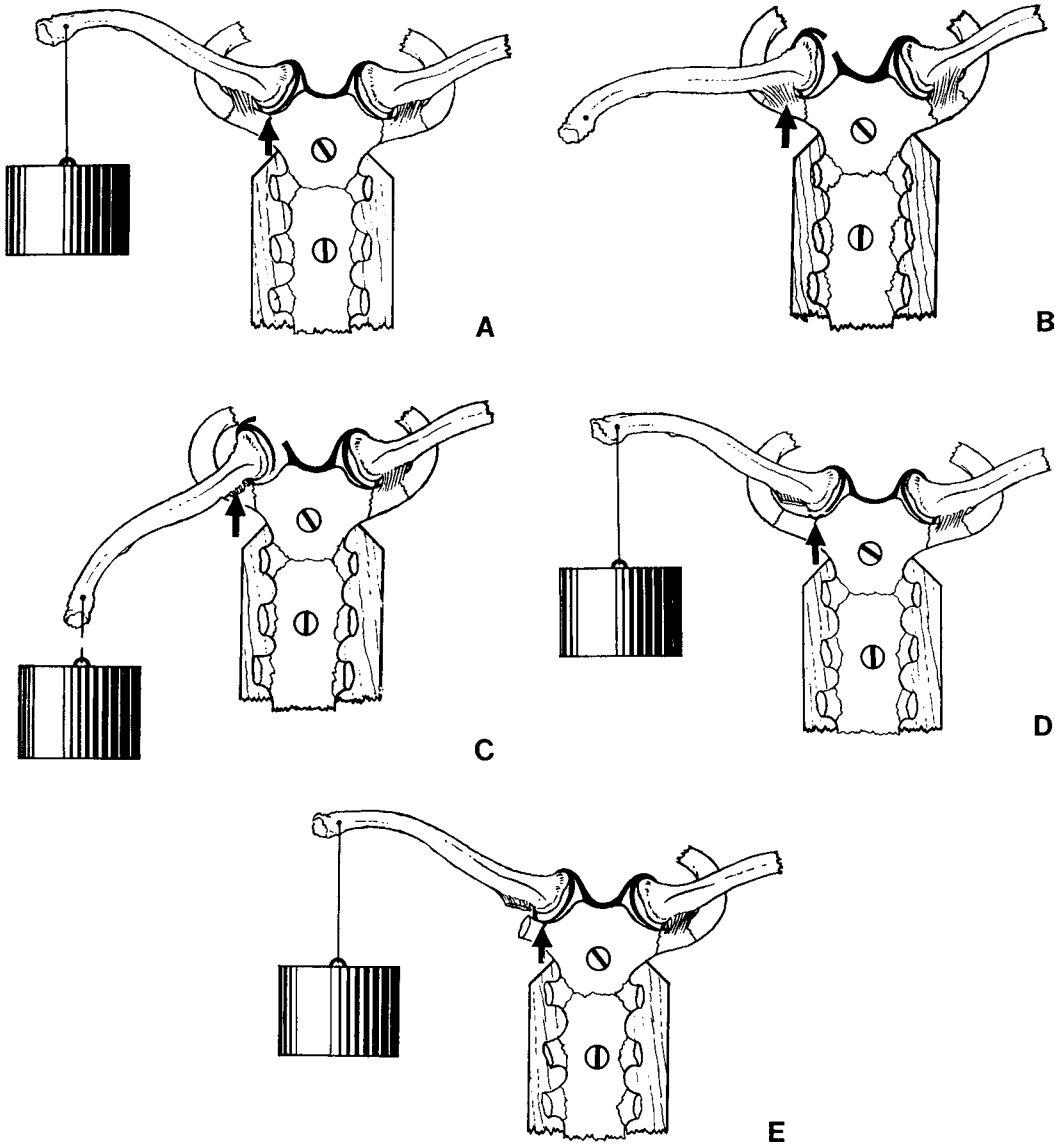


Figure 33-4 The importance of the various ligaments around the sternoclavicular joint in maintaining normal shoulder poise. **(A)** The lateral end of the clavicle is maintained in an elevated position through the sternoclavicular ligaments. The arrow indicates the fulcrum. **(B)** When the capsule is divided completely, the lateral end of the clavicle descends under its own weight without any loading. The clavicle will seem to be supported by the intraarticular disk ligament. **(C)** After division of the capsular ligament, it was determined that a weight of less than 5 lb was enough to tear the intraarticular disk ligament from its attachment on the costal cartilage junction of the first rib. The fulcrum was transferred laterally, so that the medial end of the clavicle hinged over the first rib in the vicinity of the costoclavicular ligament. **(D)** After division of the costoclavicular ligament and the intraarticular disk ligament, the lateral end of the clavicle could not be depressed as long as the capsular ligament was intact. **(E)** After resection of the medial first costal cartilage, along with the costoclavicular ligament, there was no effect on the poise of the lateral end of the clavicle, as long as the capsular ligament was intact. (Reprinted with permission from Bearn JG. Direct observation on the function of the capsule of the sternoclavicular joint in clavicular support. *J Anat* 1967;101:159–170.)

to 20th year of life, and it fuses with the shaft of the clavicle around the 23rd to 25th year.[81,124,128,162,219] Webb and Suchey,[219] in an extensive study of the physis of the medial clavicle in 605 males and 254 females at autopsy, reported that complete unions may not be present until 31 years of age. This knowledge of the epiphysis is important, because it is believed that many so-called sternoclavicular dislocations are actually fractures through the physeal plate.[22,45,68,93,124,,128,133,162,192,226,229]

Applied Surgical Anatomy

The surgeon who is planning an operative procedure on or near the sternoclavicular joint should be completely

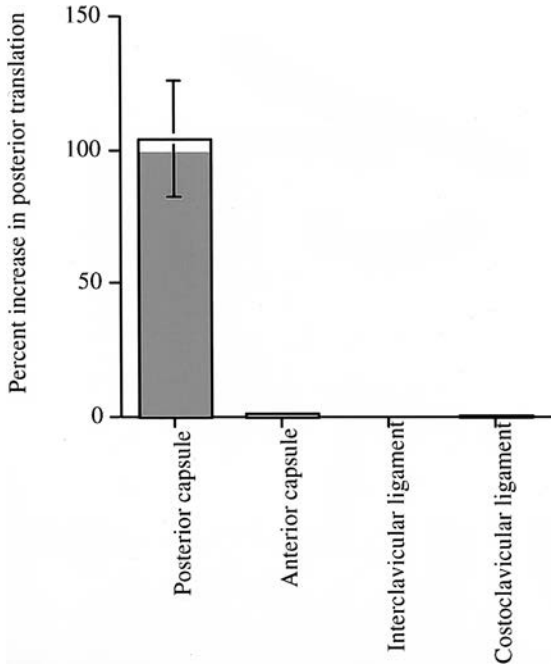


Figure 33-5 Restraints for posterior translation of the sternoclavicular joint. (Reprinted from Spencer EE, Kuhn JE, Huston LJ, Carpenter JE, Hughes RE. Ligamentous restraints to anterior and posterior translation of the sternoclavicular joint. *J Shoulder Elbow Surg* 2002;11(1):43–47.)

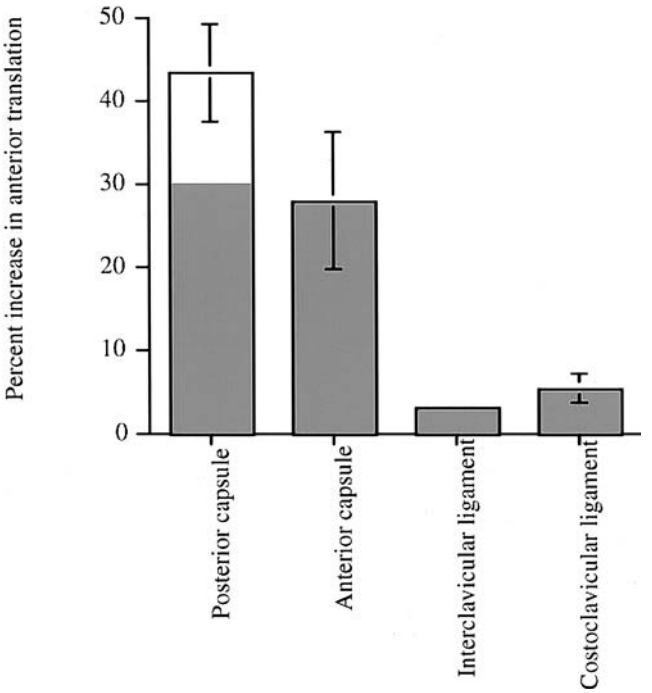


Figure 33-6 Restraints for anterior translation of the sternoclavicular joint. (Reprinted from Spencer EE, Kuhn JE, Huston LJ, Carpenter JE, Hughes RE. Ligamentous restraints to anterior and posterior translation of the sternoclavicular joint. *J Shoulder Elbow Surg* 2002;11(1):43–47.)

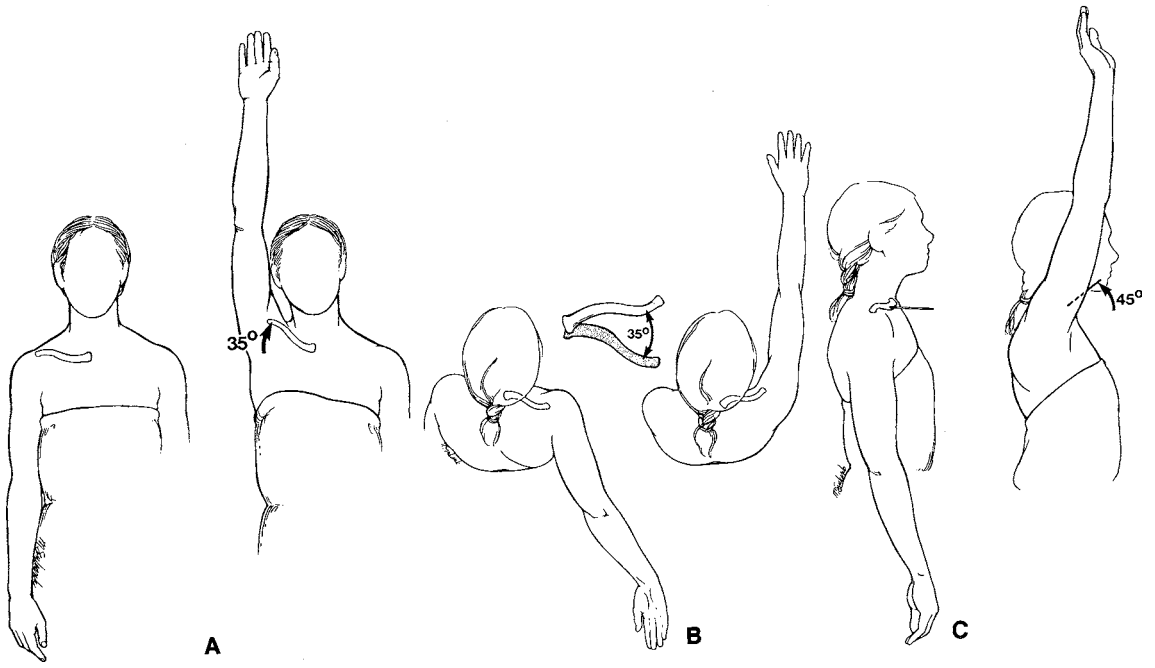


Figure 33-7 Motions of the clavicle and the sternoclavicular joint. **(A)** With full overhead elevation, the clavicle rises 35 degrees. **(B)** With adduction and extension, the clavicle displaces anteriorly and posteriorly 35 degrees. **(C)** The clavicle rotates on its long axis 45 degrees as the arm is elevated to the full overhead position.

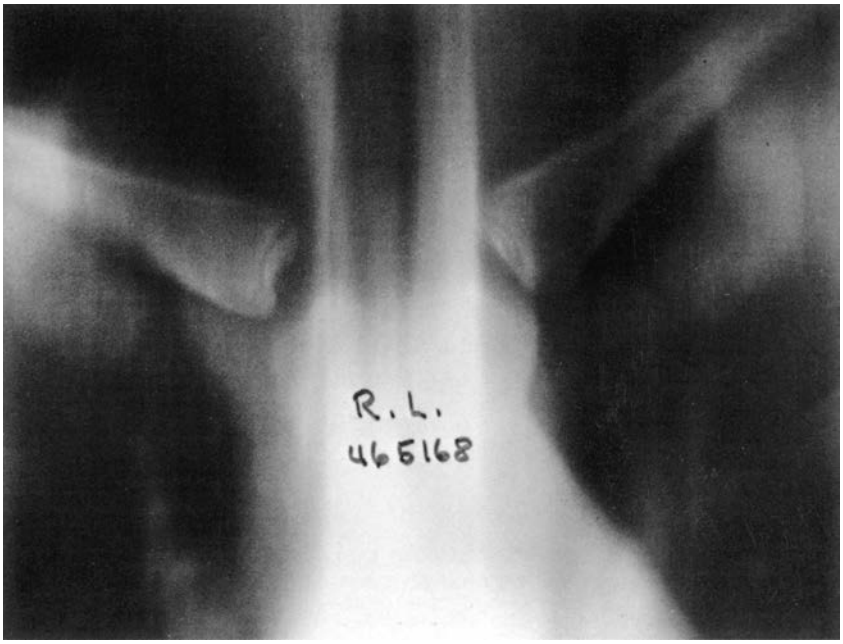


Figure 33-8 Tomogram demonstrating the thin, wafer-like disk of the epiphysis of the medial clavicle.

knowledgeable about the vast array of anatomic structures immediately posterior to the sternoclavicular joint.[168] There is a "curtain" of muscles—the sternohyoid, sternothyroid, and scaleni—posterior to the sternoclavicular joint and the inner third of the clavicle, and this curtain blocks the view of the vital structures. Some of these vital structures include the innominate artery, innominate vein, vagus nerve, phrenic nerve, internal jugular vein, trachea, and esophagus (Fig. 33-9). If one is considering stabilizing the sternoclavicular joint, knowledge of the anatomy is crucial, and we usually have a thoracic or vascular surgeon on standby to help treat an unintended injury of these structures on an emergent basis.

Another structure to be aware of is the anterior jugular vein, which is located between the clavicle and the curtain of muscles. The anatomy books state that it can be quite variable in size; we have seen it as large as 1.5 cm in diameter. This vein has no valves, and when it is nicked, it looks like someone has opened up the flood gates.

ATRAUMATIC CONDITIONS OF THE STERNOCLAVICULAR JOINT

There are several atraumatic conditions that affect the sternoclavicular (SC) joint. Although most are treated nonoperatively, they should not be overlooked. These include osteoarthritis, inflammatory arthritides, sternocostoclavicular hyperostosis (SCCH), osteitis condensans, Friedrich's disease, infections, and spontaneous subluxations or dislocations. Table 33-1 provides a list of these conditions. The following section will explore the pathophysiology, diagnosis, and treatment. Spontaneous subluxations and dislocations will be discussed separately.

Sternocostoclavicular Hyperostosis

Sternocostoclavicular hyperostosis is characterized by bone overgrowth and soft tissue ossification of the medial clavicle, upper ribs, and sternum. According to most investigators,[55,66,71,77,79,85,91,96,99,166,186,195,199,205,209] sternocostoclavicular hyperostosis was first described by Sonozaki et al. in 1974.[199] It is also known as intersternocostoclavicular ossification, pustulotic arthrosteitis, and juxtasternal arthrosteitis.

This condition, usually bilateral, affects adults of both sexes between 30 and 60 years of age. Unilateral cases have been reported and could represent various stages of the disease. It has been reported in patients as young as 11 years of age to as old as 88 years.[186199] Although many cases are reported from Japan, it is recognized worldwide. Saghafi et al. reviewed 251 cases and found that 139 cases were Japanese and 114 were Caucasian.[186]

SCCH is characterized by ossification of the SC joint and medial ribs and sternum and can be associated with aseptic pustular lesions on the palms and soles. The process begins at the junction of the medial clavicle, the first rib, and the sternum as an ossification in the ligaments (primarily the costoclavicular ligament) and later involves the bones. Because the primary lesion is not hyperostosis of the bones but rather new bone formation in the periarticular tissue, it should be classified as an enthesopathy.[71] Fritz et al. emphasized that the osseous hypertrophy caused by periosteal new bone formation occurs in the entire circumference of the clavicle before ossification of

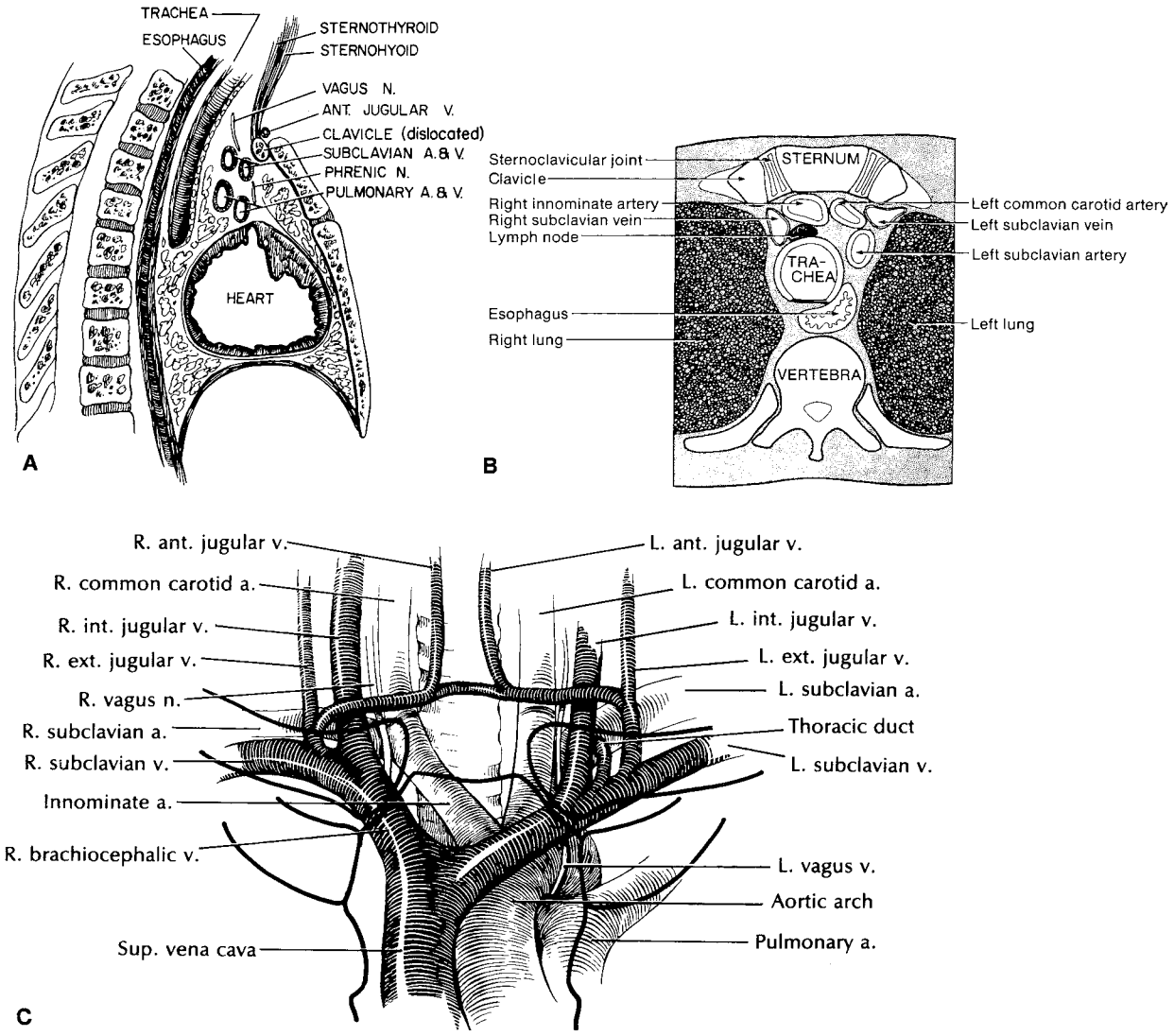


Figure 33-9 Applied anatomy of the vital structures posterior to the sternoclavicular joint. **(A,B)** Sagittal views in cross-section demonstrating the structures posterior to the sternoclavicular joint. **(C)** A diagram demonstrating the close proximity of the major vessels posterior to the sternoclavicular joint. **(D)** An aortogram showing the relationship of the medial end of the clavicle to the major vessels in the mediasternum.

TABLE 33-1
CONDITIONS AND CHARACTERISTICS

Condition	Radiographic Findings	Physical Findings	Treatment
Sternocostoclavicular hyperostosis	Ranges from localized ossification of costoclavicular ligaments to frank bone formation between clavicle, sternum, and upper ribs	Restricted shoulder motion, aseptic pustular lesions of palms and soles in 10%–30% of patients	Most patients respond to NSAIDs, heat, and activity modification; patients with severe pain and severely restricted motion due to complete fusion of clavicle, sternum, and first rib may benefit from surgical excision of hypertrophic bone
Osteitis condensans	Sclerosis and slight enlargement of medial third of clavicle	Swelling and tenderness of sternoclavicular joint	Most patients respond to NSAIDs, heat, and activity modification; excisional arthroplasty of medial end of clavicle in refractory cases
Aseptic necrosis (Friedrich's disease)	Irregularity of sternoclavicular joint with bony destruction of medial end of clavicle	Swelling and tenderness of sternoclavicular joint	Most patients respond to NSAIDs, heat, and activity modification; excisional orthroplasty of medial end of clavicle in refractory cases
Tietze's syndrome	Radiographic studies are of little diagnostic use	Tenderness most commonly noted at costosternal junction of second rib; mild swelling common	Most patients respond to NSAIDs, heat, and activity modification
Osteoarthritis	Subtle erosions and cystic changes particularly involving inferior aspect of medial end of clavicle	Mild swelling and tenderness usually involving sternoclavicular joint of dominant extremity	Most patients respond to NSAIDs, heat, and activity modification
Spontaneous subluxation	Subluxation (anterosuperior) when arm is placed in overhead position	Palpable and visual anterosuperior subluxation of medial end of clavicle with overhead elevation of arm	Self-limiting condition; operative procedures to stabilize sternoclavicular joint are generally unsuccessful
Infection	Soft tissue swelling with erosion of medial end of clavicle	Localized erythema, swelling, warmth, and tenderness to palpation	Irrigation and débridement of sternoclavicular joint with excision of involved portion of medial end of clavicle

NSAIDs = nonsteroidal antiinflammatory drugs.

the CC ligament, suggesting primary marrow pathology.[71] Lagier and colleagues published an excellent review of the x-ray findings and pathology from an autopsy specimen.[120] In some cases, the hyperostosis is extensive and forms a solid block of bone of the sternum, ribs, and clavicle. As might be expected with the fusion of the sternoclavicular joint, shoulder motion is severely restricted. Dohler[50] reported that as a result of the fusion of the sternoclavicular joint, his patient developed compensatory dislocation of the acromioclavicular joint. Patients may have peripheral arthritis. Subperiosteal bone changes have been noted in x-ray films of other bones (i.e., humerus, pelvis, tibia, ribs, and vertebral bodies).

The condition has been graded into three stages by Sonozaki et al.[199] Stage I is mild ossification in the area of the costoclavicular ligament; stage II is characterized by ossification beyond the CC ligament and an ossific mass between the clavicle and the first rib; and in stage III, a bone mass exists between the clavicle, sternum, and first rib. Fritz et al. have described a modification to this classification to include extrasternal manifestations.[71] The subscript "E" denotes extrasternal manifestations while the subscript "R" denotes involvement beyond the first rib. In the early stages a computed tomography (CT) scan will reveal ossification in the cartilaginous portions of the SC joint and first rib. The SC joint itself is well preserved even in the later stages.[195] A bone scan will reveal increased uptake even if the radiographic appearance is normal.[195]

Clinically, patients present with intermittent pain and swelling in and about the SC joints. There is usually localized warmth and swelling. The pain may radiate to the shoulder or arm. Some patients present with an asymptomatic mass.[66] Loss of motion of the shoulder may ensue if the disease progresses to complete ankylosis of the SC joint.[50]

The most consistent laboratory finding is an elevated sedimentation rate, which may increase with progression of the disease. In some cases C-reactive protein and α-1 globulin levels are elevated. Although there is a strong association with seronegative spondyloarthropathies, most patients had a negative HLA-B27.[103] Alkaline phosphatase may be elevated in some.

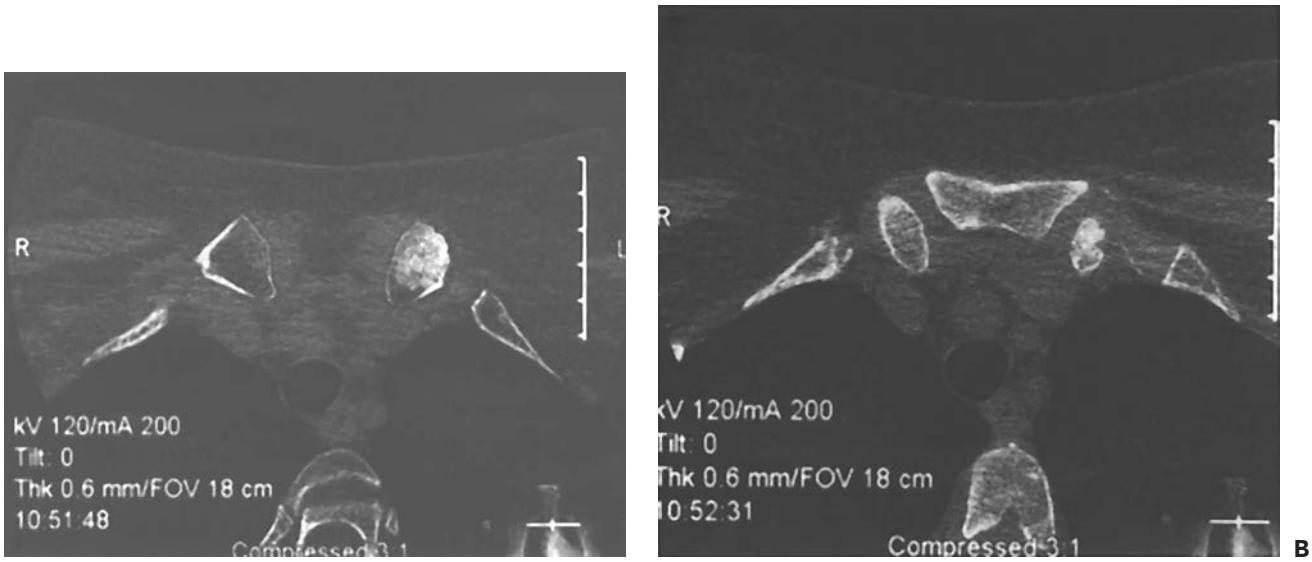
Bone marrow biopsy has demonstrated chronic inflammation with lymphocytes, plasma cells, and macrophagic histiocytes. Other biopsy specimens have revealed bone remodeling and thickening of the trabeculae of the cancellous bone and osteoid formation. Analysis of the periosteum also demonstrates fibroblastic scar remodeling and chronic inflammatory cells. Most culture results are negative, but Edlund et al. did report a positive culture for propionibacterium acne.[55] This is interesting as there is a strong association with pustulosis palmaris et plantaris (PPP), which is a dermatologic condition characterized by aseptic pustular lesions on the palms and soles. In fact, a review of the literature found a 10% to 30% association between the two conditions.[55,71,91,99] Other dermatologic conditions have also been reported in conjunction with SCCH and include acne conglobata, acne fulminans, pustulotic psoriasis, hidradenitis suppurativa, and dissecting cellulites of the scalp.[153] In the dermatologic literature the acronym SAPHO has been applied, which includes the constellation of symptoms of *s*ynovitis, *a*cne, *p*ustulosis, *h*yperostosis, and *o*steitis.

Management of SCCH is usually nonoperative with control of the symptoms with nonsteroidal antiinflammatory drugs. Other options have included radiation, corticosteroids, antibiotics, and surgical excision. None of these options has seemed to affect the natural history of the disease. Surgical excision is usually reserved for those patients with complete ankylosis and loss of shoulder motion.[55,199]

Condensing Osteitis

Brower and associates first described in detail the rare condition known as condensing osteitis of the medial clavicle in two patients in 1974.[23] Although a rare condition, several other authors have described this condition.[14,37,52,69,92,101,116,149,155] It usually occurs unilaterally in women of late child-bearing age and may occur secondary to chronic stress on the joint. No bilateral cases have been reported. The joint is swollen and tender, and radionuclide studies show an increased uptake of the isotope. The pain may be intermittent and worsened with heavy or repetitive lifting. Radiation to the axilla or shoulder is not uncommon. Routine laboratory studies are inconsistent but may reveal an elevated sedimentation rate, but the white blood cell count is usually normal. X-ray films show sclerosis and slight expansion of the medial third of the clavicle. The inferior portion of the sternal end of the clavicle shows sclerotic changes. Some osteophytes may be present, but the SC joint space is preserved. The changes of the medial clavicle are best detected via CT, which reveals sclerosis of the marrow space and enlargement of the medial end of the clavicle[116](Fig. 33-10). The differential diagnosis includes Paget's disease, sternoclavicular hyperostosis, Friedrich's avascular necrosis of the medial clavicle epiphysis, infection, Tietze's syndrome, and osteoarthritis. More recently, Vierboom and associates have described the use of magnetic resonance imaging (MRI) as an adjunctive method for diagnosing this entity.[215]Typically the MRI will reveal low-signal intensity on both T1- and T2-weighted images.

Jones et al. have called into question the actual existence of condensing osteitis of the clavicle based on histologic and serologic data.[101] They reported three cases in children with similar radiographic findings, but incisional biopsies revealed a mixed inflammatory process (neutrophils and lymphocytes) and new bone formation (woven and lamellar)



A B

Figure 33-10 Computed tomography (CT) findings for a 32-year-old woman with dull aching in her left medial clavicle for 18 months. There was no history of trauma. **(A)** CT scan of both medial clavicles reveals dense ossification of the left medial clavicle. **(B)** There is no involvement of the SC joint space. (From Hiramuro-Shoji F, Wirth MA, Rockwood CA Jr. Atraumatic conditions of the sternoclavicular joint. *J Shoulder Elbow Surg* 2003;12(1):79–88, with permission.)

covered by large osteoblasts. Although the cultures and stains were all negative, two had elevated anti-α hemolysin titers, raising the question of staphylococcal infectious cause. In addition, Appell et al. reported seven cases of condensing osteitis in children, four of whom responded to antibiotics.[3] Kruger et al. reported three cases of middle-aged females with similar radiographic findings with incisional biopsies revealing reactive sclerosis, but no mention was made regarding an inflammatory cellular infiltrate.[116] It might be that these cases represent different pathologic processes and may be separate entities entirely. It is interesting that condensing osteitis of the ilium and pubis also occur in women of child-bearing age and have similar radiographic appearance, but no definite associations have been made.[141,172]

Treatment has included antiinflammatory medications, radiation, antibiotics, and surgical excision.[11,23,37,63,116] Varying results have been reported, with complete clinical and radiographic remission reported with antiinflammatory medications.[14] Excisional biopsy is reserved for refractory cases.[23]

Friedrich's Disease

Avascular necrosis of the medial end of the clavicle has been termed Friedrich's disease owing to its original description in 1924 by Friedrich.[70] Its original description included two cases and highlighted the similarities between this condition and other osteonecrotic lesions such as Perthes' disease, Köhler's disease, Kienböck's disease, and Preiser's disease.

It is a rare condition presenting with pain and swelling over the medial end of the clavicle. It usually has an insidious onset and seems to be more common in females. Age of onset has varied from skeletally immature patients to a 58-year-old male.[36,107,127,194] It is usually unilateral but one bilateral case has been reported in a female, but the presentations were 2 years apart.[127]

Radiographs and tomograms reveal irregularity at the medial end of the clavicle with bony destruction. Bone scan usually reveals increased uptake.[194] The sedimentation rate may be slightly elevated, but other laboratory studies including aspirations have been negative, differentiating it from other osteolytic lesions such as infection.[127] Incisional biopsies reveal necrotic bone with empty lacunae.[127]

Most patients will improve within several months with nonoperative treatment with antiinflammatory medications. Although some have been treated successfully with curettage, this was felt to be unnecessary, and simple observation is advocated.[127] Resection arthroplasty has been reported successfully in one case, but it should be reserved for refractory cases.[129]

Infection

Sternoclavicular joint infections account for 1% of all joint infections in the general population but 17% of those found in IV drug abusers.[41,75,181,221] Ross and Shamsuddin[181] reviewed 180 cases of sternoclavicular joint infections and found that predisposing factors included IV drug abuse in 21%, distant infection in 15%, diabetes in 13%, trauma in 12%, and central line placement/infection in 9%. Interestingly, 23% were in otherwise healthy adults, which has been found in other studies as well.[8,17,84] This highlights the importance of a heightened clinical suspicion, even in those patients without a known risk factor. Immunosuppression secondary to concomitant diseases such as cancer, inflammatory arthritides, or HIV or secondary to steroid use are also known risk factors for infection.[181]

The most common isolated organism is *Staphylococcus aureus* even in IV drug abusers.[8,26,30,163,181,197] There are many case reports of isolated infections of the sternoclavicular joint that have been caused by a variety of other microorganisms, including *Escherichia coli*,[44] *Citrobacter diversus*,[72] *Pasteurella multocida*,[34] *Streptococcus pyogenes*,[143] *Pseudomonas aeruginosa*,[108] *Brucella species*,[13] *Neisseria gonorrhoeae*,[142] *Bacteroides*,[169] *Fusiformbacterium*,[122] *Candida albicans*,[54] *Haemophilus influenza*,[115] and Group B streptococcal species.[31] Blankstein and associates reported a septic sternoclavicular joint that cultured *S. aureus* secondary to bacteremia infection from a paronychia of the finger.[17] Richter and associates reported on nine patients with infection of the sternoclavicular joint secondary to tuberculosis.[175] The average time from onset of the disease until diagnosis was 1.4 years. Higoumenakis reported that unilateral enlargement of the sternoclavicular joint is a diagnostic sign of congenital syphilis.[87] The enlargement of the sternoclavicular joint can be mistaken for an anterior dislocation. He reported the sign to be positive in 170 of 197 cases of congenital syphilis. Others have reported on the same condition.[15,49,193] The enlargement is a hyperostosis of the medial clavicle, occurring in the sternoclavicular joint of the dominant extremity, which reaches its permanent stage and size at puberty. The theory of why it affects the sternoclavicular joint relates it to spirochete invasion of the sternal end of the clavicle at the time of early ossification.

Patients most commonly present with pain and swelling localized to the SC joint. In a review by Ross et al., 78% presented with chest pain and 24% with shoulder pain, emphasizing that the pain can radiate. Fever was also common, with 62% having concomitant bacteremia.[181]

A high index of suspicion should be maintained as complications are high, with osteomyelitis occurring in 55%, abscess formation in at least 20%, mediastinitis in 13%, and pleural effusion.[30,181,190,198,213,221] Plain radiographs are helpful but do not exclude the diagnosis, as 80% were read as normal in one series.[26] A CT scan or MRI is mandatory to evaluate the joint and surrounding bones and retrosternal area. The CT scan will reveal bony destruction and, frequently, gas or abscess formation (Figs. 33-11 and 33-12).

Treatment has varied from parenteral use of antibiotics to aggressive en bloc resection of the SC joint with or

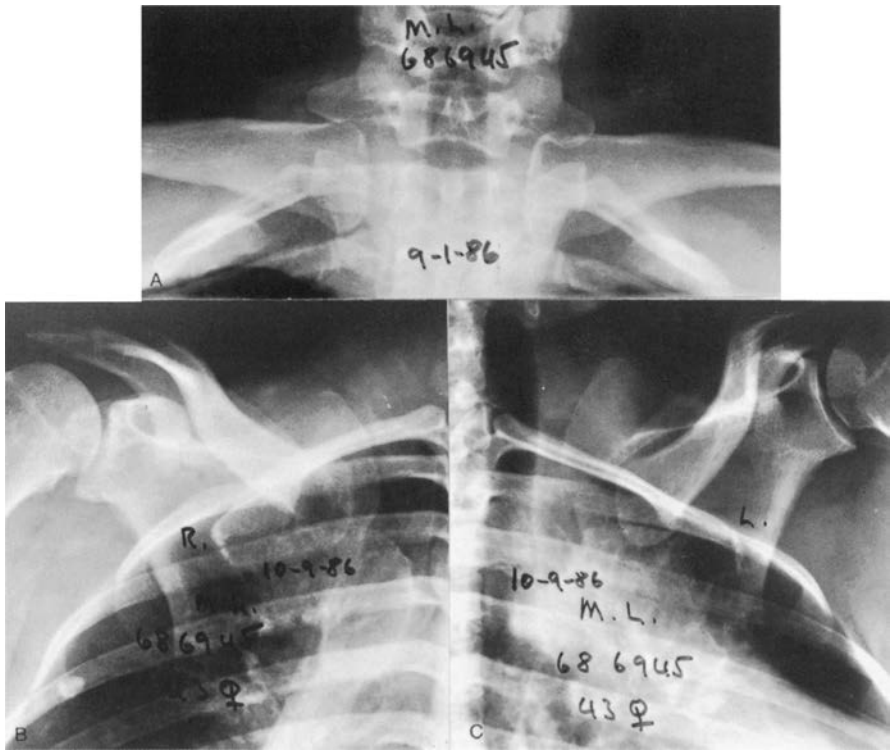


Figure 33-11 Infection in the right sternoclavicular joint. **(A)** On the initial x-ray, there is little difference between the right and left medial clavicles, as seen on the 30-degree cephalic tilt view. **(B,C)** Thirty-eight days later, the medial end of the right clavicle is seen to be dissolving, compared with the medial end of the left clavicle. The patient had a *Staphylococcus aureus* infection in the right sternoclavicular joint, which was managed by open débridement. (Reprinted with permission from Rockwood CA, Matsen F III, eds. *The shoulder.* Philadelphia: WB Saunders, 1990:Fig. 13-14.)

without pectoralis flap augmentation. More aggressive surgical treatment has recently been advocated due to failures of simple débridement.[2630] The relatively small joint capsule of the SC joint allows for minimal distention of purulent fluid and rapid extension beyond the joint. If the infection is isolated to the SC joint without any bony destruction, then simple irrigation and débridement may be performed with good results. The results do seem better if a delayed closure is performed or the wound is allowed to heal by secondary intention.[8,26] If there is bony involvement, then more aggressive treatment is advocated. Song et al. reported recurrence of infection in five of six patients treated with irrigation and débridement alone. These were then revised to en bloc resection of the SC joint and portion of the first rib with pectoralis major advancement, with a cure in these cases.[197] Burkhart et al. reviewed 26 patients, two-thirds of whom required en bloc resection. Fifteen percent had a previous irrigation and débridement. A pectoralis major muscle advancement into the defect based on the thoracoacromial pedicle was performed in 55% with excellent results.[26] Carlos et al. also advocated aggressive treatment if the infection had spread beyond the SC joint.[30] The primary author currently performs these procedures in conjunction with a thoracic surgeon and prescribes 6 weeks of postoperative antibiotics.

In summary, there should be a high index of clinical suspicion for an SC joint infection if a patient presents with pain and swelling, even if there are no other risk factors. Routine serologic examination should be performed including blood cultures. A CT scan or MRI will help determine if there is extension beyond the SC joint, and if present, more aggressive surgical treatment is recommended.

Osteoarthritis

Osteoarthritis is characterized by narrowing of the joint space, osteophytes, subchondral sclerosis, and cysts on both sides of the joint.[112,114,123] Because most of the wear occurs in the inferior part of the head of the medial clavicle, most of the degenerative changes occur in that region. The sometimes discrete degenerative changes are best seen on tomograms and CT scans.[112,114] (Figs. 33-13, 33-14, and 33-15) Kier and associates correlated the x-ray films and the pathologic specimens of patients with osteoarthritis of the sternoclavicular joint.[112] They noted increasing frequency of degenerative changes with age, with 90% to 100% of specimens over the age of 70 having significant degenerative changes. Sternoclavicular joint arthritis and hypertrophy can develop following radical neck surgery, particularly when the spinal accessory nerve is sacrificed, and the incidence is reported to be as high as 54%.[78,160,208] The reason for the arthritis is the downward and forward droop of the shoulder, which puts extra stress on the sternoclavicular joint. The senior author observed one patient who had such stress on the sternoclavicular joint following a radical neck and spinal accessory nerve sacrifice that he developed a posterior dislocation of the sternoclavicular joint. The posterior displacement was so severe that the medial end of the clavicle compressed his trachea and esophagus.

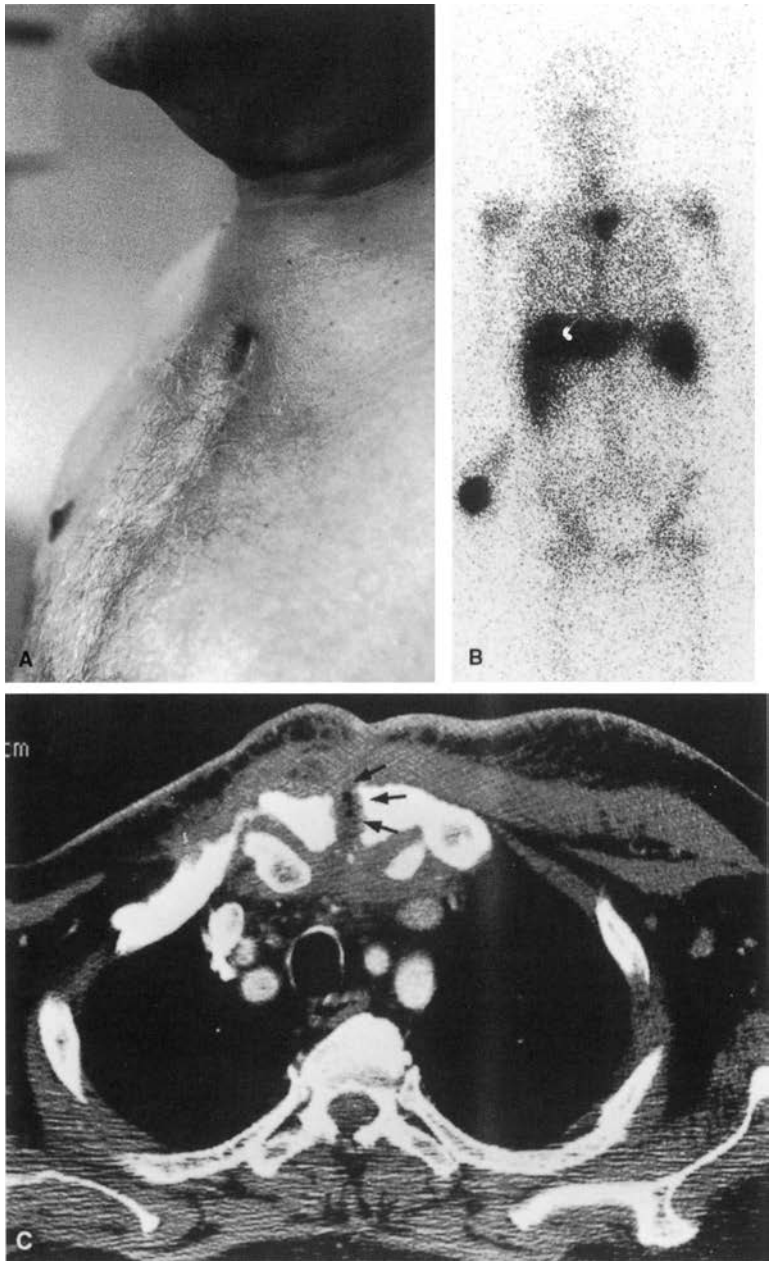


Figure 33-12 **(A)** This patient had long-standing insulin-dependent diabetes and had had a coronary artery bypass procedure that was complicated by a postoperative wound infection. **(B)** Indium-enhanced white blood cell scan consistent with infection in the region of the left sternoclavicular joint. **(C)** Computed tomography scan revealing significant soft tissue swelling, interspersed locules of air within the sternal osteotomy site, and focal irregularity of the posterior aspect of the sternum consistent with an infectious process. (From Wirth MA, Rockwood CA. Injuries to the sternoclavicular joint. In: Rockwood CA, Green DP, Bucholz RW, Hechman JD, eds. *Fractures in adults.* Philadelphia: JB Lippincott, 1996.)

Bremner and Bonnin have both reported on "postmenopausal arthritis," so named because it is most often seen in postmenopausal women.[18,20] Sadr and Swann reported on 22 patients with this problem who were seen in a 5-year study; 20 of the cases were in women, and the majority involved the sternoclavicular joint of the dominant arm.[185] Nonoperative treatment was recommended. The condition is the result of normal degeneration of a frequently moved joint. It is almost without symptoms; a lump develops at the sternoclavicular joint, which occasionally is accompanied by a vague ache (Fig. 33-16). There is no previous history of injury or disease. X-ray studies show sclerosis and enlargement of the medial end of the clavicle, reactive sclerosis of the sternum, and subluxation of the joint. The pathologic changes are those of degenerative arthritis.

In general, standard osteoarthritis and postmenopausal arthritis are managed nonoperatively with nonsteroidal antiinflammatory drugs and activity modification. If that fails, then operative intervention can be considered. Several authors have reported on isolated resection of the medical end of the clavicle with good results.[20,161,177] It is important to note that if there is concomitant instability of the sternoclavicular joint, then a resection arthroplasty will not yield a good result. Rockwood and associates reported on a series of 23 patients who had undergone a resection of the medial end of the clavicle.[177] The patients were divided into two groups: group I, who underwent resection

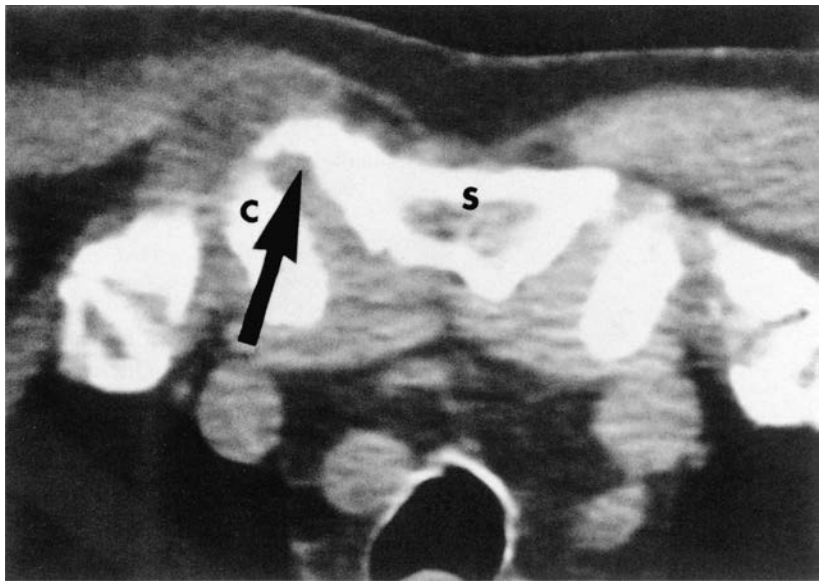


Figure 33-13 Computed tomography scan of a 63-year-old right hand–dominant man who worked more than 45 years as a landscaper. Image demonstrates a hypertrophic, degenerative right sternoclavicular joint consistent with osteoarthritis (*arrow*). C = medial clavicle; S = sternum. (Reprinted with permission from Wirth MA, Rockwood CA. Acute and chronic traumatic injuries of the sternoclavicular joint. *J Am Acad Orthop Surg* 1996;4:268–278.)

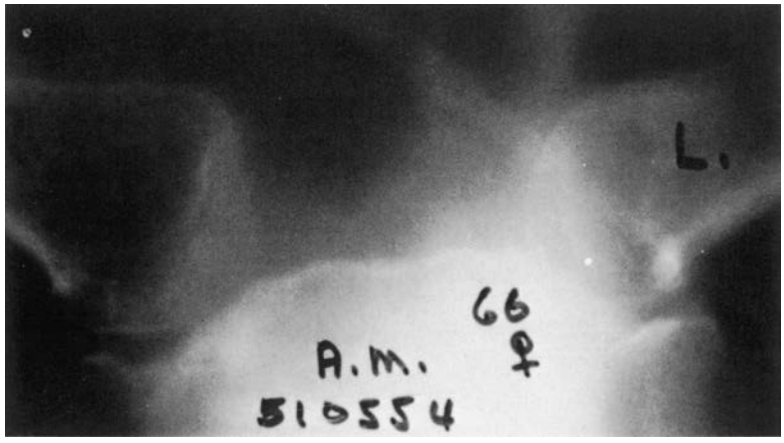


Figure 33-14 Tomogram of the sternoclavicular joints in a 66-year-old patient with degenerative arthritis. (Reprinted with permission from Rockwood CA, Matsen F III, eds. *The shoulder.* Philadelphia: WB Saunders, 1990:Fig. 13-11.)

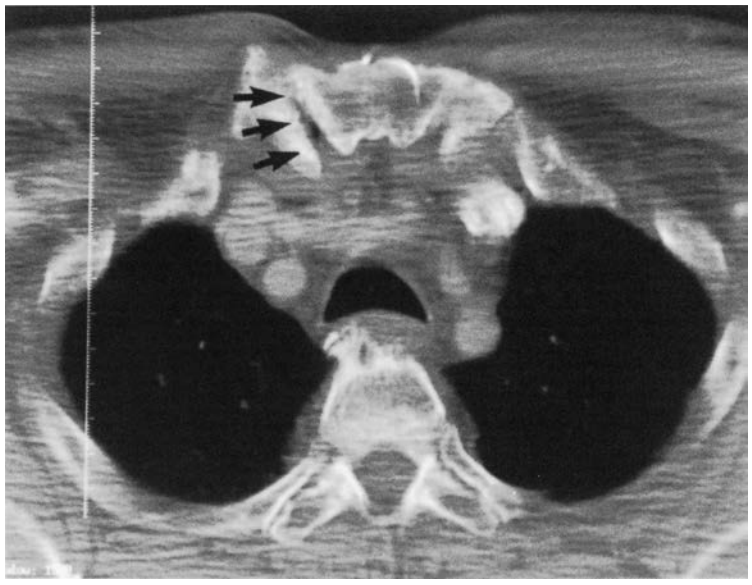


Figure 33-15 Computed tomography scan of a 69-year-old patient with degenerative arthritis. The patient recalled that he injured his right sternoclavicular joint approximately 50 years earlier, while completing an obstacle course during basic training for the armed services.

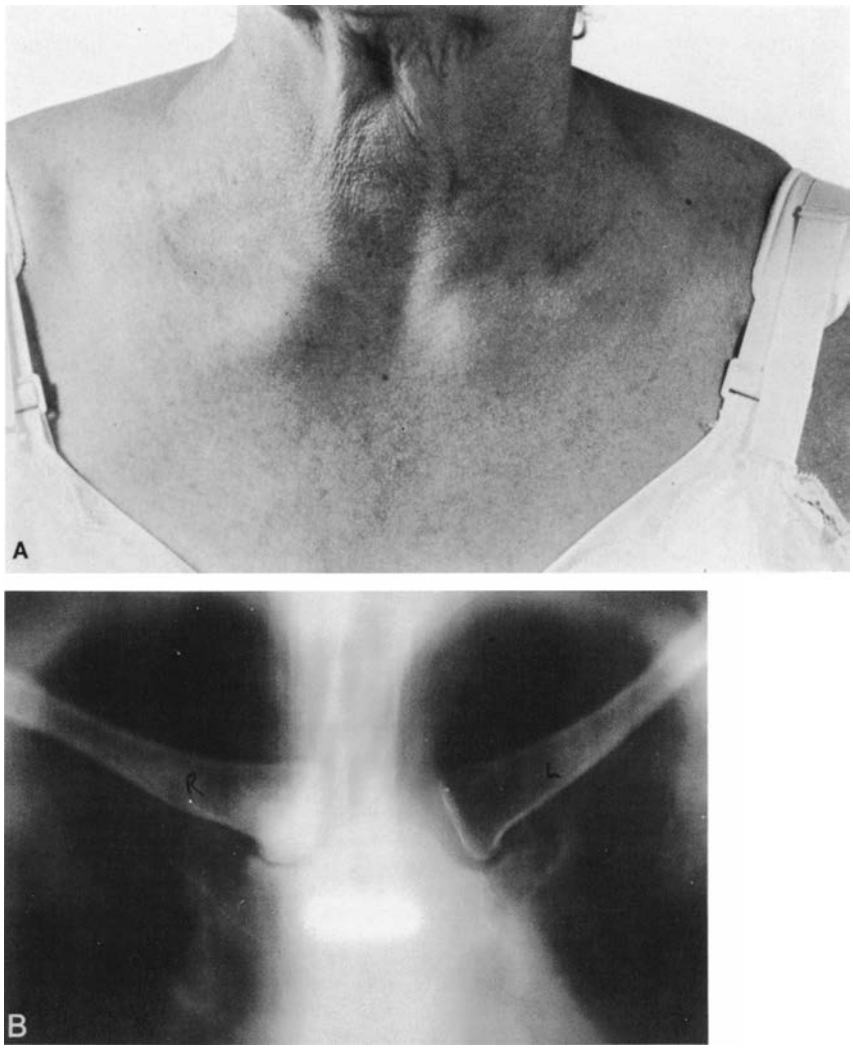


Figure 33-16 **(A)** Bilateral anterior swelling of the sternoclavicular joints in a 67-year-old woman. The right medial clavicle was more prominent because she was right-handed. **(B)** The tomogram demonstrates sclerosis and degenerative changes in the right sternoclavicular joint consistent with ordinary degenerative arthritis. (**B** reprinted with permission from Rockwood CA, Matsen F III, eds. *The shoulder.* Philadelphia: WB Saunders, 1990:Fig. 13-13b.)

of the medial end of the clavicle with maintenance or reconstruction of the costoclavicular ligament; and group II, who had a resection without maintaining or reconstructing the costoclavicular ligament. The outcome in all but one of the seven patients in group II was poor, with persistence or worsening of preoperative symptoms. The only patient of this group with a successful result experienced a posterior epiphyseal separation, in which the costoclavicular ligament remained attached to the periosteum, thus preventing instability. All of the eight patients in group I, who underwent primary surgical resection of the medial end of the clavicle with maintenance of the costoclavicular ligaments, had an excellent result. When the operation was performed as a revision of a previous procedure with reconstruction of the costoclavicular ligaments, the results were less successful, but only one patient of seven was not satisfied with the outcome of treatment.

When operating on the sternoclavicular joint, care must be taken to evaluate the residual stability of the medial clavicle. It is the same analogy as used when resecting the distal clavicle for an old acromioclavicular joint problem. If the coracoclavicular ligaments are intact, an excision of the distal clavicle can be performed (Fig. 33–17). If the coracoclavicular ligaments are insufficient, then in addition to excision of the distal clavicle, one must reconstruct the coracoclavicular ligaments or the capsular ligaments. Stabilization techniques are described in the trauma section of this chapter. If too much clavicle is resected, or if the clavicle is not stabilized, an increase in symptoms can occur (Fig. 33-18). The technique for resection of the medical end of the clavicle involves only resecting enough bone so that there is about 1 cm of space between the medial end of the clavicle and the sternum. The resection should never be carried more lateral than the costoclavicular ligament or the insertions of the sternoclavicular ligament/joint capsules.[16] The anterior sternoclavicular ligament/joint capsule should be repaired over the resection site. Postoperative therapy parallels that of a resection of the lateral end of the clavicle.

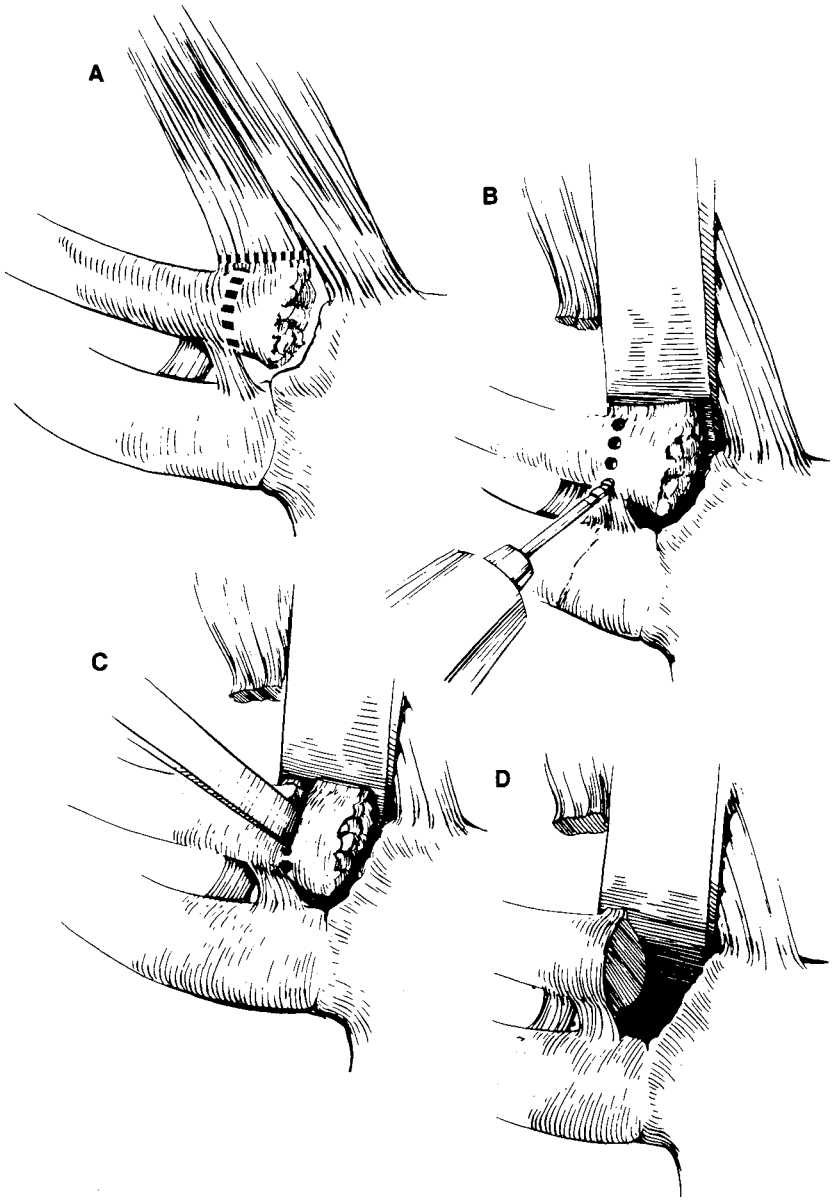


Figure 33-17 Technique for resecting the medial clavicle for degenerative arthritis. **(A)** Care must be taken to remove only that part of the clavicle medial to the costoclavicular (rhomboid) ligaments. There must be adequate protection for the vital structures that lie posterior to the medial end of the clavicle. **(B,C)** An air drill with a side-cutting burr can be used to perform the osteotomy. **(D)** When the fragment of bone has been removed, the dorsal and anterior borders of the clavicle should be smoothed, to give a better cosmetic appearance.

Tietze's Syndrome

Tietze first described the condition in 1921; it is also known as costosternal syndrome, parasternal chondrodynia, costochondritis, and thoracochondralgia.[211] Tietze's syndrome is a benign, self-limiting condition characterized by tender swelling in the region of the anterior chest wall without any other clear evidence that could establish another definite diagnosis.[126,211] Thus, it is often a diagnosis of exclusion.

Although the reported incidence has diminished over time, Jurik and Graudal reviewed the literature in 1988 and found 346 cases.[104] The exact incidence is unknown, but in a group of 320 patients evaluated for chest pain, 10% were found to have Teitze's syndrome.[222]

Patients present with pain in the anterior chest wall with fusiform swelling of the involved costal cartilage. The pain may radiate to the shoulder or arm and is frequently aggravated by coughing or sneezing. The most commonly involved area is the costosternal junction of the second rib. There is usually localized tenderness and warmth, but erythema is usually absent.

Chest radiographs are usually normal, but a bone scan will usually reveal increased uptake in the area. Yang et al. described the use of pinhole skeletal scintigraphy with the appearance of a club-like "C" or an inverted "C" at the affected cartilage.[227] A CT scan frequently reveals enlargement of the cartilage at the sternal junction, but biopsies demonstrate normal cartilage.[21,53]

Clearly the most important aspect of the condition is ruling out a primary cardiac or pulmonary cause necessitating referral to medical specialists. Once that is confirmed, the patient should be reassured that this is a

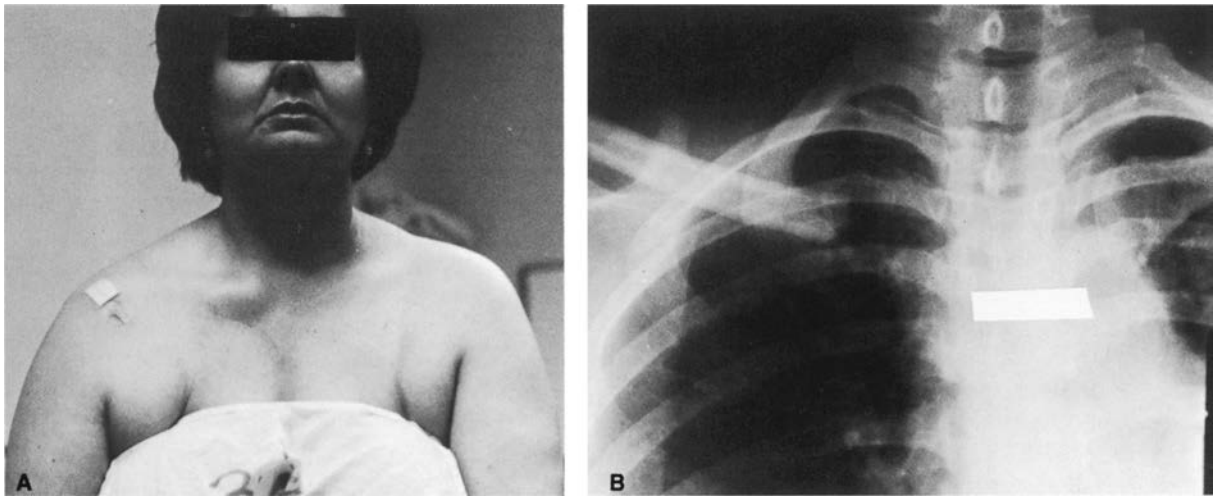


Figure 33-18 **(A)** This postmenopausal, right-handed woman had resection of the right medial clavicle, because of preoperative diagnosis of "possible tumor." The postoperative microscopic diagnosis was degenerative arthritis of the right medial clavicle. After surgery, the patient complained of pain and discomfort, marked prominence, and gross instability of the right medial clavicle. **(B)** An x-ray confirmed that the excision of the medial clavicle extended lateral to the costoclavicular ligaments; hence, the patient had an unstable medial clavicle.

benign condition that is often characterized by relapsing and remitting symptoms. The discomfort is managed nonoperatively with nonsteroidal antiinflammatory drugs and judicious use of corticosteroid injections.

Other Arthropathies

Many other arthropathies and maladies affecting the sternoclavicular joint have been reported. The sternoclavicular joint can be involved with systemic autoimmune arthropathies such as rheumatoid arthritis, psoriatic arthritis, ankylosing spondyloarthritis, gout, and polymyalgia rheumatica.[2,148,173,174,188,206] Hypertrophy after radial neck dissection has also been reported and might be due to increased stress across the joint with trapezial dysfunction.[78,160,208] Neuropathic changes in the sternoclavicular joint have also been noted with a syringomyelia.[35] Synovial osteochondromatosis has also been reported by Vrdojak and Irha.[216] Cameron et al. reported hemodialysis-related amyloid deposition in the sternoclavicular joint.[29] The sternomanubrial joint may also be affected by psoriatic arthritis, and fusion of this joint can be performed when it is refractory to conservative management.

RADIOGRAPHIC FINDINGS

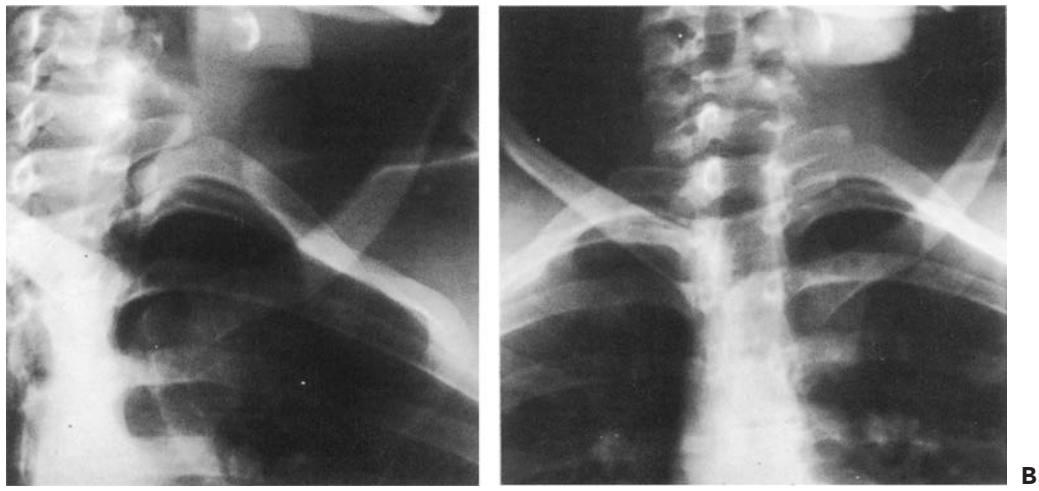
Anteroposterior Views

The older literature reflects that routine x-rays of the sternoclavicular joint, regardless of the special views, are difficult to interpret. Special oblique views of the chest have been recommended, but because of the distortion of one clavicle over the other, interpretation is difficult (Fig. 33-19). The older literature also suggests that the diagnosis of dislocation of the sternoclavicular joint is best made from a clinical examination, not from the x-rays. However, it does indicate that tomography offers more detailed information, often showing small fractures in the vicinity of the sternoclavicular joint. Occasionally, the routine anteroposterior or posteroanterior x-rays of the chest or sternoclavicular joint suggest that something is wrong with one of the clavicles, because it appears to be displaced as compared with the normal side (Figs. 33-20 and 33-21). It would be ideal to take a view at right angles to the anteroposterior plane, but because of our anatomy, it is impossible to obtain a true 90-degree cephalic-to-caudal lateral view. Lateral x-rays of the chest are at right angles to the anteroposterior plane, but they cannot be interpreted because of the density of the chest and the overlap of the medial clavicles with the first rib and the sternum.

Regardless of a clinical impression that suggests an anterior dislocation, x-rays must be obtained to confirm one's suspicions.

Special Projected Views

Kattan[105] has recommended a special projection, as have Ritvo,[176] Fery and Leonard,[64] Tricoire and coworkers.[212] Kurzbauer has recommended special lateral projections.[119] Hobbs, in 1968, recommended a view that comes close to being a 90-degree cephalocaudal lateral view of the sternoclavicular joints.[88] In the same year, Heinig recommended an x-ray projection of the sternoclavicular joint that resembles a "swimmer's view" of the cervical spine.[86]



A B

Figure 33-19 Routine radiographs of the sternoclavicular joint are difficult to interpret, even with a classic posterior dislocation of the joint.

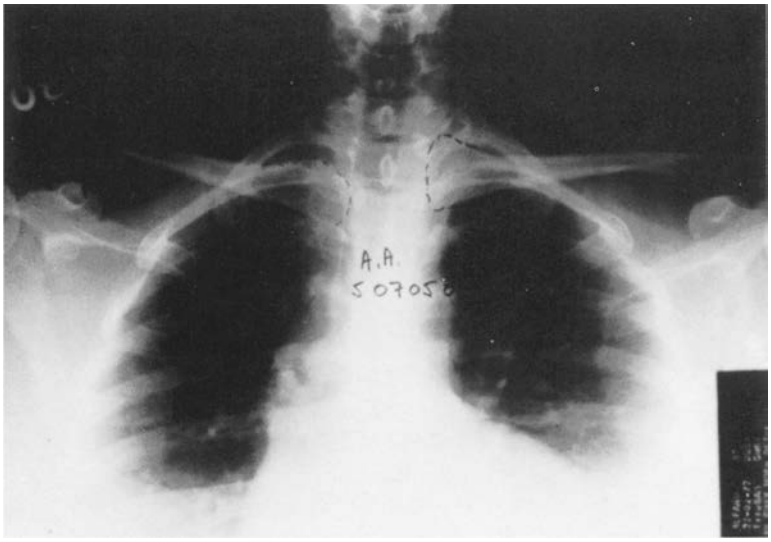


Figure 33-20 Anteroposterior x-ray of the sternoclavicular joints with anterosuperior displacement of the left medial clavicle. The displacement is quite noticeable when the clavicles are outlined.

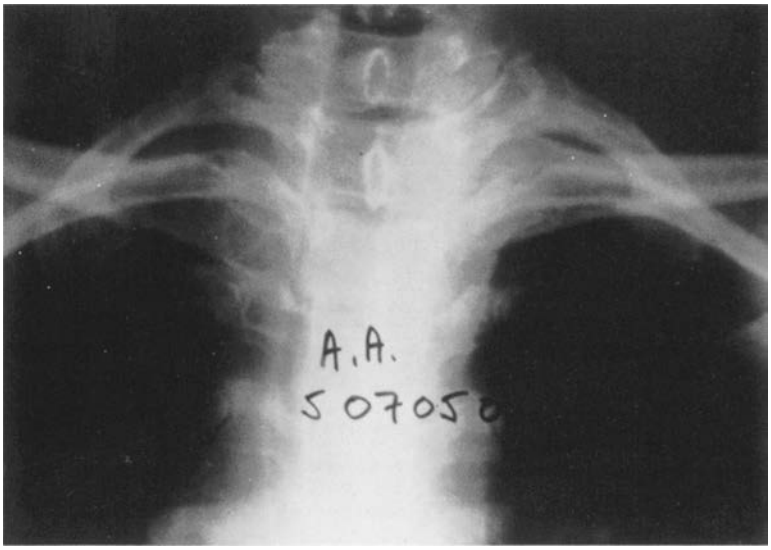
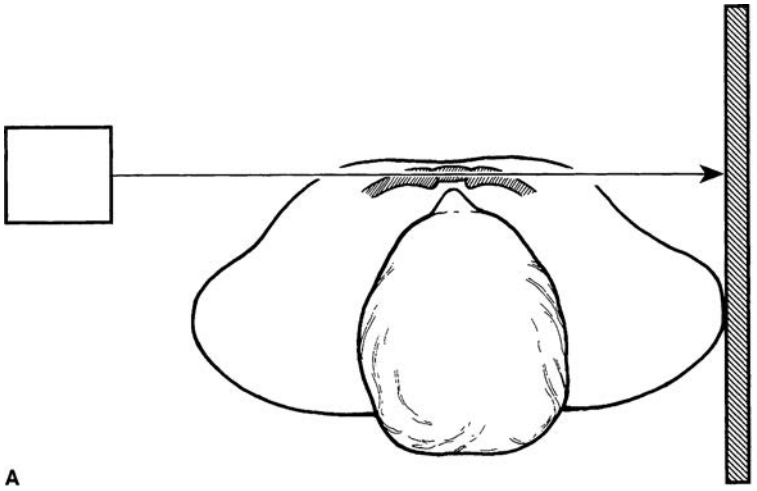


Figure 33-21 Anteroposterior x-ray of the sternoclavicular joints with anterosuperior displacement of the left medial clavicle. The displacement is not as noticeable when the clavicles are not outlined.



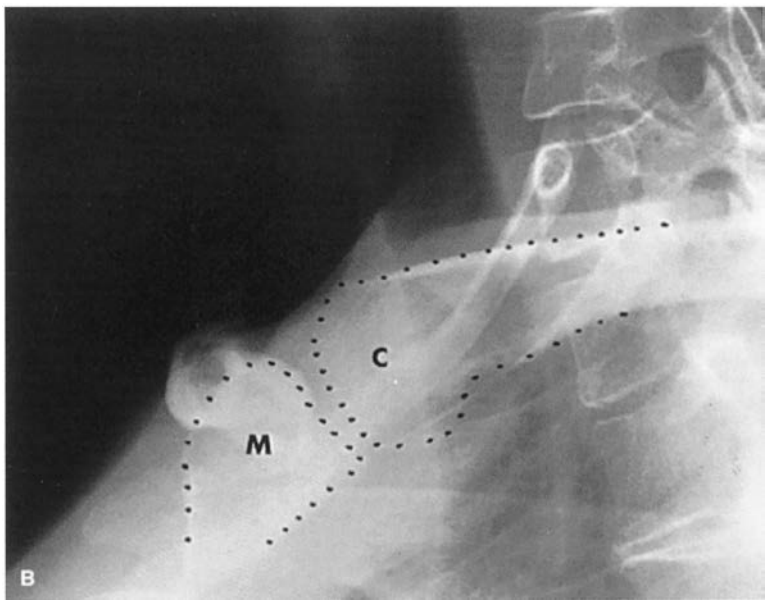


Figure 33-22 **(A)** Positioning of the patient for x-ray evaluation of the sternoclavicular joint, as described by Heinig. **(B)** Heinig view demonstrating a normal relationship between the medial end of the clavicle (*C*) and the manubrium (*M*). (From Wirth MA, Rockwood CA. Injuries to the sternoclavicular joint. In: Rockwood CA, Green DP, Bucholz RW, Hechman JD, eds. *Fractures in adults*. Philadelphia: JB Lippincott, 1996.)

Heinig View

With the patient in a supine position, the x-ray tube is placed approximately 30 in. from the involved sternoclavicular joint, and the central ray is directed tangential to the joint and parallel to the opposite clavicle. The cassette is placed against the opposite shoulder and centered on the manubrium (Fig. 33-22).

Hobbs View

In the Hobbs view, the patient is seated at the x-ray table, high enough to lean forward over the table. The cassette is on the table, and the lower anterior rib cage is against the cassette (Fig. 33-23). The patient leans forward, so that the nape of his or her flexed neck is almost parallel to the table. The flexed elbows straddle the cassette and support the head and neck. The x-ray source is above the nape of the neck, and the beam passes through the cervical spine to project the sternoclavicular joints onto the cassette.

Serendipity View

The serendipity view is rightfully named because that is the way it developed. The senior author, accidentally, noted that the next best thing to having a true cephalocaudal lateral view of the sternoclavicular joint is a 40-degree cephalic tilt view. The patient is positioned on his or her back squarely and in the center of the x-ray table. The tube is tilted at a 40-degree angle off the vertical and is centered directly on the sternum (Fig. 33-24). A nongrid 11 $\times$ 14-in. cassette is placed squarely on the table and under the patient's upper shoulders and neck, so that the beam aimed at the sternum will project both clavicles onto the

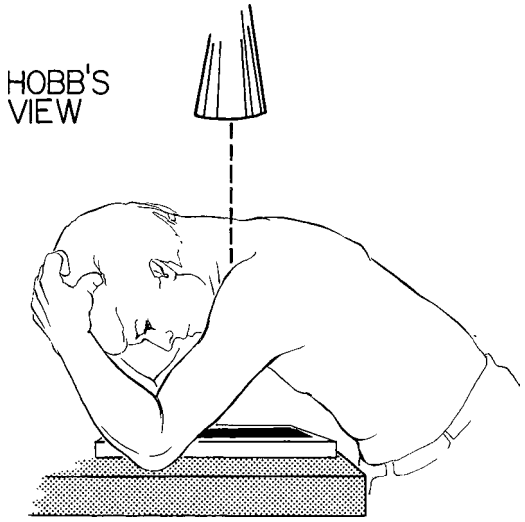


Figure 33-23 Positioning of the patient for x-ray evaluation of the sternoclavicular joint, as recommended by Hobbs. (Redrawn with permission from Hobbs DW. The sternoclavicular joint: a new axial radiographic view. *Radiology* 1968;90:801.)

film. The tube is adjusted so that the medial half of both clavicles is projected onto the film. It is important to note that the cassette should be placed squarely on the x-ray table (i.e., not angulated or rotated) and that the patient should be positioned squarely on top of the cassette.

For children, the distance from the tube to the cassette is 45 in.; for adults, whose anteroposterior chest diameter is greater, the distance should be 60 in. The technical setting of the machine is essentially the same as for a posteroanterior view of the chest.

To understand this view, imagine that your eyes are at the level of the patient's knees and you are looking up toward his or her clavicles at a 40-degree angle. If the right sternoclavicular joint is dislocated anteriorly, the right clavicle will appear to be displaced more anteriorly or riding higher on an imaginary horizontal line when compared with the normal left clavicle (Fig. 33-25). The reverse is true if the left sternoclavicular joint is dislocated posteriorly (i.e., the left clavicle displaced inferiorly or riding lower on an imaginary horizontal plane than the normal right clavicle) (see Fig. 33-27). The idea, then, is to take a 40-degree cephalic tilt x-ray showing both medial clavicles and compare the injured clavicle with the normal clavicle (Fig. 33-26).

Special Techniques

Tomograms

Tomograms can be very helpful in distinguishing between a sternoclavicular dislocation and a fracture of the medial clavicle. They are also helpful in questionable anterior and posterior injuries of the sternoclavicular joint—to distinguish fractures from dislocations and to evaluate arthritic changes (Fig. 33-27). Tomograms have essentially been supplanted by computed tomography and are mentioned for completeness sake.

CT Scans

Without question, the CT scan is the best technique to study problems of the sternoclavicular joint (Fig. 33-28). It clearly distinguishes injuries of the joint from fractures of the medial clavicle and defines minor subluxations of the joint. The orthopedist must remember to ask for CT scans of both sternoclavicular joints and the medial half

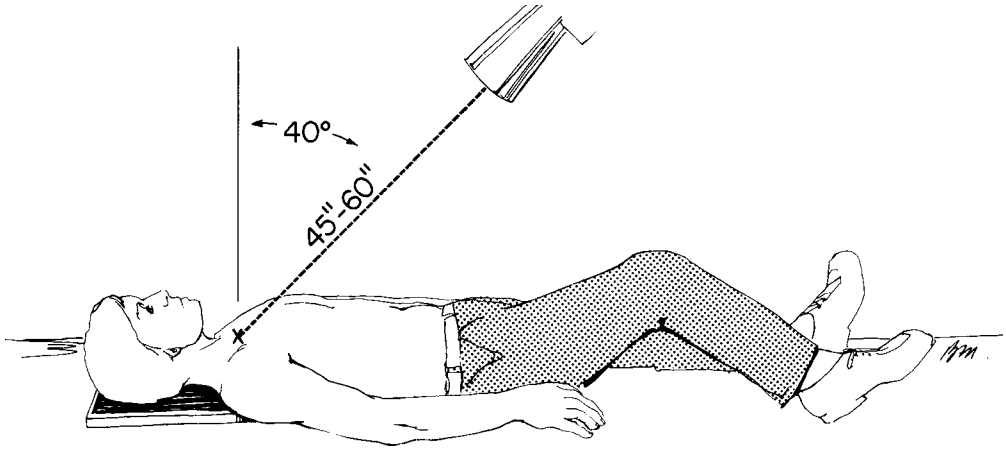


Figure 33-24 Positioning of the patient to take the serendipity view of the sternoclavicular joints. The x-ray tube is tilted 40 degrees from the vertical position and aimed directly at the manubrium. The nongrid cassette should be large enough to receive the projected images of the medial halves of both clavicles. In children, the tube distance from the patient should be 45 in.; in thicker-chested adults, the distance should be 60 in.

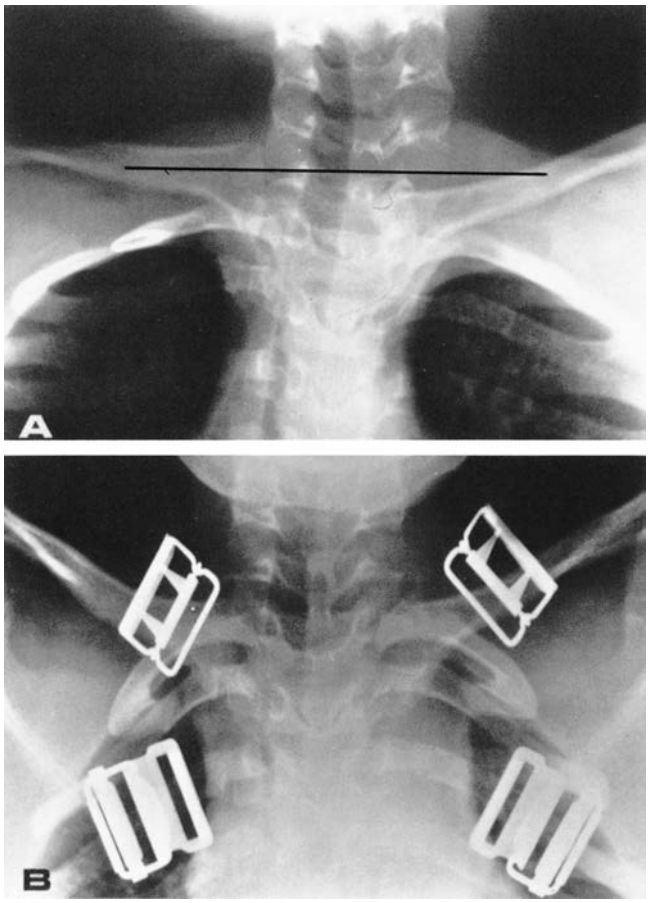


Figure 33-25 **(A)** Posterior dislocation of the left sternoclavicular joint as seen on a 40-degree caphalic tilt x-ray film of a 12-year-old boy. The left clavicle is displaced inferiorly to a line drawn through the normal right clavicle. **(B)** After closed reduction, the medial ends of both clavicles are in the same horizontal position. The buckles are part of the figure-of-eight clavicular harness used to hold the shoulders back after reduction.

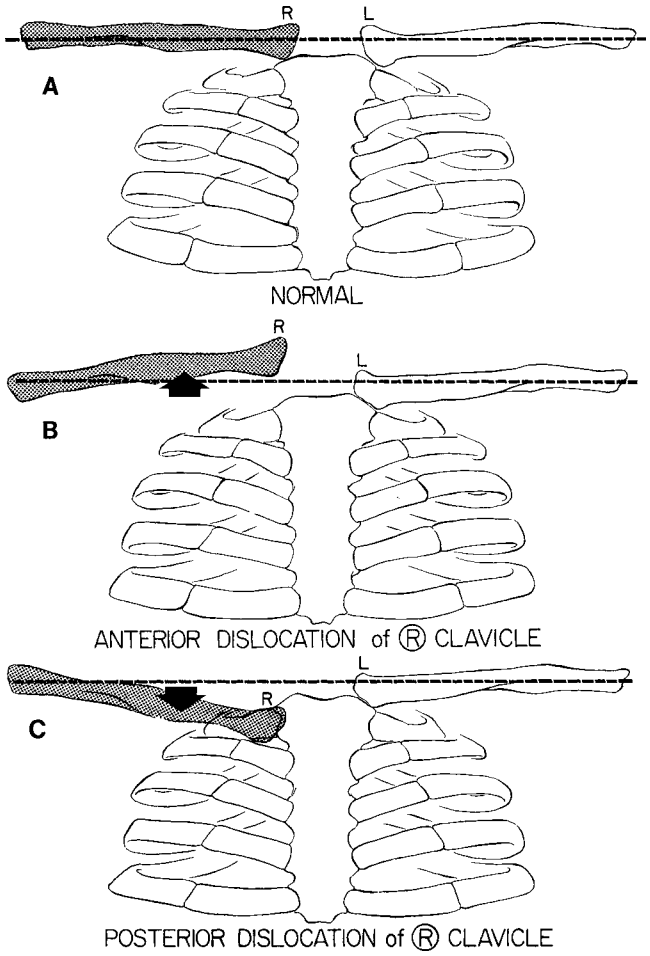


Figure 33-26 Interpretation of the cephalic tilt x-ray films of the sternoclavicular joints. **(A)** In the normal person, both clavicles appear on the same imaginary line drawn horizontally across the film. **(B)** In a patient with anterior dislocation of the right sternoclavicular joint, the medial half of the right clavicle is projected above the imaginary line drawn through the level of the normal left clavicle. **(C)** If the patient has a posterior dislocation of the right sternoclavicular joint, the medial half of the right clavicle is displaced below the imaginary line drawn through the normal left clavicle.

of both clavicles, so that the injured side can be compared with the normal side. The patient should lie flat in the supine position. If one requests a study of the right sternoclavicular joint, the x-ray technician may rotate the patient to the affected side and provide views of only the one joint.

STERNOCLAVICULAR SUBLUXATIONS AND DISLOCATIONS

The section will review traumatic, atraumatic, and developmental subluxations and dislocations of the sternoclavicular joint. The direction of subluxation or dislocation is based on the location of the medial end of the clavicle with respect to the sternum. Although subluxations and dislocations can have an inferior or superior component, the majority of the displacement occurs in an anterior or posterior direction. By convention, they are therefore termed anterior or posterior.

Sternoclavicular injuries are rare, and many of the authors apologize for reporting only three or four cases. Attesting to this rarity is the fact that some orthopedists have never treated or seen a dislocation of the sternoclavicular joint.

The incidence of sternoclavicular dislocation, based on the series of 1,603 injuries of the shoulder girdle reported by Cave and associates, is 3%.[33] (Specific incidences in the study were glenohumeral dislocations, 85%; acromioclavicular, 12%; and sternoclavicular, 3%). In the series by Cave and in our experience, dislocation of the sternoclavicular joint is not as rare as posterior dislocation of the glenohumeral joint.

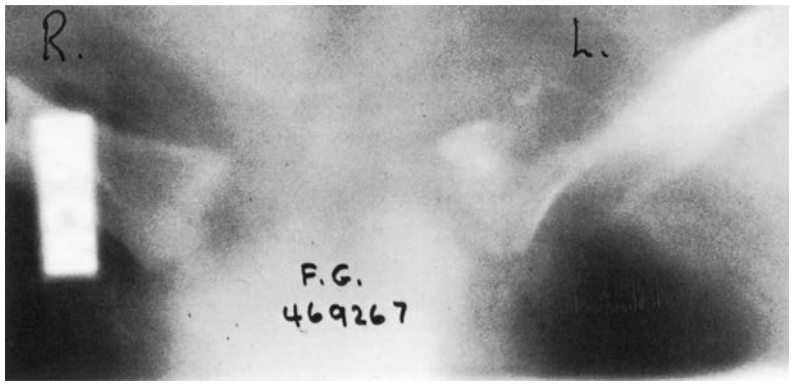


Figure 33-27 Tomogram demonstrating a fracture of the left medial clavicle. The clinical diagnosis before radiography was an anterior dislocation of the left sternoclavicular joint.

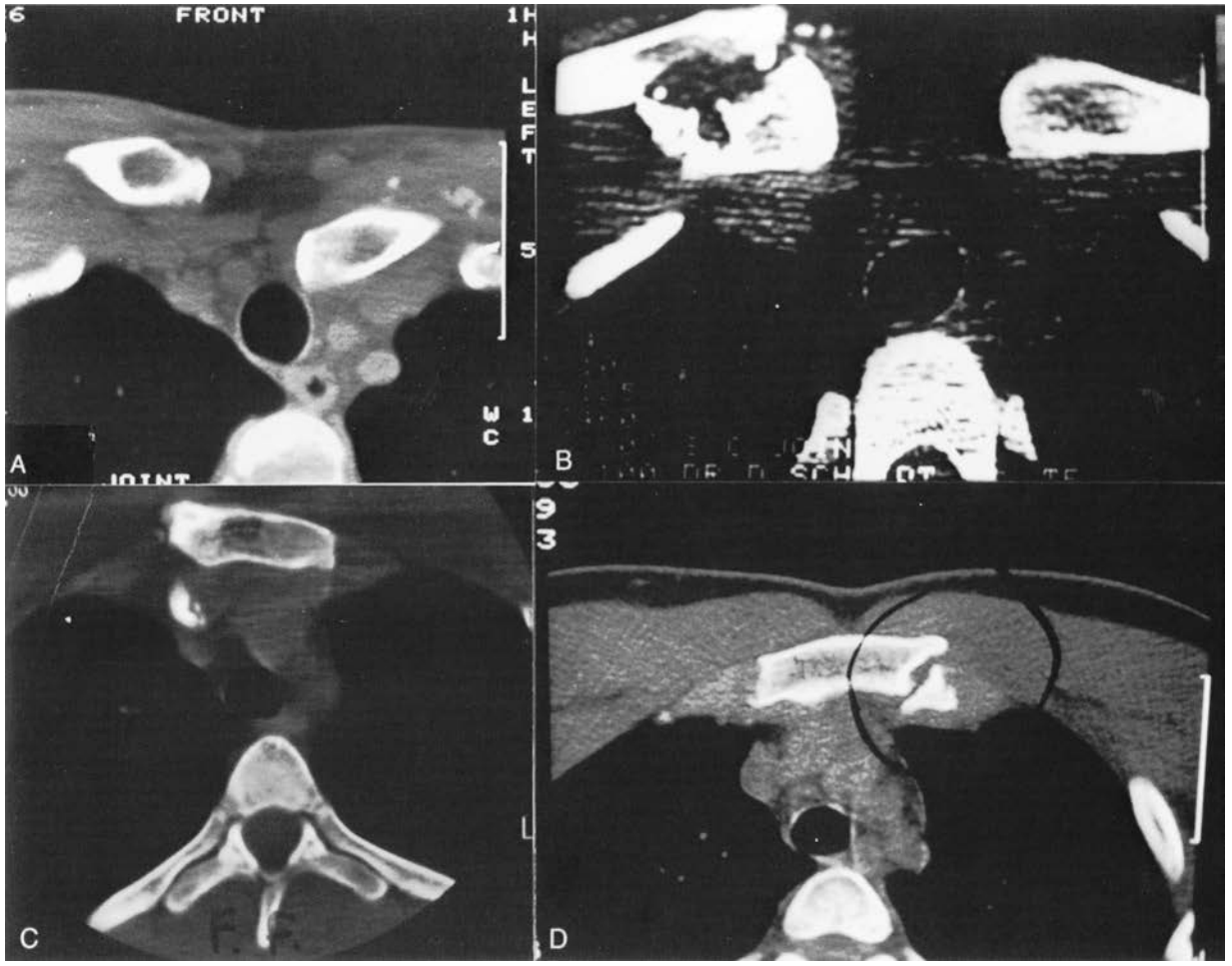


Figure 33-28 Computed tomography scans of the sternoclavicular joint, demonstrating various types of injuries. **(A)** Posterior dislocation of the left clavicle compressing the great vessels and producing swelling of the left arm. **(B)** Fracture of the medial clavicle that does not involve that articular surface. **(C)** Fragment of bone displaced posteriorly into the great vessel. **(D)** Fracture of the medial clavicle into the sternoclavicular joint. (Reprinted with permission from Rockwood CA, Matsen F III, eds. *The shoulder.* Philadelphia: WB Saunders, 1990:Fig. 13-22.)

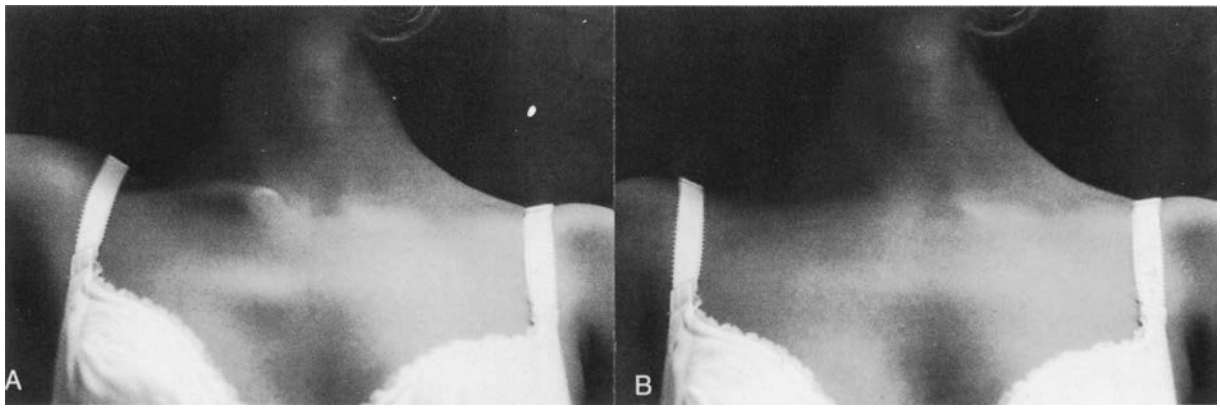


Figure 33-29 Spontaneous anterior subluxation of the sternoclavicular joint. **(A)** With the arms in the overhead position, the medial end of the right clavicle spontaneously subluxates anteriorly without any trauma. **(B)** When the arm is lowered to the side, the medial end of the clavicle spontaneously reduces. Usually this is not associated with significant discomfort. (Reprinted with permission from Rockwood CA, Matsen F III, eds. *The shoulder.* Philadelphia: WB Saunders, 1990:Fig. 13-10.)

Congenital or Developmental Subluxation or Dislocation

Newlin reported a 25-year-old man who had bilateral congenital posterior dislocation of the medial ends of the clavicle that simulated an intrathoracic mass.[146] Guerin first reported congenital luxations of the sternoclavicular joint in 1841.[82] Nakazato et al. reported on a newborn with a concomitant clavicle fracture and posterior dislocation.[144] Congenital defects with loss of bone substance on either side of the joint can predispose the patient to subluxation or dislocation. Cooper et al. described a patient with scoliosis so severe that the shoulder was displaced forward enough to posteriorly dislocate the clavicle behind the sternum.[38]

Atraumatic or Spontaneous Subluxations and Dislocations

As with classification of glenohumeral joint instability, the importance of distinguishing between traumatic and atraumatic instability of the sternoclavicular joint must be recognized if complications are to be avoided. The vast majority of spontaneous or atraumatic dislocations are anterior, with only a few cases reported in the literature of atraumatic posterior dislocations.

Anterior

Rowe described several patients who had undergone one or more unsuccessful attempts to stabilize the sternoclavicular joint.[182] In all cases, the patient was able to voluntarily dislocate the clavicle after surgery. In addition, he has described several young patients who were able to "flip the clavicle out and back in" without elevation of the arms.

In our experience, spontaneous subluxations and dislocations of the sternoclavicular joint are seen most often in patients under 20 years of age, and more often in females. Sadr and Swann reported on 22 patients with atraumatic anterior dislocations.[185] The majority of patients were middle-aged females presenting with a "lump" in the anterior portion of the neck. In no case was the correct diagnosis made by the referring physician.

Without significant trauma, one or both of the medial clavicles spontaneously displace anteriorly during abduction or flexion to the overhead position (Fig. 33-29). The clavicle reduces when the arm is returned to the side. Patients seem to have only one symptom: The medial end of the clavicle subluxates or dislocates anteriorly when they raise their arms over their head. This occurs spontaneously and without any significant trauma. Many of these patients have the characteristic finding of generalized ligamentous laxity (i.e., hyperextension of the elbows, knees, and fingers, as well as hypermobility of the glenohumeral joints) (Fig. 33-30). This problem might be considered voluntary or involuntary, because it occurs whenever the patient raises the arms to the overhead position. Some patients seen for another shoulder problem are completely unaware that with the overhead motion the medial end of the clavicle subluxates or dislocates.

In the review by Rockwood and Odor of 37 patients with spontaneous atraumatic subluxation, 29 were managed without surgery and eight were treated (elsewhere) with surgical reconstruction.[178] With an average follow-up of more than 8 years, all 29 nonoperated patients were doing just fine without limitations of activity or lifestyle. The eight patients treated with surgery had increased pain, limitation of activity, alteration of lifestyle, persistent instability, and significant scars. In many instances, before reconstruction or resection, these patients had minimal discomfort and excellent range of motion and only complained of a

Figure 33-30 This patient has developed spontaneous subluxation of her sternoclavicular joints. She also has generalized ligamentous laxity of the wrists, fingers, and elbows.

"bump" that slipped in and out of place with certain motions. Postoperatively, these patients still had the bump, along with scars and painful range of motion (Fig. 33-31). Twenty-two patients with atraumatic anterior dislocations were treated nonoperatively by Sadr and Sawnn with resolution of their symptoms.[185] They emphasized the importance of reassuring the patient that this is a benign condition and did not advocate surgery.

Only occasionally does the patient with atraumatic anterior displacement complain of pain during the displacement. Because it is difficult to stabilize the joint and prevent the subluxation or dislocation and end up with a pain-free range of motion, we manage the problem with skillful neglect. The anatomy of the problem is explained to the patient and the family. We explain further that surgery is of little benefit, that they should discontinue the voluntary aspect of the dislocation, and that in time either the symptoms will disappear or they will completely forget that the dislocation is a problem.

Posterior

There are only three radiographically documented atraumatic posterior dislocations of the sternoclavicular joint in the literature.[109,138,139] In the 1800s and early 1900s, three others were described but there was no radiographic confirmation. In 1824, Cooper described a posterior dislocation that occurred spontaneously in a patient with severe scoliosis.[39] In 1911, Preiser reported on a young girl with an atraumatic dislocation.[165] In 1903, Katzenstein described a manual laborer with a slow spontaneous posterior dislocation.[106]

More recently Martin et al. described a spontaneous posterior dislocation in a 50-year-old female.[138] The patient presented with pain and dysphagia, and a posterior dislocation was confirmed with a CT scan. A closed reduction was attempted 5 days after presentation. Although a palpable "clunk" was felt, a postreduction CT scan revealed a persistent posterior dislocation. The joint was left dislocated and she was managed nonoperatively with resolution of her symptoms within a year. Martinez et al. described a 19-year-old female with a radiographically confirmed atraumatic posterior dislocation treated successfully with operative stabilization.[139] Finally, Kayias et al. reported on a 30-year-old male with a radiographically confirmed posterior dislocation treated successfully 10 days out with a closed reduction.[109]

Traumatic Injuries of the Sternoclavicular Joint

Traumatic injuries of the sternoclavicular joint are rare. Undoubtedly, anterior dislocations of the sternoclavicular joint are much more common than the posterior type. It is a diagnosis that can be easily overlooked, especially when the dislocation is in the posterior direction.[40,113,210,224,225] However, the ratio of anterior to posterior dislocations is only rarely reported. Theoretically, one could survey the literature and develop the ratio of anterior to posterior dislocations, but most of the published material on sternoclavicular dislocations is on the rare posterior dislocation. Of the references listed at the end of this chapter that address injuries of the sternoclavicular joint, more than 60% discuss only posterior dislocations and their various complications. The largest series from a single institution is reported by Nettles and Linscheid, who studied 60 patients with sternoclavicular dislocations (57 anterior and three posterior).[145] This gives a ratio of anterior dislocations to posterior dislocations of the sternoclavicular joint of approximately 20:1. Waskowitz reviewed 18 cases of sternoclavicular dislocations, none of which was posterior.[217] However, in our series of 185 traumatic sternoclavicular injuries, there have been 135 patients with anterior dislocation and 50 patients with posterior dislocation.

In 1896, Hotchkiss reported a bilateral traumatic dislocation of the sternoclavicular joint.[89] A 28-year-old man was run over by a cart and suffered an anterior dislocation of the right shoulder and a posterior dislocation of the left one. The senior author has treated four cases of bilateral sternoclavicular dislocation.

To our knowledge, the first reported case of dislocation of both ends of the clavicle was mentioned by Porral in

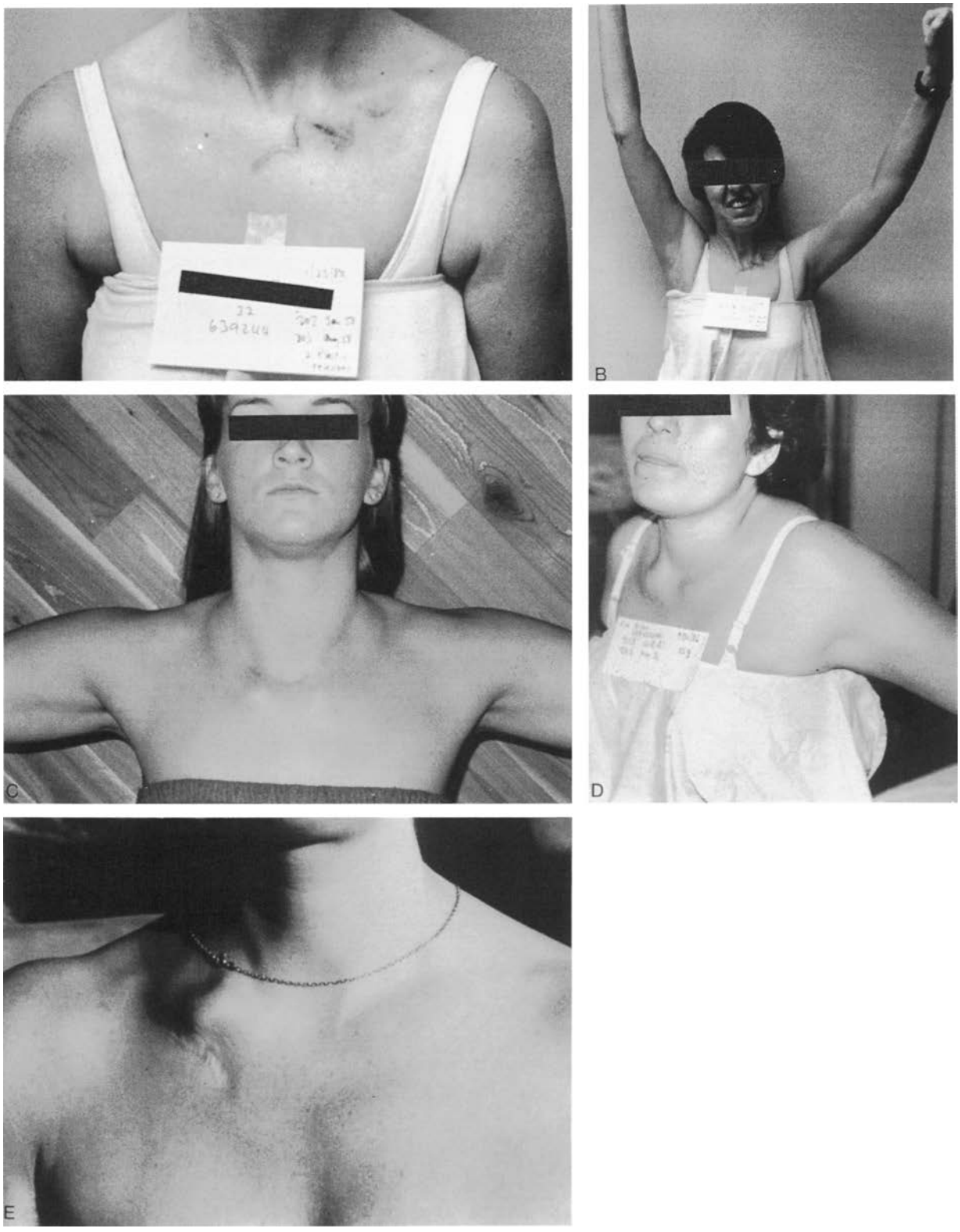


Figure 33-31 Patients treated with surgery for spontaneous, atraumatic subluxation of the sternoclavicular joint had increased pain, limitation of activity, alteration of lifestyle, persistent instability of the joint, and a significant scar. **(A,B)** Not only was the cosmetic scarring a problem, but motion and pain were worse than before the reconstruction. **(C,D)** Despite surgical reconstruction, both patients shown here had persistent subluxation and pain. **(E)** This patient had bilateral spontaneous, atraumatic subluxation of the sternoclavicular joints. Following reconstruction, the right shoulder continued to subluxate, was painful, and significantly altered the patient's lifestyle. The left shoulder had minimal subluxations and was essentially asymptomatic. (Reprinted with permission from Rockwood CA, Matsen F III, eds. *The shoulder.* Philadelphia: WB Saunders, 1990:Fig. 13-30.)

1831.[164] In 1923, Beckman reported a single case and reviewed the literature on 15 cases that had been previously reported.[12] With the exception of this patient, all patients had been treated nonoperatively with acceptable function. In one patient, a brachial plexus neuropathy developed and was treated by excision of a portion of the clavicle. Until recently, to our knowledge, only four additional cases have been reported.[97,167] In 1990, Rockwood and associates reported on six patients who had a dislocation of both ends of the clavicle (an anterior dislocation of the sternoclavicular joint).[179] Two patients who had fewer demands on the shoulder did well with only minor symptoms after nonoperative management. The other four patients had persistent symptoms that were localized to the acromioclavicular joint. Each of these patients had a reconstruction of the acromioclavicular joint that resulted in a painless, full range of motion and a return to normal activity.

Mechanism of Injury

Because the sternoclavicular joint is subject to practically every motion of the upper extremity, and because the joint is so small and incongruous, one would think that it would be the most commonly dislocated joint in the body. However, the ligamentous supporting structure is so strong and so well designed that it is, in fact, one of the least commonly dislocated joints in the body. A traumatic dislocation of the sternoclavicular joint usually occurs only after tremendous forces, either direct or indirect, have been applied to the shoulder.

Direct Force

When a force is applied directly to the anteromedial aspect of the clavicle, the clavicle is pushed posteriorly behind the sternum and into the mediastinum (Fig. 33-32). This may occur in a variety of ways: One athlete jumps on another athlete lying on his back, and the knee of the jumper lands directly on the medial end of the clavicle; a kick is delivered to the front of the medial clavicle; a person is run over by a vehicle; or a person is pinned between a vehicle and a wall (Fig. 33-33). Because of our anatomy, it would be most unusual for a direct force to produce an anterior sternoclavicular dislocation.

Indirect Force

A force can be applied indirectly to the sternoclavicular joint from the anterolateral or posterolateral aspects of the shoulder. This is the most common mechanism of injury to the sternoclavicular joint. Mehta and coworkers reported that three of four posterior sternoclavicular dislocations were produced by indirect force, and Heinig reported that indirect force was responsible for eight of nine cases of

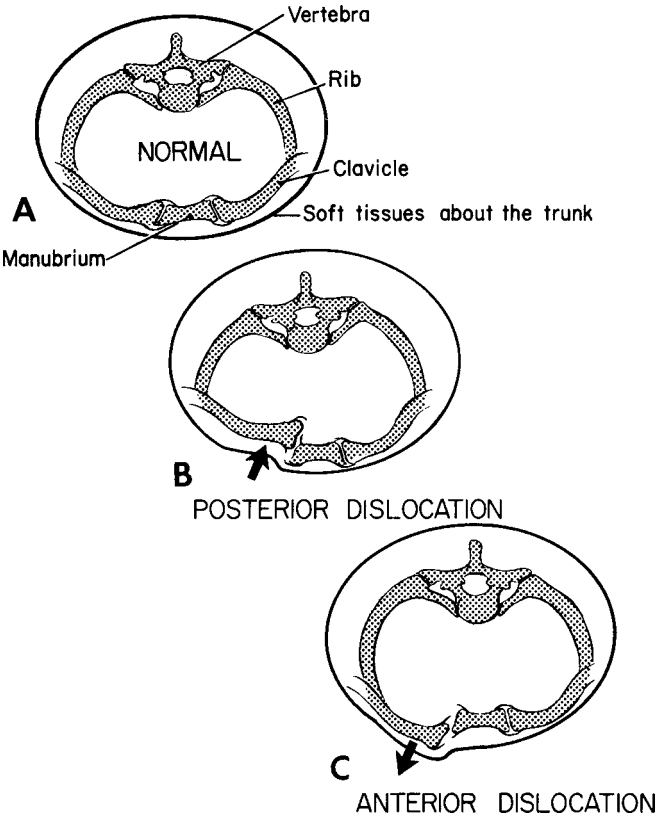


Figure 33-32 Cross-sections through the thorax at the level of the sternoclavicular joint. **(A)** Normal anatomic relationships. **(B)** Posterior dislocation of the sternoclavicular joint. **(C)** Anterior dislocation of the sternoclavicular joint.

posterior sternoclavicular dislocations.[86141] It was the most common mechanism of injury in our series of 185 patients. If the shoulder is compressed and rolled forward, an ipsilateral posterior dislocation results; if the shoulder is compressed and rolled backward, an ipsilateral anterior dislocation results (Fig. 33-34). One of the most common causes we have seen is a pile-on in a football game. In this instance, a player falls on the ground, landing on the lateral shoulder; before he can get out of the way, several players pile on top of his opposite shoulder, which applies significant compressive force on the clavicle down toward the sternum. If, during the compression, the shoulder is rolled forward, the force directed down the clavicle produces a posterior dislocation of the sternoclavicular joint. If the shoulder is compressed and rolled backward, the force directed down the clavicle produces an anterior dislocation of the sternoclavicular joint. Other types of indirect forces that can produce sternoclavicular dislocation are a cave-in on a ditch-digger with lateral compression of the shoulders by the falling dirt; lateral compressive forces on the shoulder when a person is pinned between a vehicle and a wall; and a person's falling on the outstretched abducted arm, which drives the shoulder medially in the same manner as a lateral compression on the shoulder.

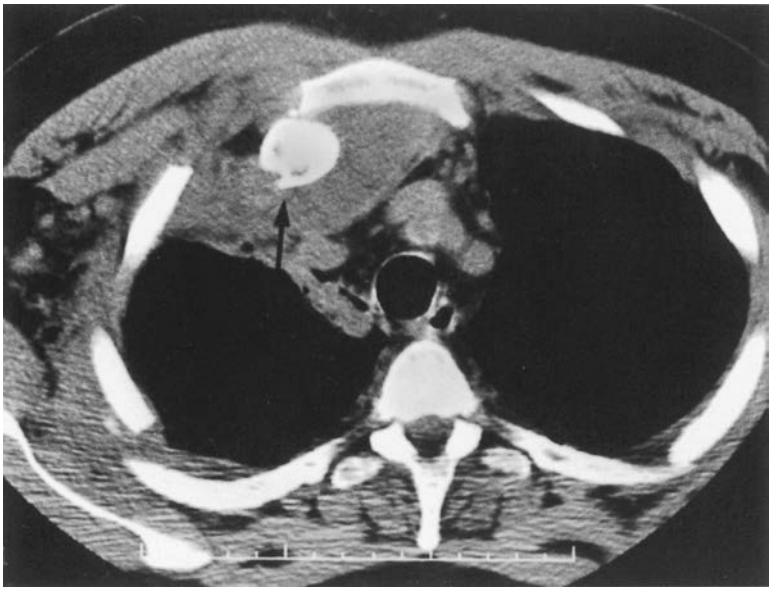


Figure 33-33 Computed axial tomogram of a posterior sternoclavicular joint dislocation that occurred when the driver's chest impacted the steering wheel during a motor vehicle accident. The vehicle was totaled, and the steering wheel was fractured from the driving column. (From Wirth MA, Rockwood CA. Injuries to the sternoclavicular joint. In: Rockwood CA, Green DP, Bucholz RW, Hechman JD, eds. *Fractures in adults*. Philadelphia: JB Lippincott, 1996.)

Most Common Causes of Injury

The most common cause of dislocation of the sternoclavicular joint is vehicular accidents; the second is an injury sustained during participation in sports.[4,137,157,158] Omer reviewed patients from 14 military hospitals and found 82 cases of sternoclavicular dislocations with 80% of these occurring secondary to motor vehicle accidents.

Probably the youngest patient to have a traumatic sternoclavicular dislocation was reported by Wheeler and associates.[220] They described an anterior dislocation in a 7-month-old infant. The injury occurred when she was lying on her left side and her older brother accidentally fell on her, compressing her shoulders together. The closed reduction was unstable, and the child was immobilized in a figure-eight bandage for 5 weeks. At 10 weeks she had full range of motion, and there was no evidence of instability. The senior author has seen an anterior injury in a 3-year-old that occurred as a result of an automobile accident (Fig. 33-35).

Sprains

Acute sprains to the sternoclavicular joint can be classified as mild, moderate, or severe. In a mild sprain, all the ligaments are intact and the joint is stable. In a moderate sprain, there is subluxation of the sternoclavicular joint. The capsular, intraarticular disk, and costoclavicular ligaments may be partially disrupted. The subluxation may be anterior or posterior. In a severe sprain or frank dislocation,

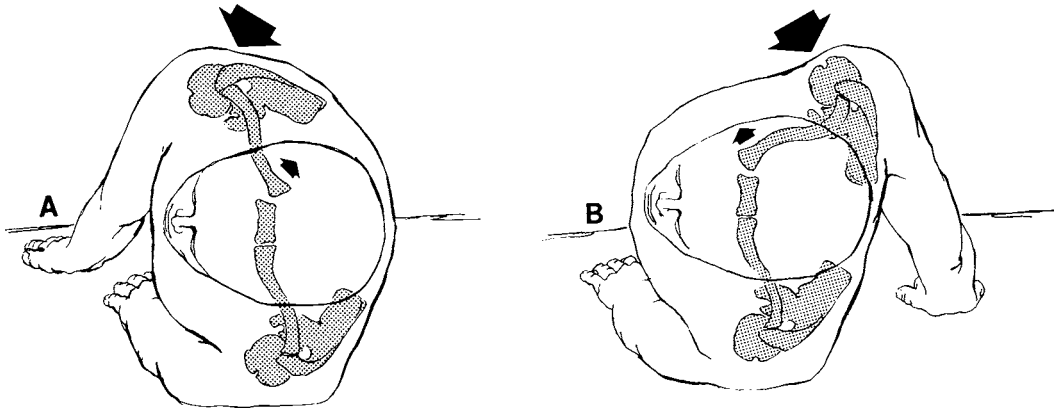


Figure 33-34 Mechanisms that produce anterior or posterior dislocations of the sternoclavicular joint. **(A)** If the patient is lying on the ground and a compression force is applied to the posterolateral aspect of the shoulder, the medial end of the clavicle will be displaced posteriorly. **(B)** When the lateral compression force is directed from the anterior position, the medial end of the clavicle is dislocated anteriorly.

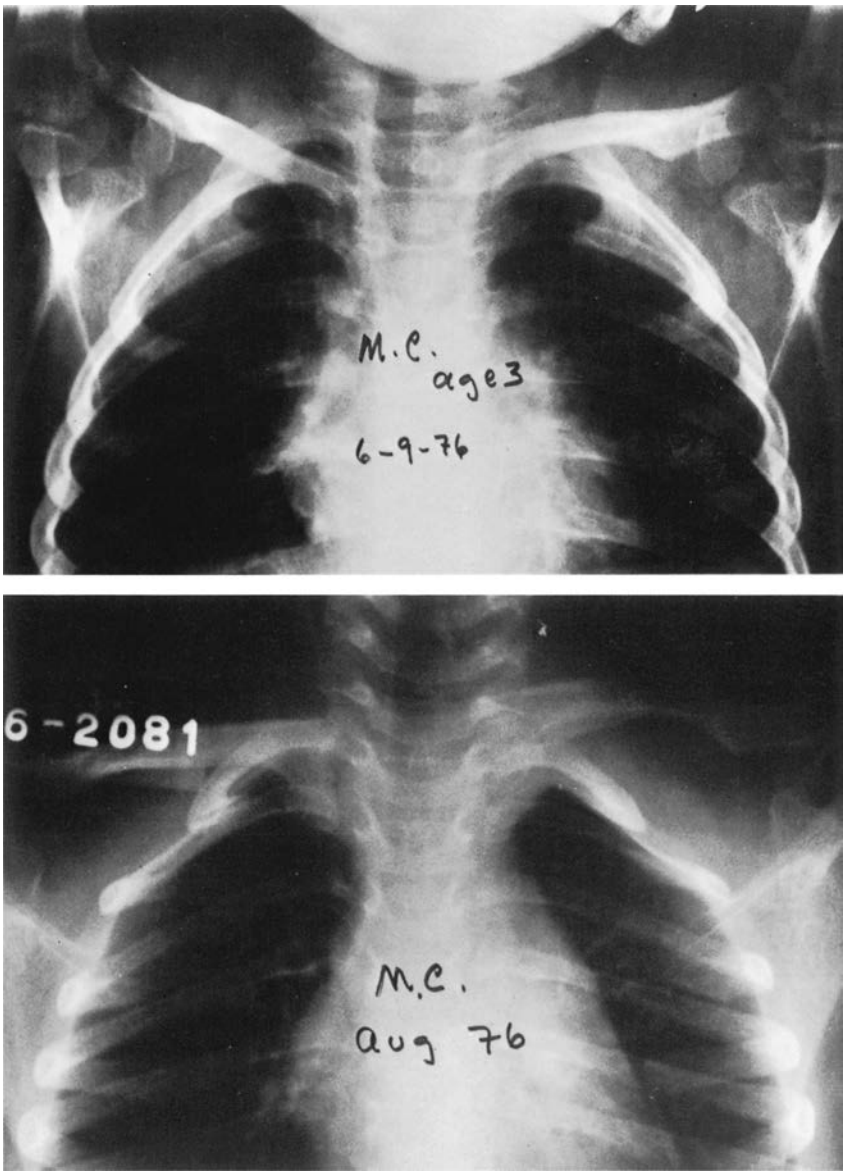


Figure 33-35 X-rays of a 3-year-old child with traumatic anterior dislocation of the left sternoclavicular joint. The chest film demonstrates that the left clavicle is superior to the right, suggesting an anterior displacement of the left medial clavicle.

there is complete disruption of the sternoclavicular ligaments, and the dislocation may be anterior or posterior.

Mild Sprain

In a mild sprain, the ligaments of the joint are intact. The patient complains of a mild to moderate amount of pain, particularly with movement of the upper extremity. The joint may be slightly swollen and tender to palpation, but instability is not noted. The mildly sprained sternoclavicular joint is stable but painful. Application of ice for the first 12 to 24 hours, followed by heat, is helpful. The upper extremity should be immobilized in a sling for 3 to 4 days; then, gradually the patient can regain use of the arm in everyday activities. The senior author undertook a fascinating case of a young woman who, after childbirth, developed aching pain in both sternoclavicular joints. Her bra size, over a period of 4 weeks, had jumped from a 36B to a 38EE. The increase in weight depressed both shoulders and produced pain while upright in both sternoclavicular joints. The discomfort was completely relieved by pushing both elbows up, thus elevating the distal clavicles, which in turn took the strain off her sternoclavicular joints. She was requested to consult her gynecologist and surgeon to determine the quickest way to reduce the size of her breasts.

Moderate Sprain (Subluxation)

A moderate sprain results in a subluxation of the sternoclavicular joint. The ligaments are either partially disrupted or severely stretched. Swelling is noted and pain is marked, particularly with any movement of the arm. Anterior or posterior subluxation may be obvious to the examiner

when the injured joint is compared with the normal sternoclavicular joint. For subluxation of the sternoclavicular joint, application of ice is recommended for the first 12 hours, followed by heat for the next 24 to 48 hours. The joint may be subluxed anteriorly or posteriorly, which may be reduced by drawing the shoulders backward as if reducing and holding a fracture of the clavicle. A clavicle strap can be used to hold up the shoulder and to prevent motion of the arm. The patient should be protected from further injury for 4 to 6 weeks. De Palma[46] has suggested a plaster figure-eight dressing, and McLaughlin[140] has recommended the same type of treatment that would be used for fracture of the clavicle, with the addition of a sling to support the arm. We often use a soft, padded figure-eight clavicle strap to gently hold the shoulders back to allow the sternoclavicular joint to rest. The harness can be removed after a week or so; then the arm is placed in a sling for about another week, or the patient is allowed to return gradually to everyday activities.

Dislocations

The patient with a sternoclavicular dislocation has severe pain that is increased with any movement of the arm, particularly when the shoulders are pressed together by a lateral force. The patient usually supports the injured arm across the trunk with the normal arm. The affected shoulder appears to be shortened and thrust forward when compared with the normal shoulder. The head may be tilted toward the side of the dislocated joint. The discomfort increases when the patient is placed in the supine position, at which time it will be noted that the involved shoulder will not lie back flat on the table.

Anterior Dislocation

With an anterior dislocation, the medial end of the clavicle is visibly prominent anterior to the sternum (Fig. 33-36) and can be palpated anterior to the sternum. It may be fixed anteriorly or may be quite mobile.

There still is some controversy regarding the treatment of acute or chronic anterior dislocation of the sternoclavicular joint. In 1990, de Jong and Sukul reported long-term follow-up results in 10 patients with traumatic anterior sternoclavicular dislocations.[43] All patients were treated nonoperatively with analgesics and immobilization. The results of treatment were good in seven patients, fair in two patients, and poor in one patient at an average follow-up of 5 years. Nettels and Linscheid reported on 14 patients with an anterior dislocation who were treated with a closed reduction.[145] Eleven had no recurrence or pain. Other reports also support nonoperative treatment for anterior dislocations.[189] Some have advocated various forms of operative stabilization for those patients that remain symptomatic.[19,62]

Method of Closed Reduction. Closed reduction of the sternoclavicular joint may be accomplished with local or general anesthesia or, in stoic patients, without anesthesia. Most investigators recommend the use of narcotics or muscle relaxants. The patient is placed supine on the table, lying on a 3- to 4-in. thick pad between the shoulders. In this position, the clavicle may reduce with direct gentle pressure over the anteriorly displaced clavicle. However, when the pressure is released, the clavicle might redislocate. Sometimes, the physician will need to push both shoulders back onto the table while an assistant applies pressure to the anteriorly displaced clavicle. Laidlaw treated an interesting case of a patient who had a dislocated clavicle. The sternoclavicular joint was dislocated anteriorly and was mildly symptomatic.[121] The acromioclavicular joint was most symptomatic and was treated by excision of the distal clavicle. Surprisingly, the anteriorly dislocated sternoclavicular joint reduced and became pain free. Although some if not many patients will redislocate anteriorly, we still believe an attempt at a closed reduction is warranted.

Postreduction Care. If, with the shoulders held back, the sternoclavicular joint remains reduced, the shoulders can be stabilized with a soft figure-eight dressing, a commercial clavicle strap harness, or a plaster figure-eight cast. Some investigators recommend a bulky pressure pad over the anteromedial clavicle that is held in place with elastic

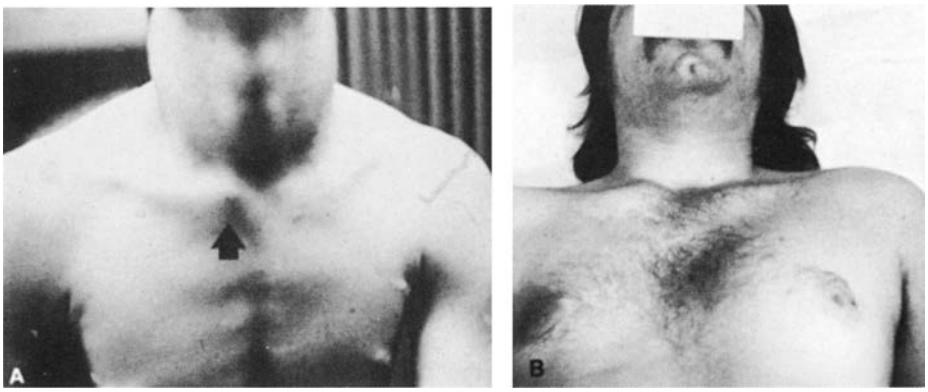


Figure 33-36 **(A)** An anterior dislocation of the right sternoclavicular joint is clinically evident *(arrow)*. **(B)** When the clavicles are viewed from around the level of the patient's knees, it is apparent that the right clavicle is dislocated anteriorly.

tape. A sling might be used, because it holds up the shoulder and prevents motion of the arm. Immobilization should be maintained for at least 6 weeks, and then the arm should be protected for another 2 weeks before strenuous activities are undertaken. If the sternoclavicular joint redislocates when the reduction pressure is released, a figure-eight dressing or a sling can be used until the patient's symptoms subside.

Although some investigators have recommended operative repair of anterior dislocations of the sternoclavicular joint, we believe that operative stabilization should be used with great caution as most patients will do well with nonoperative treatment even if the joint remains dislocated. The technique of operative stabilization for anterior dislocations is the same as those for posterior dislocations and is described later in that section. In most cases of an anterior dislocation the risks of surgery outweigh the potential benefits. Certainly in children, in whom many if not most of the injuries are physeal fractures, a nonoperative approach should be strongly considered.

Physeal Injuries

As described earlier in this chapter, the epiphysis on the medial end of the clavicle is the last epiphysis in the body to appear on x-ray and the last one to close. The epiphysis on the medial end of the clavicle does not appear on x-rays until about the 18th year of life and does not unite with the clavicle until the 23rd to 25th year.

This is important to remember, because many so-called dislocations of the sternoclavicular joint are not dislocations but physeal injuries.[22,45,68,93,124,128,162,192,226,229] Most of the anterior injuries that we have treated in patients up to 25 years of age are not dislocations of the sternoclavicular joint but type I or II physeal injuries, which heal and remodel without operative treatment. In time, the remodeling process eliminates any bone deformity or displacement.

Even though most of the posterior dislocations in this age group also represent physeal injuries, a reduction should be attempted. This is to prevent the potential complications of an unreduced posterior dislocation such as compression of the posterior neurovascular structures, trachea, and esophagus. If the posterior dislocation cannot be reduced closed and the patient is having no significant symptoms, the displacement can be observed while remodeling occurs.

Zaslav and associates have reported successful treatment of a posteriorly displaced medial clavicle physeal injury in an adolescent athlete with CT documentation of remodeling, most probable within an intact periosteal tube.[229] Because of its high osteogenic potential, spontaneous healing and remodeling to the preinjury "reduced" position can occur within this periosteal conduit. Similarly, Hsu and associates reported successful treatment of a posterior epiphyseal fracture dislocation of the medial end of the clavicle in a 15-year-old patient.[93]

If the posterior displacement is symptomatic and cannot be reduced closed, the displacement must be reduced during surgery (Fig. 33-37). At the time of surgery, the unossified or ossified epiphyseal disk, depending on the age of the patient, stays with the sternum. Anatomically, the epiphysis is lateral to the articular disk ligament, and it is held in place by the capsular ligament and can be mistaken for the intraarticular disk ligament.

For posteriorly displaced clavicles, the periosteal sleeve is usually still intact anteriorly. A Darrach retractor can be placed in the physeal fracture site and can be used as a "shoe horn" to reduce the clavicle back into the physis. After reduction the clavicle and physis are usually inherently stable and the periosteum is simply closed. After

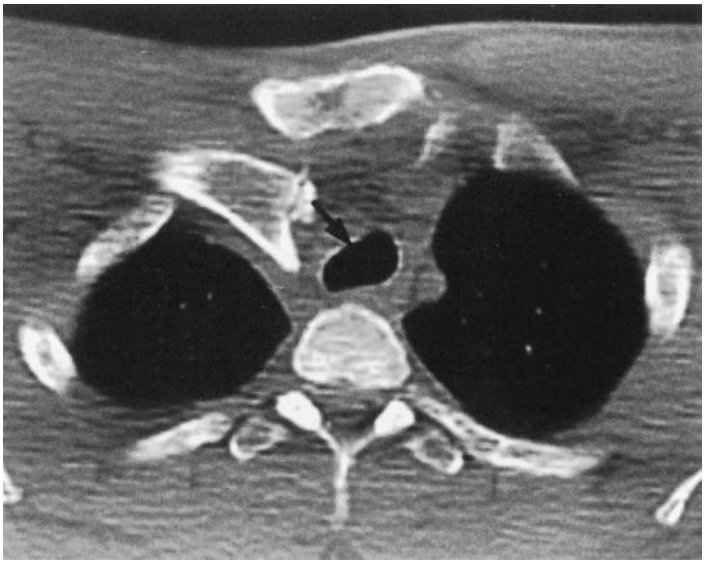


Figure 33-37 Computed tomography scan of a 19-year-old patient who was involved in a motor vehicle accident and presented with complaints of chest pain and a "choking" sensation that was exacerbated by lying supine. Note the physeal injury of the medial clavicle and compression of the trachea (*arrow*). (From Wirth MA, Rockwood CA. Injuries to the sternoclavicular joint. In: Rockwood CA, Green DP, Bucholz RW, Hechman JD, eds. *Fractures in adults.* Philadelphia: JB Lippincott, 1996.)

reduction, the shoulders are held back with a figure-eight strap or dressing for 3 to 4 weeks. Gentle active assisted range-of-motion exercises are initiated with full active motion started at 6 weeks. It is important to remember that open reduction of the physeal injury is seldom indicated, except for an irreducible posterior displacement in a patient with significant symptoms of compression of the vital structures in the mediastinum.

Posterior Dislocation

The patient with a posterior dislocation has more pain than does a patient with an anterior dislocation. The anterosuperior fullness of the chest produced by the clavicle is less prominent and visible when compared with the normal side. The usually palpable medial end of the clavicle is displaced posteriorly. The corner of the sternum is easily palpated as compared with the normal sternoclavicular joint. Venous congestion may be present in the neck or in the upper extremity. Breathing difficulties, shortness of breath, or a choking sensation may be noted.[133] Circulation to the ipsilateral arm may be decreased. The patient may complain of difficulty in swallowing or a tight feeling in the throat, or may be in a state of complete shock or possibly have a pneumothorax.

We have seen a number of patients who clinically appeared to have an anterior dislocation of the sternoclavicular joint but on x-ray studies were shown to have complete posterior dislocation. The point is that one cannot always rely on the clinical findings of observing and palpating the joint to make a distinction between anterior and posterior dislocations. This emphasizes the need for more detailed radiographic studies such as a CT scan. It has been noted by several authors that this is a diagnosis that can be easily missed, and there should be a heightened clinical suspicion for any patient with the above clinical symptoms.[40,48,113,210,224,225] Multiple authors have also described devastating complications with posterior dislocations. Venous compression of the brachiocephalic and subclavian veins has been reported.[56,58,154,200] Arterial compression has also been described.[9074147] Wasylenko and Busse reported a case of a tracheoesophageal fistula that occurred secondary to a chronic posterior dislocation.[218] Other authors have described thoracic outlet syndrome in conjunction with posterior dislocations.[73,98]

A careful examination of the patient is extremely important. Clearly complications are common with posterior dislocation of the sternoclavicular joint, and the patient should receive prompt attention. A careful history and physical examination should be undertaken to rule out damage to the pulmonary and vascular systems. The sternoclavicular joint must be carefully evaluated by all available x-ray techniques and computed tomography, including, when indicated, combined aortogram–CT scan for potential vascular injuries (Figs. 33-38 and 33-39). If specific

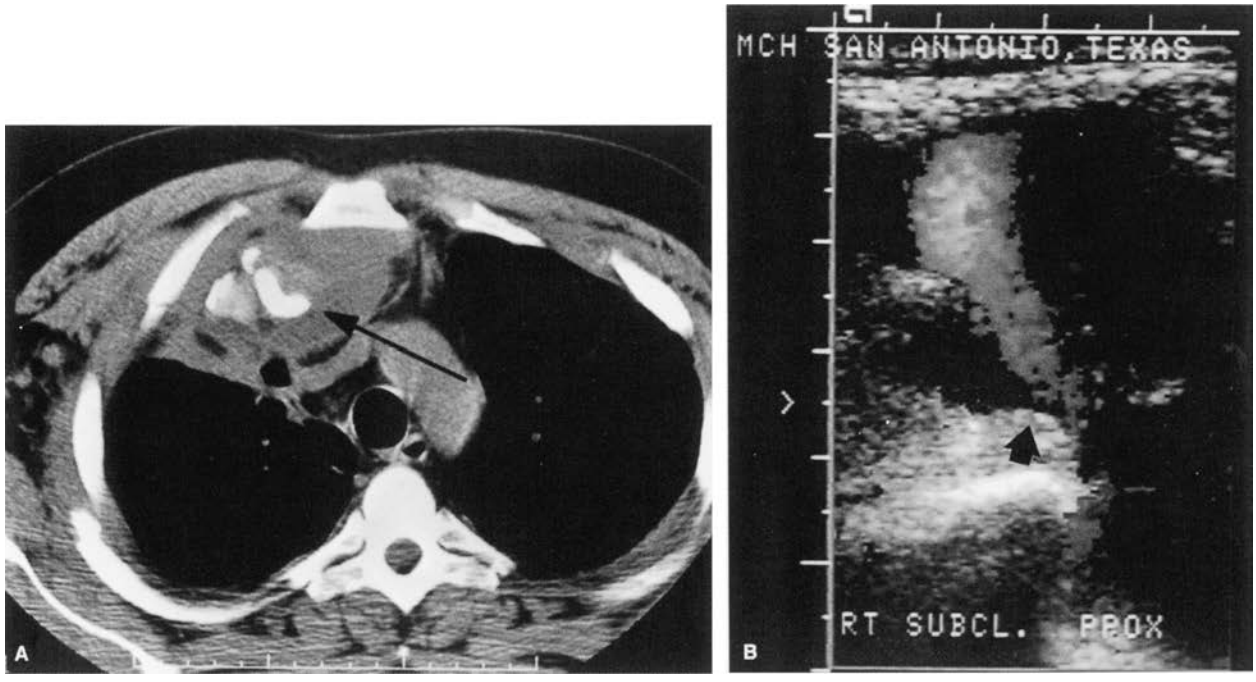


Figure 33-38 **(A)** Computed tomography scan revealing a posterior fracture–dislocation of the sternoclavicular joint with significant soft tissue swelling and compromise of the hilar structures. **(B)** Duplex ultrasound study revealing a large pseudoaneurysm of the right subclavian artery. Note the large neck of the pseudoaneurysm, which measured approximately 1 cm in diameter (*arrow*). (From Wirth MA, Rockwood CA. Injuries to the sternoclavicular joint. In: Rockwood CA, Green DP, Bucholz RW, Hechman JD, eds. *Fractures in adults.* Philadelphia: JB Lippincott, 1996.)

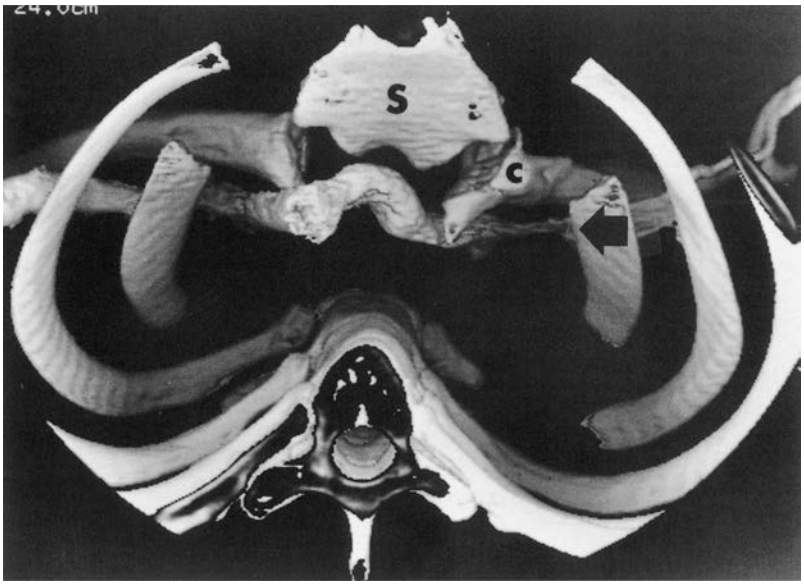


Figure 33-39 Contrast material–enhanced computed tomography scan of 16-year-old boy who presented with left arm swelling and cyanosis 14 months after a wrestling injury. A stenotic lesion of the subclavian vein can be seen adjacent to a posteriorly displaced physeal fracture of the medial clavicle (*arrow*). Note the relationship between the posteriorly displaced clavicle (*C*) and sternum (*S*). (Reprinted with permission from Wirth MA, Rockwood CA. Acute and chronic traumatic injuries of the sternoclavicular joint. *J Am Acad Orthop Surg* 1996;4:268–278.)

complications are noted, appropriate consultants should be called in before reduction is performed. Worman and Leagus reported a posterior dislocation of the sternoclavicular joint, in which it was noted at surgery that the displaced clavicle had put a hole into the right pulmonary artery.[223] The clavicle had prevented exsanguination, because the vessel was still impaled by the clavicle. Had a closed reduction been performed in the emergency department, the result could have been disastrous. Cooper and coworkers reported a posterior sternoclavicular dislocation that transected the internal mammary artery and lacerated the brachiocephalic vein.[38] The vascular injuries were associated with fractures of the anterior ends of the first to third ribs and marked posterior instability of the medial end of the clavicle. The brachiocephalic vein was repaired, but the posteriorly displaced medical clavicle impinged on the suture line. To maintain reduction of the sternoclavicular joint, a novel method of stabilization was employed using an external fixator.

From a review of the earlier literature, it would appear that the treatment of choice for posterior sternoclavicular dislocation was by operative procedures. However, since the 1950s, the treatment of choice has been closed reduction.[25,63,86,228] Some investigators, who had previously performed open reductions, reported that they were amazed at how easily the dislocation reduced under direct vision, and thereafter they used closed reductions with complete success. Many different techniques have been described for closed reduction of a posterior dislocation of the sternoclavicular joint. General anesthesia is usually required for reduction of a posterior dislocation of the sternoclavicular joint, because the patient has so much pain and has muscle spasms. However, for the stoic patient, some investigators have performed the reduction under intravenous narcotics and muscle relaxants. Heinig has successfully used local anesthesia in a posterior dislocation reduction.[86]

Abduction Traction Technique. For the abduction traction technique, the patient is placed on his or her back, with the dislocated shoulder near the edge of the table. A 3- to 4-in.–thick sandbag is placed between the shoulders (Fig. 33-40). Lateral traction is applied to the abducted arm, which is then gradually brought back into extension. This may be all that is necessary to accomplish the reduction. The clavicle usually reduces with an audible snap or pop, and it is almost always stable. Too much extension can bind the anterior surface of the dislocated medial clavicle on the back of the manubrium. Occasionally, it may be necessary to grasp the medial clavicle with one's fingers to dislodge it from behind the sternum. If this fails, the skin is prepared, and a sterile towel clip is used to grasp the medial clavicle to apply lateral and anterior traction (Fig. 33-41).[63,187,228] This has been the most effective technique in our experience.

Adduction Traction Technique. In this technique, the patient is supine on the table with a 3- to 4-in. bolster between the shoulders. Traction is then applied to the arm in adduction, while a downward pressure is exerted on the shoulders. The clavicle is levered over the first rib into its normal position. Buckerfield and Castle reported that this technique has succeeded when the abduction traction technique has failed.[25]

Other Techniques. Heinig and Elting have reported that they accomplished reduction by placing the patient supine

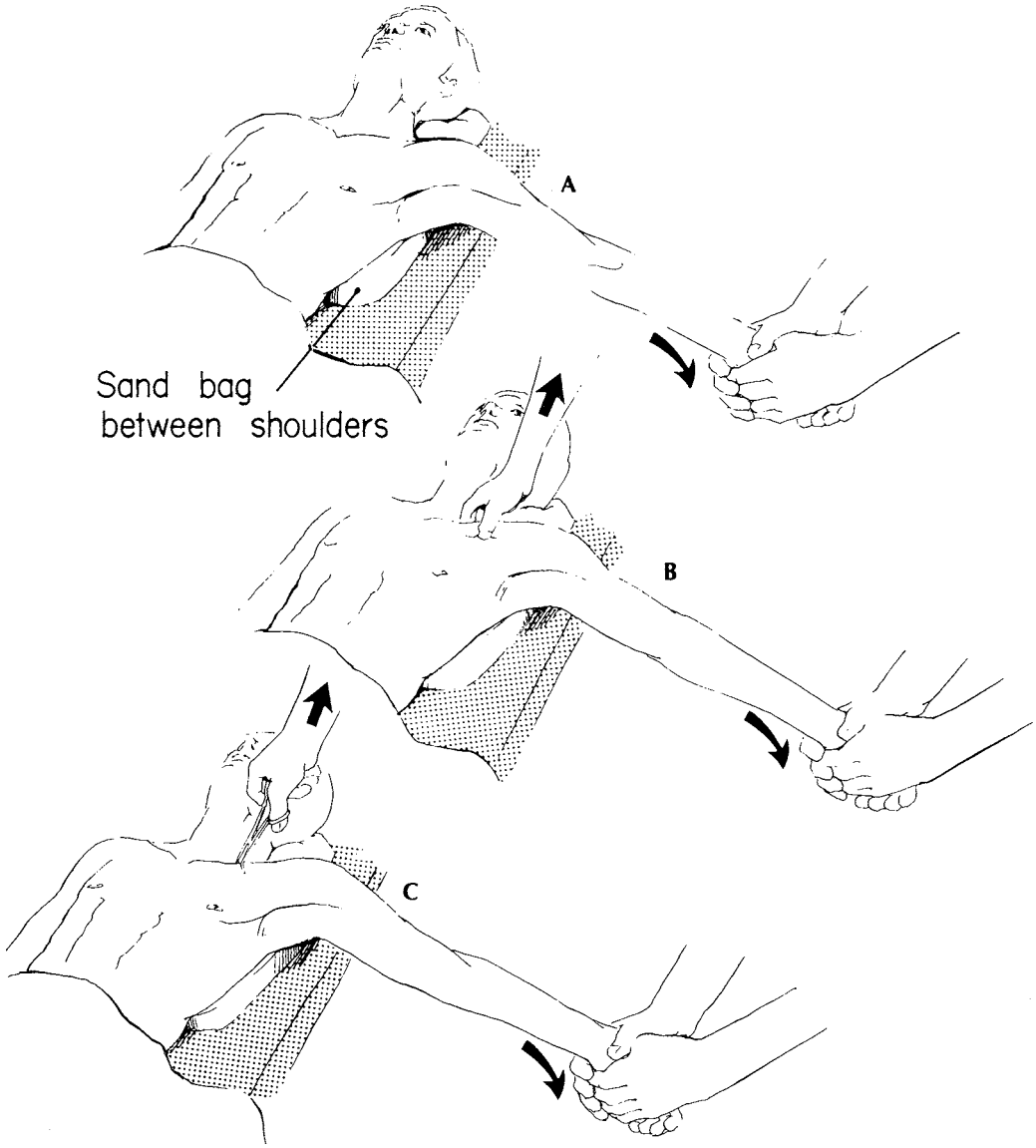


Figure 33-40 Technique for closed reduction of the sternoclavicular joint. **(A)** The patient is positioned supine with a sandbag placed between the two shoulders. Traction is then applied to the arm against countertraction in an abducted and slightly extended position. In anterior dislocations, direct pressure over the medial end of the clavicle may reduce the joint. **(B)** In posterior dislocations, in addition to the traction, it may be necessary to manipulate the medial end of the clavicle with the fingers to dislodge the clavicle from behind the manubrium. **(C)** In stubborn posterior dislocations, it may be necessary to sterilely prepare the medial end of the clavicle and use a towel clip to grasp around the medial clavicle to lift it back into position.

on the table with three of four folded towels between the two shoulders.[57,86] Forward pressure was then applied on both shoulders, which accomplished the reduction. Other investigators have put a knee between the shoulders of the seated patient and, by pulling back on both shoulders, have accomplished a reduction. Stein used skin traction on the abducted and extended arm to accomplish the reduction gently and gradually.[204] Many investigators have reported that closed reduction usually cannot be accomplished after 48 hours. However, others have reported closed reductions as late as 4 and 5 days after the injury.[25]

Postreduction Care. Unlike anterior dislocations, most posterior dislocations are stable after reduction. After reduction, to allow ligament healing, the shoulders should be held back for 4 to 6 weeks with a figure-eight dressing or one of the commercially available figure-eight straps used to treat fractures of the clavicle. Active assisted range-of-motion

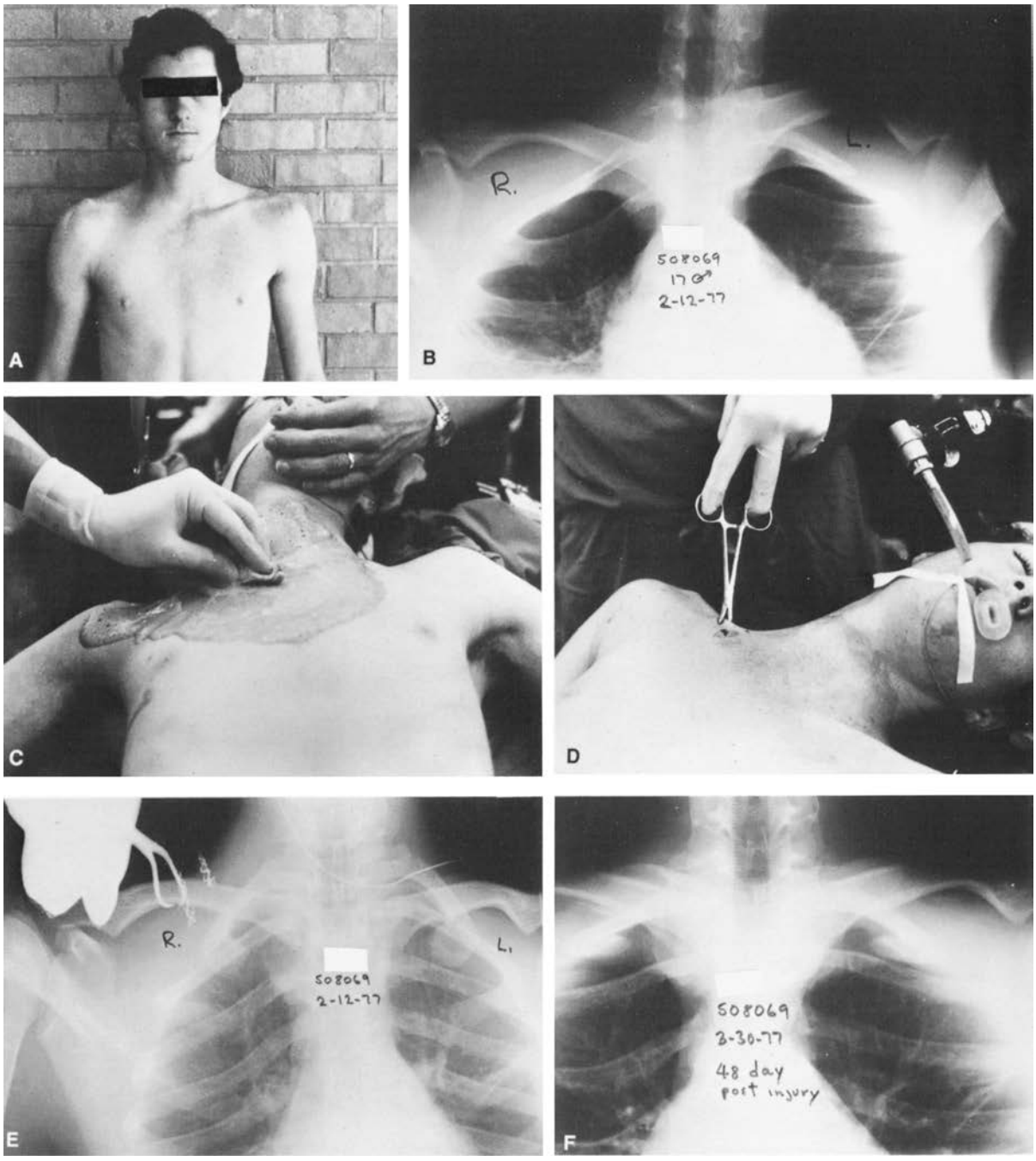


Figure 33-41 Posterior dislocation of the right sternoclavicular joint. **(A)** A 16-year-old boy has a 48-hour-old posterior displacement of the right medial clavicle that occurred from direct trauma to the anterior right clavicle. He noted immediate onset of difficulty in swallowing and some hoarseness in his voice. **(B)** A 40-degree cephalic tilt x-ray film confirmed the posterior displacement of the right medial clavicle, as compared with the left clavicle. Because of the patient's age, this was considered most likely to be a physeal injury of the right medial clavicle. **(C)** Because the injury was 48 hours old, we were unable to reduce the dislocation with simple traction on the arm. The right shoulder was surgically cleansed, so that a sterile towel clip could be used. **(D)** With the towel clip securely around the clavicle and with continued lateral traction, a visible and audible reduction occurred. **(E)** Postreduction x-rays showed that the medial clavicle had been restored to its normal position. The reduction was quite stable, and the patient's shoulders were held back with a figure-eight strap. **(F)** The right clavicle has remained reduced. Note the periosteal new bone formation along the superior and inferior borders of the right clavicle. This is the result of a physeal injury, whereby the epiphysis remains adjacent to the manubrium while the clavicle is displaced out of a split in the periosteal tube.

exercises are initiated at 4 weeks and full active motion by 8 weeks. Full return to sport and activities should be anticipated by 12 weeks.

Recurrent or Unreduced Dislocation

Should closed maneuvers fail in the adult, an operative procedure should be performed, because most adult patients cannot tolerate posterior displacement of the clavicle into the mediastinum. Fig. 33-42 outlines a treatment algorithm for anterior and posterior dislocations. Multiple authors have reported complications following unreduced posterior dislocations.[56,74,102,147,154,200] Gangahar and Flogaites reported a case of late thoracic outlet syndrome following an unreduced posterior dislocation, and Howard and Shafer reported late and significant vascular problems.[73,90] Tircoire and colleagues reported a case of respiratory compromise and dyspnea on exertion in a 65-year-old patient with a posterior sternoclavicular dislocation of 3 months' duration.[212] Rayan reported a thoracic outlet syndrome and brachial plexopathy caused by a chronic posterior sternoclavicular dislocation of 4 years' duration.[170] The injury was presumably missed, due to an associated dislocation of the glenohumeral joint. The patient experienced paresthesias and decreased sensation, and at one time she unknowingly burned her medial forearm with a hot iron. One year following medial clavicle excision and stabilization of the remaining clavicle to the first rib, she was asymptomatic except for mild discomfort related to the glenohumeral joint.

Several of our patients have had unusual complications from traumatic injuries to the sternoclavicular joint. One patient, as the result of a posterior dislocation and rupture of the trachea, developed massive subcutaneous emphysema (Fig. 33-43). Another patient had an anterior dislocation on the right and a posterior dislocation on the left. When first seen, his blood pressure was very low. Following reduction of the posterior dislocation, his blood pressure, as recorded on his monitor, instantly returned to normal (Fig. 33-44). It was theorized that the posteriorly displaced clavicle was irritating some of the vital structures of the mediastinum. The senior author was asked to evaluate a patient who, following a significant injury, complained of swelling and bluish coloration of his left arm after any type of physical activity. He did not have many local sternoclavicular joint symptoms, but by physical examination the left clavicle was displaced posteriorly. CT demonstrated a major posterior displacement of the left clavicle (Fig. 33-45). Because of the marked displacement and the vascular compromise, arteriography combined with CT was performed, which did not reveal any vascular leak. With the help of the chest surgeon, the clavicle was removed from the mediastinum, the medial 1.5 in. were removed, and the shaft was stabilized to the first rib. The greatest displacement we have seen was in a patient with a posteroinferior dislocation of the medial clavicle down into an intrathoracic position.

Worman and Leagus, in an excellent review of the complications associated with posterior dislocations of the sternoclavicular joint, reported that 16 of 60 patients had suffered complications of the trachea, esophagus, or great vessels.[223] We should point out that even though the incidence of complications was 25%, only four deaths have been reported as a result of this injury. Clearly, significant complications can occur with acute and chronic posterior dislocations of the clavicle; therefore, every attempt should be made to reduce a posterior dislocation in the skeletally mature patient.

Surgical Management. Occasionally, following conservative treatment of a subluxation of the sternoclavicular joint, the pain lingers and the symptoms of popping and grating persist. This may require joint exploration. Bateman has commented on the possibility of finding a tear of the intraarticular disk, which should be excised.[10] Duggan reported a case in which, several weeks after an injury to the sternoclavicular joint, the patient still had popping in the joint.[51] Through a small incision, Duggan exposed the capsule and out through the capsule popped the intraarticular disk, which looked like "an avulsed fingernail." Following repair of the capsule, the patient had no more symptoms. The operative procedure should be performed in a manner that disturbs as few of the anterior ligament structures as possible.

For posterior dislocations that fail closed reduction, open reduction is indicated. This should be performed with a thoracic surgeon in the room or on standby notification. The patient lies supine on the table, and the three to four towels or sandbag should be left between the scapulas. The thorax from the superior extent of the neck to the xiphoid process should be prepped on both sides of the sternum. This allows for sterile exposure of the major vessels of the neck and sternotomy to be performed. The upper extremity should be draped free, so that lateral traction can be applied during the open reduction. An anterior incision is used that parallels the superior border of the medial 3 to 4 in. of the clavicle and then extends downward over the sternum just medial to the involved sternoclavicular joint (Fig. 33-46). The trick is to remove sufficient soft tissues to expose the joint but to leave the anterior capsular ligament intact. The reduction usually can be accomplished with traction and countertraction while lifting up anteriorly with a clamp around the medial clavicle. Along with traction and countertraction, it may be necessary to use an elevator to pry the clavicle back to its articulation with the sternum. When the reduction has been obtained, and with the shoulders held back, the reduction and stability should be assessed. If the joint is stable, then it can be managed postoperatively like a closed reduction. If the clavicle is unstable, we recommend reconstruction. The various reconstruction techniques are described below.

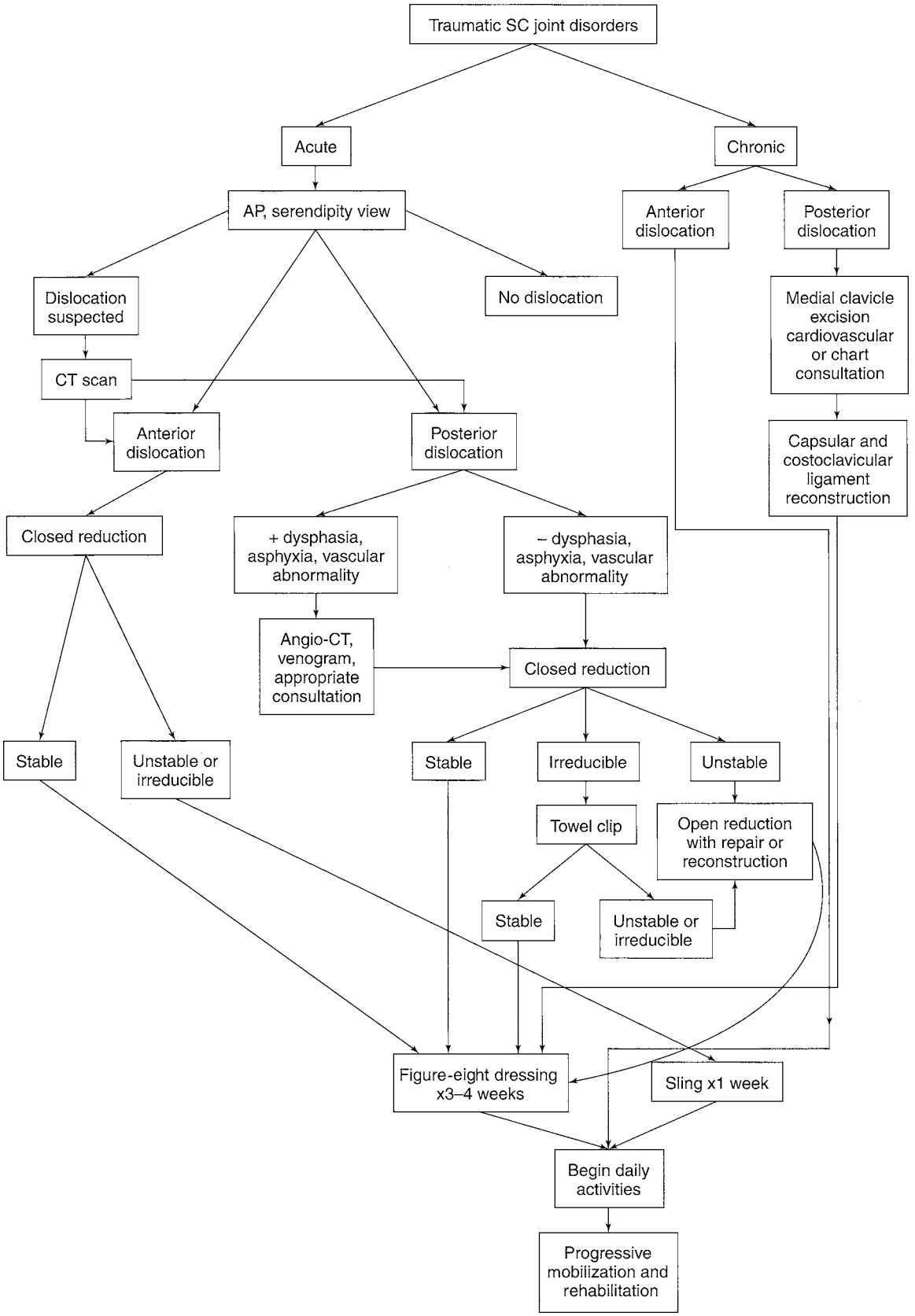


Figure 33-42 Treatment algorithm for traumatic sternoclavicular joint disorders. AP = anteroposterior; CT = computed tomography; SC = sternoclavicular.

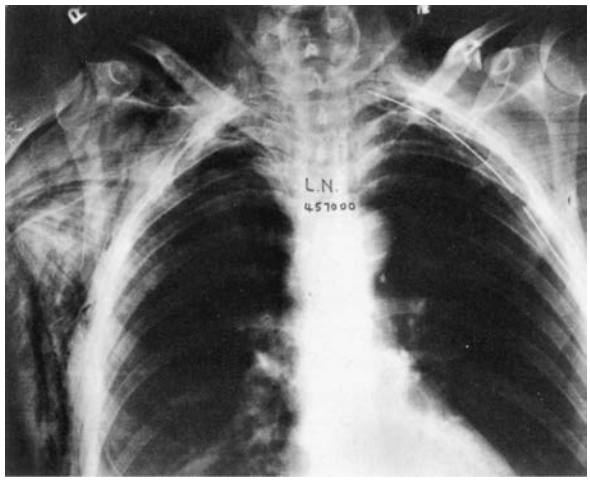


Figure 33-43 Complications of sternoclavicular dislocation. As a result of posterior dislocation of the sternoclavicular joint, the patient had a lacerated trachea and developed massive subcutaneous emphysema.

If degenerative changes become severe in the sternoclavicular joint, excision of the medial end of the clavicle may be required. This is done in a fashion as described for arthritis of the sternoclavicular joint. Again, it should be emphasized that this is analogous to a resection of the lateral end of the clavicle. If there is residual instability with arthritic changes, not only should the medial end of the clavicle be resected, but the joint should also be stabilized as well. Simple resection of the medial end of the clavicle when there is residual instability can result in continued symptoms.

Some of the literature from the 1960s and 1970s recommended stabilization of the sternoclavicular joint with pins. Elting used Kirschner wires to stabilize the joint and supplemented ligament repairs with a short-toe extensor tendon.[57] Denham and Dingley and Brooks and Henning used Kirschner wires.[4522] DePalma recommended repair of the ligaments and stabilized the sternoclavicular joint with one or two Steinmann pins.[46]

Habernek and Hertz; Nutz; Pfister and Weller; Kennedy and Mawhinney; Tagliabue and Riva; Bankart; and Stein avoided the use of pins across the sternoclavicular joint and used loops of various types of suture wires across the joint.[20,46,83,110,151,159,207] Burri and Neugebauer recommended the use of a figure-eight loop of carbon fiber.[27] Maguire; Booth and Roper; Barth and Hagen; and Lunseth and associates reconstructed the sternoclavicular joint using local tendons of the sternocleidomastoid, subclavius, or pectoralis major tendons for repair.[9,19,134,136] Franck et al. reported on the use of a special plate to stabilize the joint.[67] The complications of fixation of the sternoclavicular joint with Kirschner wires or Steinmann pins are horrendous and are discussed in the section on complications.

In 1982, Pfister and Weller recommended open reduction and repair of the ligaments over nonoperative treatment.[159] In symptomatic chronic dislocation or nontraumatic cases, they recommended the use of autogenous grafts between the sternum and the first rib without placing Kirschner wires across the sternoclavicular joint. In 1988, Fery and Sommelet reported 49 cases of dislocations of the sternoclavicular joint.[65] These patients, with an average follow-up of more than 6 years, had 42% excellent results among the operative cases. Of those patients who were treated with closed reduction, 58% were satisfied. Ferrandez and colleagues reported 18 subluxations and dislocations of the sternoclavicular joint.[62] Seven had moderate sprains and 11 had dislocations. Of the three patients with posterior dislocation, all had symptoms of dysphagia. All of the subluxations were treated nonoperatively with excellent results. The remaining 10 patients with dislocations were treated with surgery (i.e., open reduction with suture of the ligaments and Kirschner wires between the clavicle and the sternum). The wires were removed 3 to 4 weeks

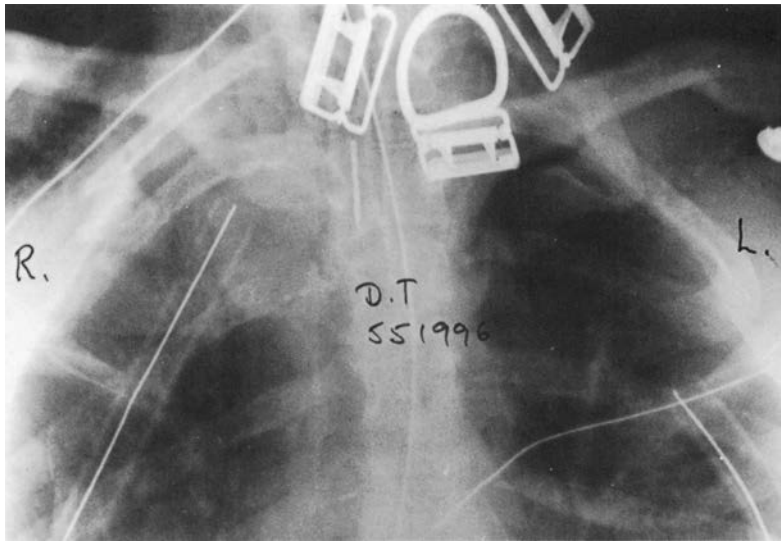


Figure 33-44 Complications of sternoclavicular joint dislocation. This patient had an anterior dislocation on the right and a posterior dislocation on the left. As a result of the posterior dislocation, he had sufficient pressure on the mediastinal structures to cause significant hypotension. When the posterior dislocation was reduced, the blood pressure on the continuous monitor promptly returned to normal.

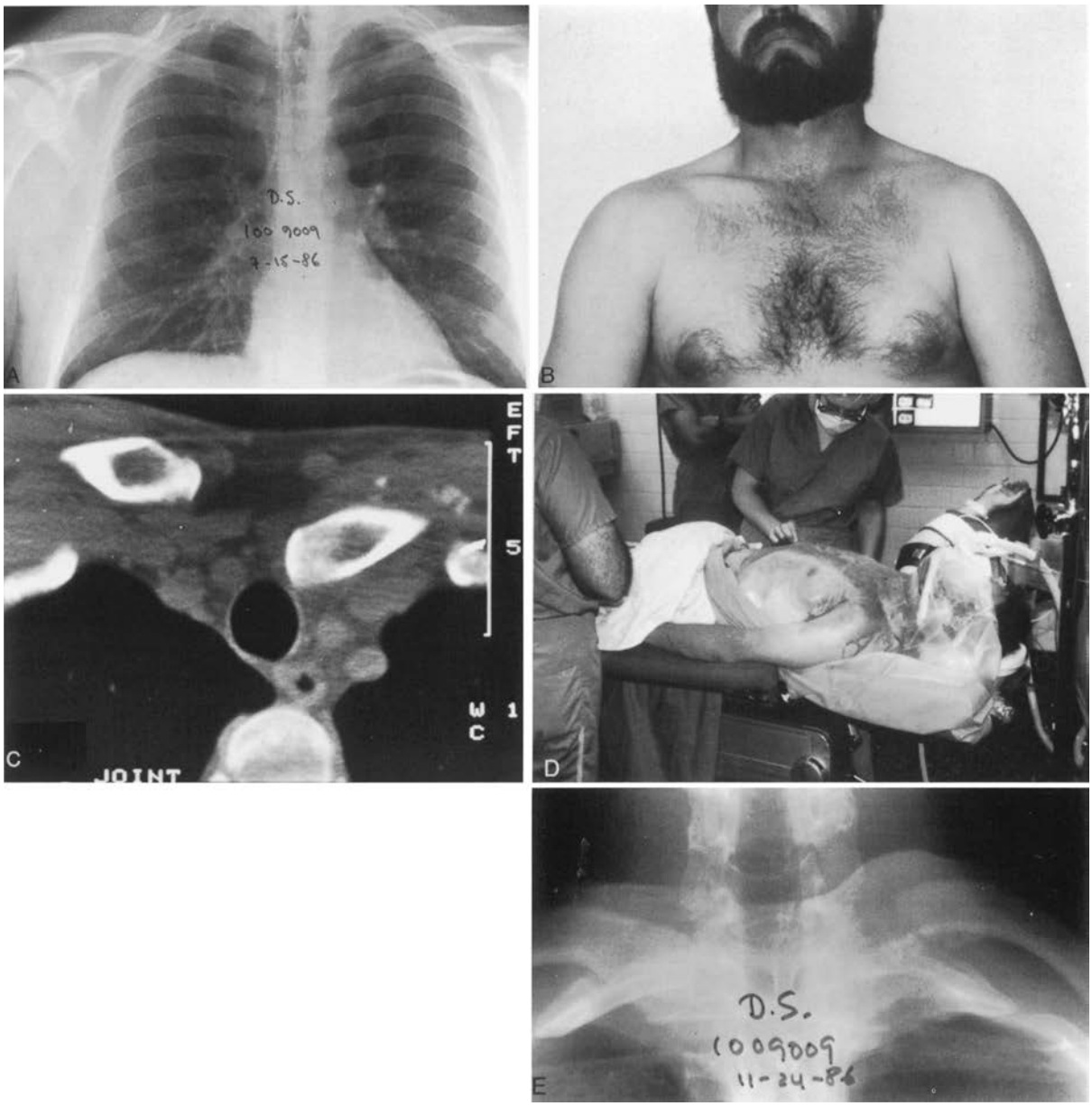


Figure 33-45 Open reduction of a posterior dislocation of the left sternoclavicular joint causing compression of the great vessels in the mediastinum and resultant swelling in the patient's left arm. **(A)** Chest film does not suggest any serious problem with the left medial clavicle. **(B)** Clinically, the medial end of the left clavicle was depressed, as compared with the right. **(C)** The computed tomography scan reveals posterior displacement of the medial clavicle back into the mediastinum, compressing the great vessels and slightly displacing the trachea. **(D)** The patient was carefully prepared for a surgical repair, in cooperation with a cardiovascular surgeon. The patient was prepared from the base of his neck down to the umbilicus, so that we could manage any type of vascular problem or complication. Open reduction was accomplished without any vascular incident. The medial end of the clavicle was totally unstable, so the medial 2 cm was resected and the remaining clavicle stabilized to the first rib. **(E)** Four months after surgery, the slight anterior displacement of the clavicle was essentially asymptomatic and the remaining clavicle was stable. (Reprinted with permission from Rockwood CA, Matsen F III, eds. *The shoulder.* Philadelphia: WB Saunders, 1990:Fig. 13-25.)

after surgery. At 1 to 4 years' follow-up, most of the operative cases had a slight deformity. In two patients, migration of the Kirschner wires was noted but was without clinical significance. Eskola and associates strongly urged operative repair of dislocations of the sternoclavicular joint.[59,60] In 1989, they reported on 12 patients treated for painful sternoclavicular joints. The average time from injury was 1.5 years, and the average follow-up after surgery was 4.7 years. In five patients, the sternoclavicular joint was stabilized with a tendon graft from either the palmaris tendon or the

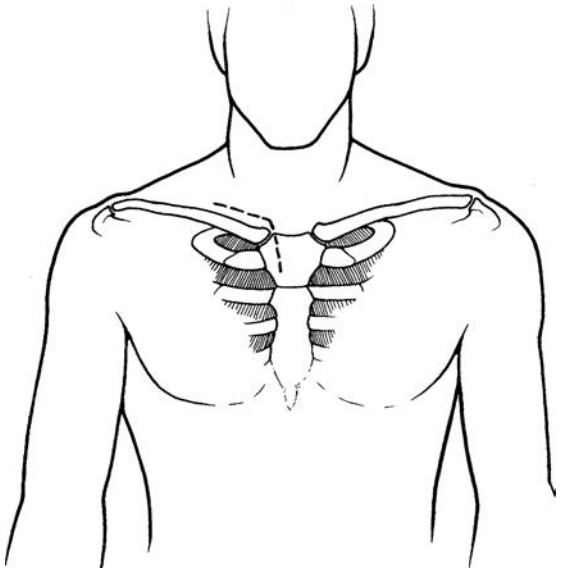


Figure 33-46 Proposed skin incision for open reduction of a posterior sternoclavicular dislocation.

plantaris tendon between the first rib and the clavicle; in four patients, the medial 2.5 cm of the clavicle was resected without any type of stabilization; in three patients, the clavicle was fixed to the first rib with a fascia lata graft. They had four fair results and four poor results in those patients who underwent only resection of the medial clavicle. There was little discussion of the preoperative symptoms, work habits, range of motion, or degree of joint reduction following the surgery. In 1990, Tricoire and colleagues reported six retrosternal dislocations of the medial end of the clavicle.[212] They recommended reduction of these injuries secondary to the possible complications arising from protrusion of the clavicle into the thorax. Sternoclavicular capsulorrhaphy was performed in two patients and a subclavius tenodesis was used in the remaining four patients. All joints were temporarily stabilized with sternoclavicular pins for 6 weeks. Results were satisfactory in all cases at a mean follow-up of 27 months. These case series emphasize the importance of stabilization of the medial end of the clavicle as opposed to simple resection in the case of instability.

Surgical Reconstructions of the Sternoclavicular Joint

There are several surgical procedures to restore stability to the medial end of the clavicle. These can be categorized by the structure or structures that they are attempting to reconstruct. There are three basic styles that have been described. There are those that reconstruct the costoclavicular ligament, those that reconstruct the intraarticular disk ligament, and those that reconstruct the capsular ligaments. Most procedures attempt to reconstruct the costoclavicular ligament with either a local tendon graft or autograft or synthetic material. These include the Burrows reconstruction, which utilizes the subclavius tendon, as well as other fascial loops of autogenous tissue such as fascia lata, which are woven between the first rib and clavicle.[28,59,134,111,171,201] Booth and Roper described a method of using a strip of the sternocleidomastoid tendon looped under the first rib and then secured back to the clavicle.[19] The second method of stabilization reconstructs the intraarticular disk ligament much like a Weaver Dunn reconstruction on the lateral end of the clavicle.[177] A conceptionally similar operation described by Lowman used a loop of fascia in and through the sternoclavicular joint, so that it acts like the ligamentum teres in the hip.[131] The third method of stabilization secures the clavicle to the sternum to reconstruct the capsular ligaments.[6,139,202]

With so many methods to stabilize the sternoclavicular joint, it is difficult to determine the best method. There is not only a lack of agreement on which ligament or ligaments to reconstruct, but also a lack of agreement on how to best reconstruct these structures. This is secondary to the fact that dislocations of the sternoclavicular joint are relatively rare injuries and surgical indications for reconstruction are even rarer. This makes clinical studies to compare one technique with another almost impossible. Thus far, several authors have reported small case series, each with a different technique, but basing reconstruction techniques on these data is almost anecdotal. This emphasizes the need to base reconstruction techniques on biomechanical data elucidating the most important structures to anterior and posterior instability. A biomechanical study by Spencer et al. found that the posterior capsular ligament was the most important structure for anterior and posterior stability, with the anterior capsular ligament serving as an important secondary stabilizer for anterior stability.[203] Therefore, reconstructing these structures would be a logical step.

A separate biomechanical study by Spencer and Kuhn evaluated the stability of the two most common methods of sternoclavicular reconstruction and a novel method that reconstructed the anterior and posterior capsular ligaments.[202] The methods of reconstruction that were compared represented one from each of the three styles described above. The first was the method described by Burrows in which the subclavius tendon is passed through a bone tunnel in the clavicle and sutured back to itself to reconstruct the costoclavicular ligament (Fig. 33-47).[28] The second was a method popularized by the senior author in which the medial end of the clavicle is resected and the intraarticular disk ligament is transferred into the medullary canal (Fig. 33-48).[177] This is similar to a Weaver Dunn reconstruction of the lateral end of the clavicle where the coracoacromial ligament is transferred into the medullary canal of the lateral end of the clavicle. The third method was a novel reconstruction of the anterior and posterior capsular ligaments with a semitendinosis autograft (Fig. 33-49).[202] The semitendinosis is passed in a figure-eight fashion through drill holes in the clavicle and

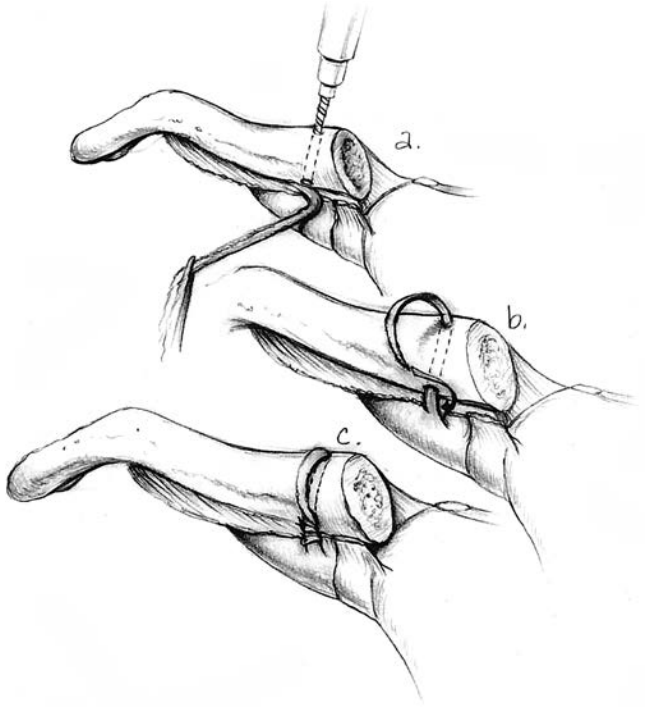


Figure 33-47 Subclavius tendon reconstruction as described by Burrows. **(A)** The subclavius tendon is dissected free, with the sternal attachment left intact, and a drill hole is made in the anterior cortex of the medial part of the clavicle. **(B)** The tendon is routed through the drill hole from inferior to superior. **(C)** The subclavius tendon is sutured to itself. (From Spencer EE Jr, Kuhn JE. Biomechanical analysis of reconstructions for sternoclavicular joint instability. *J Bone Joint Surg Am* 2004;86-A(1):98–105.)

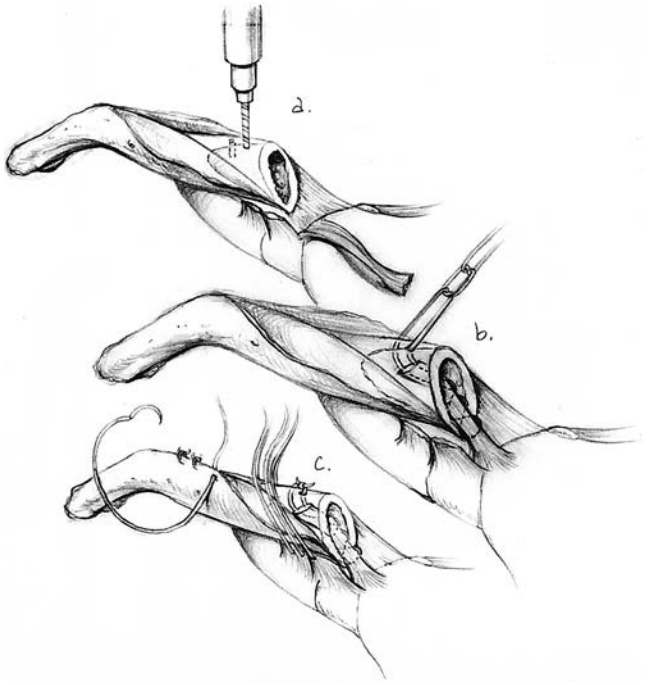


Figure 33-48 Intramedullary ligament reconstruction as described by Rockwood et al. **(A)** The medial head of the clavicle is resected, and the intraarticular ligament and disc are isolated. Two small drill holes are made in the superior surface of the clavicle. **(B)** Sutures are woven into the intraarticular ligament and disk, passed into the medullary canal and then through the drill holes on the superior surface of the clavicle, and secured. **(C)** The soft tissues are sutured to the costoclavicular ligament. (From Spencer EE Jr, Kuhn JE. Biomechanical analysis of reconstructions for sternoclavicular joint instability. *J Bone Joint Surg Am* 2004;86-A(1):98–105.)

manubrium and tied back on itself on the anterior aspect of the joint. The load to failure was almost three times greater for the semitendinosis figure-eight reconstruction in both the anterior and posterior directions.

Description of Techniques

Subclavius Tendon

Burrows recommended that the subclavius tendon be used to reconstruct a new costoclavicular ligament.[28] The origin of the subclavius muscle is from the first rib just 6 mm lateral and 1.3 mm anterior to the attachment of the costoclavicular ligament. The insertion of the tendon is to the inferior surface of the junction of the middle third with the outer third of the clavicle, and the muscle fibers arising from the tendon insert into the inferior surface of the middle third of the clavicle. The muscle fibers coming off the tendon look like feathers on a bird's wing. Burrows detaches the muscle fiber from the tendon, does not disturb the origin of the tendon, and then passes the tendon through drill holes in the anterior proximal clavicle.

In comparing his operation with the use of free strips of fascia, Burrows said that it is "safer and easier to pick up a mooring than to drop anchor; the obvious mooring is the tendon of the subclavius, separated from its muscle fiber and suitably realigned." Lunseth and associates have reported a modified Burrows procedure with the additional use of a threaded Steinmann pin across the joint.[134]

Intraarticular Ligament Transfer

The medial clavicle is exposed by careful subperiosteal dissection (Fig. 33-50). When possible, any remnant of the capsular or intraarticular disk ligaments should be identified and preserved, because these structures can be used to stabilize the medial clavicle. Ordinarily, the intraarticular disc ligament is a very dense, fibrous structure that arises from the synchondral junction of the first rib to the sternum and passes through the sternoclavicular joint, dividing the joint into two separate spaces. The capsular ligament covers the anterosuperior and posterior aspects of the joint and represents thickenings of the joint capsule. This ligament is primarily attached to the epiphysis of the medial clavicle and is usually avulsed from this structure with posterior sternoclavicular dislocation. Similarly, the intraarticular disk ligament usually is intact where it arises from the synchondral junction of the first

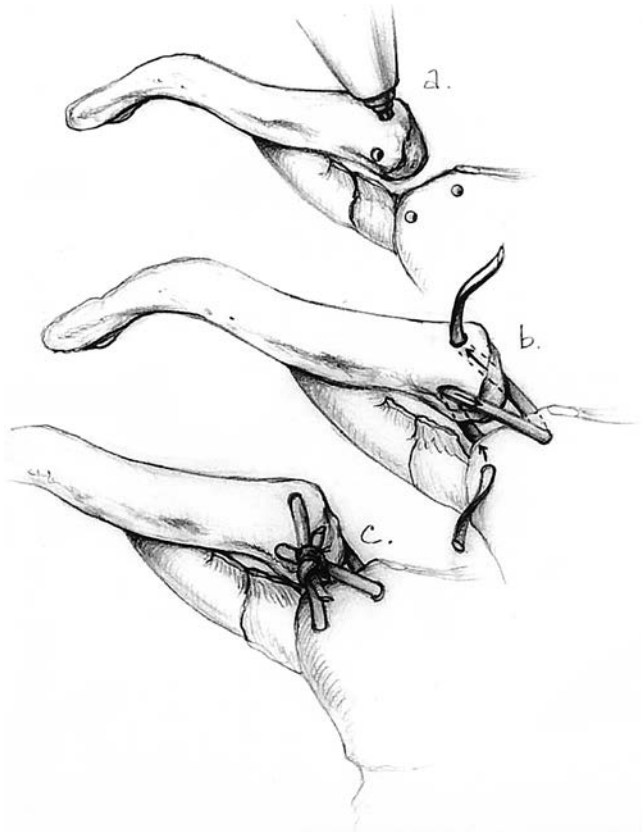


Figure 33-49 Semitendinosus figure-eight reconstruction. **(A)** Drill holes are passed from anterior to posterior through the medial part of the clavicle and the manubrium. **(B)** A free semitendinosus tendon graft is woven through drill holes such that the tendon strands are parallel to each other posterior to the joint and cross each other anterior to the joint. **(C)** The tendon is tied in a square knot and is secured with suture. (From Spencer EE Jr, Kuhn JE. Biomechanical analysis of reconstructions for sternoclavicular joint instability. *J Bone Joint Surg Am* 2004;86-A(1):98–105.)

rib and sternum and is avulsed from its attachment site on the medial clavicle.

If the sternal attachment site of these structures is intact, a nonabsorbable No. 1 cottony Dacron suture is woven back and forth through the ligament, so that the ends of the suture exit through the avulsed free end of the tissue. The medial end of the clavicle is resected, with care taken to protect the underlying structures and to not damage the costoclavicular (rhomboid) ligament or the vascular structures that are located posterior to the medial clavicle and sternoclavicular joint. These vital structures are protected by passing a curved Crego or ribbon retractor around the posterior aspect of the medial clavicle, which isolates them from the operative field during the bony resection. Excision of the medial clavicle is facilitated by creating drill holes through both cortices of the clavicle at the intended site of clavicular osteotomy. Following this step, an air drill with a side-cutting burr or an osteotome is used to complete the osteotomy. The anterior and superior corners of the clavicle are beveled with an air burr for cosmetic purposes.

The medullary canal of the medial clavicle is drilled out and curetted to receive the transferred intraarticular disk ligament (see Fig. 33-48). Two small drill holes are then placed in the superior cortex of the medial clavicle, approximately 1 cm lateral to the site of resection. These holes communicate with the medullary canal and will be used to secure the suture in the transferred ligament. The free ends of the suture are passed into the medullary canal of the medial clavicle and out the two small drill holes in the superior cortex of the clavicle. While the clavicle is held in a reduced anteroposterior position in relationship to the first rib and sternum, the sutures are used to pull the ligament tightly into the medullary canal of the clavicle. The suture is tied, thus securing the transferred ligament into the clavicle. The stabilization procedure is completed by passing several 1-mm cottony Dacron sutures around the medial end of the remaining clavicle and securing the periosteal sleeve of the clavicle to the costoclavicular ligament. Postoperatively, the patient should be wrapped with the shoulders back in a figure-eight dressing for 4 to 6 weeks to allow for healing of the soft tissues. We do not recommend the use of Kirschner wires, Steinmann pins, or any other type of metallic pins to stabilize the sternoclavicular joint. The complications are horrendous, as will be discussed in the section on complications.

Semitendinosus Figure-Eight Reconstruction

The approach is similar to the other methods. A subperiosteal dissection is used to expose the clavicle. Any remaining tissue should be preserved. If there are degenerative changes within the joint, then the medial 1 cm can be resected. Subperiosteal dissection allows a malleable retractor to be placed behind the clavicle and manubrium to protect the posterior structures. A portion of the insertion of the sternocleidomastoid tendon will need to be reflected superiorly to expose the anterior aspect of the manubrium. Drill holes are then placed in an anterior-to-posterior fashion. Two holes are drilled in the clavicle and two in the manubrium. A bone bridge of at least 1 cm should be maintained. We start with a 2.5-mm drill bit and then move up to a 4-mm drill bit. Four millimeters is usually required to pass the semitendinosus graft. The edges of the holes are beveled and the tendon is passed with a Hewson suture passer. The graft is positioned such that limbs are parallel posteriorly. The anterior limbs are crossed and tied in a square knot while the joint is held in a reduced position (see Fig. 33-49). A braided nonabsorbable suture is passed through the knot to secure the fixation. The excess tendon tissue is then placed within the joint as a small anchovy. Any remaining capsular tissue is reapproximated. The patient is placed in a sling for 6 weeks. Gentle active assisted range-of-motion exercises in the supine position are started at 6 weeks. Active motion is started at 10 to 12 weeks. Strengthening exercises can be initiated thereafter. There are no clinical case series reported yet, but

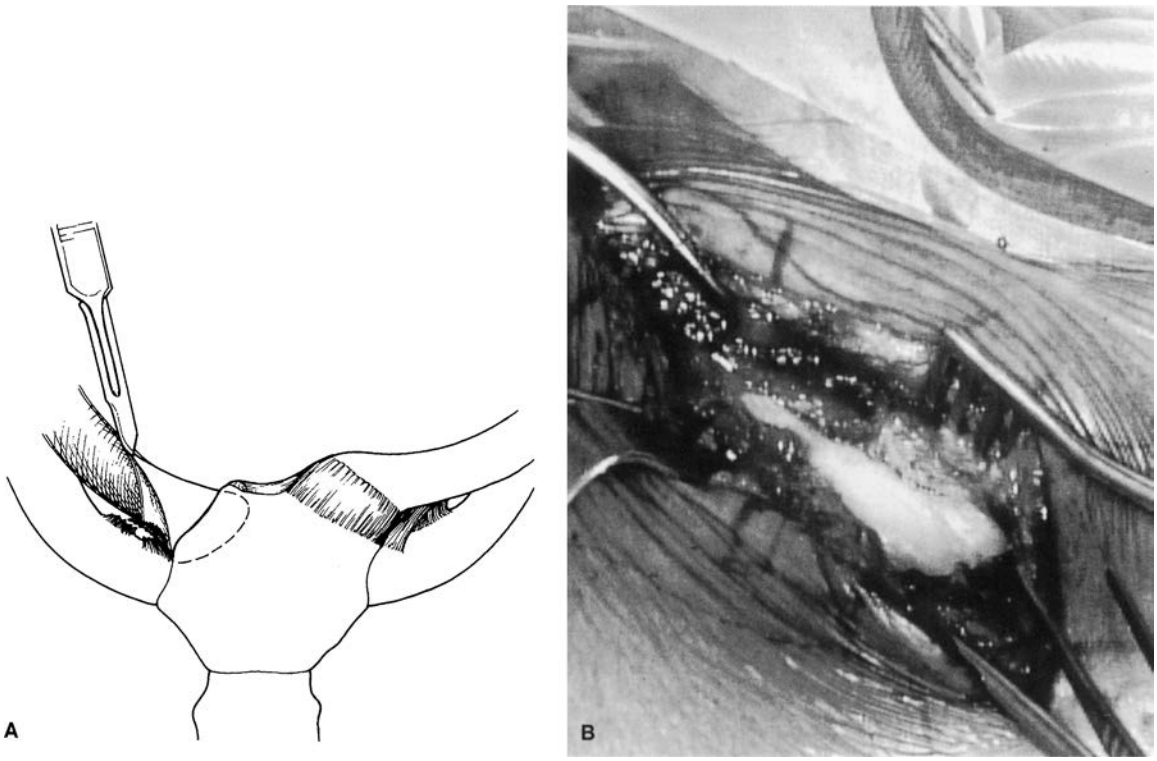


Figure 33-50 Artist's depiction **(A)** and intraoperative photo **(B)** demonstrating subperiosteal exposure of the medial clavicle. Note the posteriorly displaced medial end of the clavicle. (From Wirth MA, Rockwood CA. Injuries to the sternoclavicular joint. In: Rockwood CA, Green DP, Bucholz RW, Hechman JD, eds. *Fractures in adults.* Philadelphia: JB Lippincott, 1996.)

the primary author as well as others have reported good results with this technique.[94117] At the time of the writing of this chapter a clinical study was ongoing.

Complications

Through 1992, seven deaths and three near deaths from complications of transfixing the sternoclavicular joint with Kirschner wires or Steinmann pins were reported.[42,61,62,118,135,184,196,214] The pins, either intact or broken, migrated into the heart, pulmonary artery, innominate artery, or aorta. Tremendous leverage force is applied to pins that cross the sternoclavicular joint, and fatigue breakage of the pins is common. To our knowledge, there were no deaths reported that occurred as a result of migrating pins from the sternoclavicular joint, until the report in 1984 by Gerlach and associates from West Germany.[76] They reported two deaths from migrating nails that caused cardiac tamponade. The physicians were charged with manslaughter by negligence. We do not recommend the use of any transfixing pins—large or small, smooth or threaded, bent or straight—across the sternoclavicular joint.

Brown has reported three complications in 10 operative cases: two from broken pins that had to be removed from a window in the sternum, and one a near death, in which the pin penetrated the back of the sternum and entered the right pulmonary artery.[24] Nordback and Markkula removed a pin that migrated completely inside the aorta.[150] Jelesijevic and associates; Pate and Wilhite; and Rubenstein and colleagues reported cases where the pin migrated into the heart.[100,156,183] Leonard and Giffors and Liu and coworkers reported migration to the pulmonary artery.[125,130] Sethi and Scott reported migration of the pin to lacerate the subclavian artery.[191] Ferrandez and associates described two cases of Kirschner wire migration into the mediastinum.[61] Nettles and Linscheid as well as Salvatore reported migration of pins into the aorta and resultant death.[145,187] Grabski reported migration of the pin to the opposite breast in a 37-year-old woman.[80] In addition, the senior author has personally treated patients in whom the pin has migrated into the chest and up into the base of the neck.

Omer,[86] in a review of 14 military hospitals, reported 15 patients who had elective surgery for reduction and reconstruction of the sternoclavicular joint.[152] Eight patients were followed by the same house staff for more than 6 months with the following complications: of the five patients who had internal fixation with metal, two developed osteomyelitis, two had fracture of the pin with recurrent dislocation, and one had migration of the pin into the mediastinum with recurrent dislocation. Of the three patients who had soft tissue reconstructions, two developed recurrent dislocation (one with drainage) and the third developed arthritis and extremity weakness and

was discharged from military service. Omer commented on this series of complications: "It would seem that complications are common in this rare surgical problem." To Omer's comment we can only add, "Amen."

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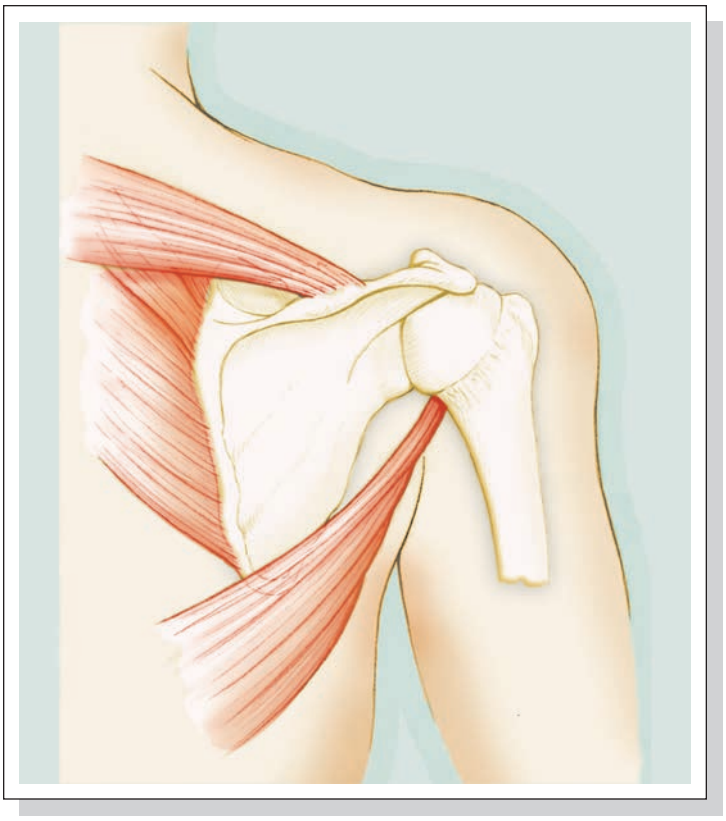
Neuromuscular and Scapulothoracic Disorders

VII



34 The Scapulothoracic Articulation: Anatomy, Biomechanics, Pathophysiology, and Management

John E. Kuhn

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INTRODUCTION

The scapulothoracic articulation is a critical component of shoulder function, yet this region, and the disorders that affect it, have received far less attention in the medical literature than the glenohumeral joint. Disorders of the scapulothoracic articulation are frequently misunderstood and misdiagnosed. An appreciation of the anatomy of this region helps in understanding the pathologic conditions that may affect the scapulothoracic articulation.

Surgical Anatomy and Biomechanics of the Normal Scapulothoracic Articulation

The scapula is a thin bone that serves as a site of muscle attachment for the upper extremity and the thorax. Seventeen muscles have their origin or insertion on the scapula (Table 34-1, Fig. 34-1), making it the essential link for coordinated upper-extremity activity. These muscles can be divided into three major groups.

The scapulothoracic muscles coordinate scapulothoracic motion and include the rhomboideus major and minor, the levator scapulae, the serratus anterior, the trapezius, the omohyoid, and the pectoralis minor. Disorders of these muscles may manifest as scapular winging, or scapulothoracic dyskinesia. The rotator cuff muscles control activities of the glenohumeral articulation and include the supraspinatus, infraspinatus, subscapularis, and teres minor. Disorders of these muscles are common and are covered in other sections of this text. The scapulohumeral muscles provide power to the humerus and include the deltoid, the long head of the biceps, the short head of the biceps, the coracobrachialis, the long head of the triceps, and the teres major. Motion of the upper extremity occurs as a result of the coordinated activity of all of these muscles.

TABLE 34-1
MUSCULAR ATTACHMENTS TO THE SCAPULA

Scapulohumeral Muscles
Long head of biceps
Short head of biceps
Deltoid
Coracobrachialis
Teres major
Long head of triceps

Scapulothoracic Muscles
Levator scapulae
Omohyoid
Rhomboid major
Rhomboid minor
Serratus anterior
Trapezius
Pectoralis minor

Rotator Cuff Muscles
Supraspinatus
Infraspinatus
Subscapularis
Teres Minor

Seventeen muscles attach the scapula to the neck, thorax, and humerus, making it the control tower for coordinated upper-extremity activity. Fig. 34-1 displays the location of the attachments of the various muscles.

While at rest, the scapula is anteriorly rotated relative to the trunk approximately 30 degrees.[101,169] The medial border of the scapula is also rotated with the inferior pole diverging away from the spine approximately 3 degrees. The scapula is also tilted forward about 20 degrees in the sagittal plane when viewed from the side[101] (Fig. 34-2). It is thought by some that deviations in this normal alignment may contribute to glenohumeral instability.[181]

Almost every functional upper-extremity movement has components of scapulothoracic and glenohumeral motion. Arm elevation has been studied extensively with efforts directed toward determining the relative contributions of scapulothoracic and scapulohumeral motion with varied and conflicting results. In general, in the first 30 degrees of elevation, the majority of motion occurs at the glenohumeral joint, although this varies between individuals.[8,39,53,185] The next 60 degrees of elevation is accomplished by equal parts of scapulothoracic and glenohumeral motion. The overall effort to obtain 90 degrees of elevation requires approximately a 2:1 ratio of glenohumeral-to-scapulothoracic motion. During arm elevation in the scapular plane, the instant center of rotation of the scapula moves, such that from 0 to 30 degrees the scapula rotates about its midportion and from 60 degrees or more, the rotation occurs near the glenoid, resulting in a medial and upward displacement of the inferior pole of the scapula. In addition, with arm elevation, the coracoid moves superiorly, and the acromion moves superiorly and posteriorly to open the impingement zone (Fig. 34-3).

Muscle Function about the Scapula

Cranial elevation of the scapula is accomplished by activating the upper portion of the trapezius as it inserts into the lateral spine of the scapula, acromion, and distal clavicle. This is countered by the effects of gravity and by the latissimus dorsi, which acts as the primary scapular depressor with the lower portion of the serratus anterior, the pectoralis minor, and the lower portion of the trapezius contributing.

Rotation of the scapula upward is initiated by the middle portion of the trapezius, which stabilizes the scapula by inserting on the medial border of the scapular spine. At

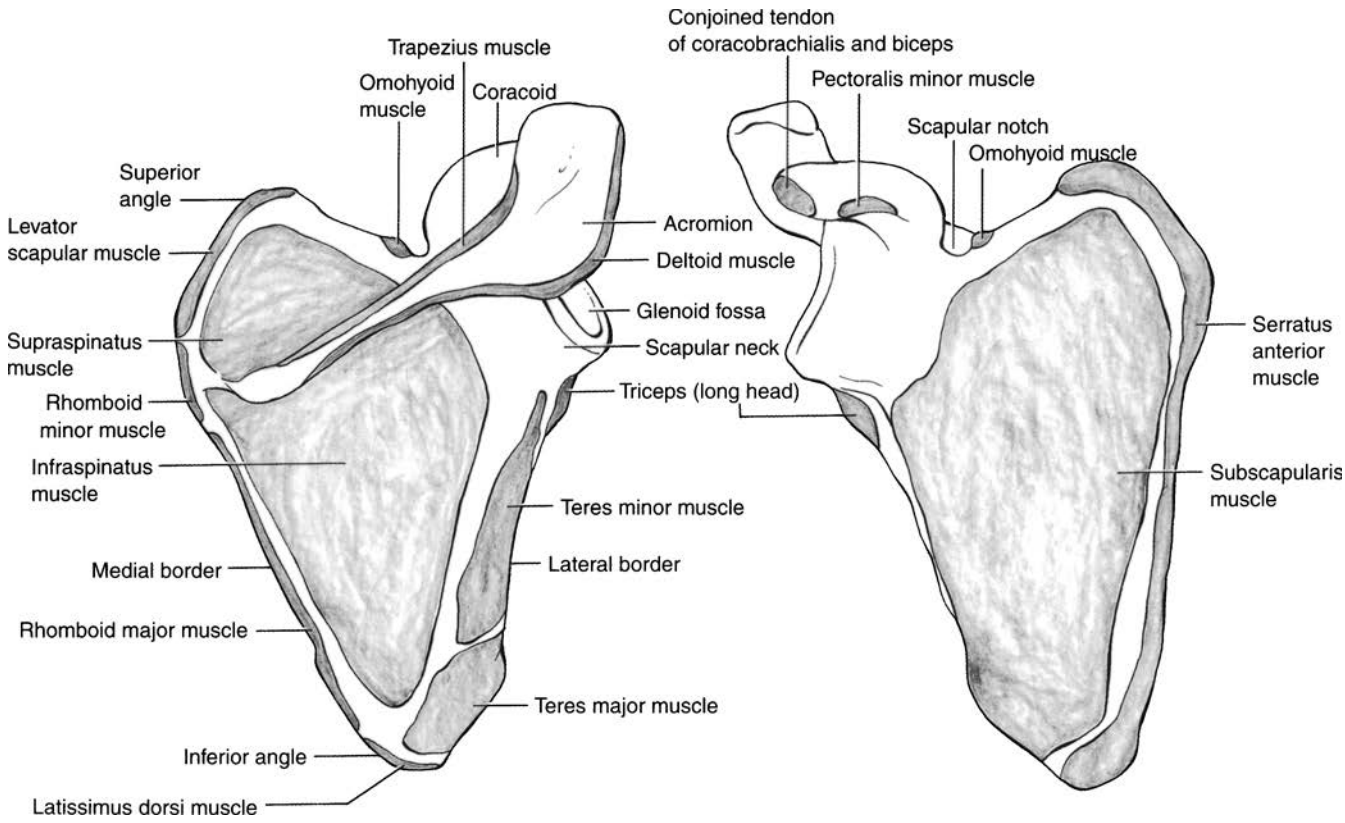


Figure 34-1 Origins and insertions of the muscles of the scapula. Anterior and posterior views of the scapula demonstrate the multiple attachment sites for muscles of the scapula, making the scapula the center for coordinated upper extremity motion.

approximately 45 degrees of scapular abduction, the serratus anterior acts to pull the inferior angle of the scapula laterally. The upper portion of the trapezius elevates the lateral angle of the scapula, while the lower portion of the trapezius pulls down on the scapula through its insertion on the medialmost portion of the scapular spine, inducing upward rotation (Fig. 34-4).

The downward rotators of the scapula include the rhomboideus major and minor and the levator scapulae, which elevate the medial border of the scapula while the pectorals minor, lower portion on the pectoralis major, and the latissimus dorsi depress the lateral portion of the scapula (Fig. 34-5).

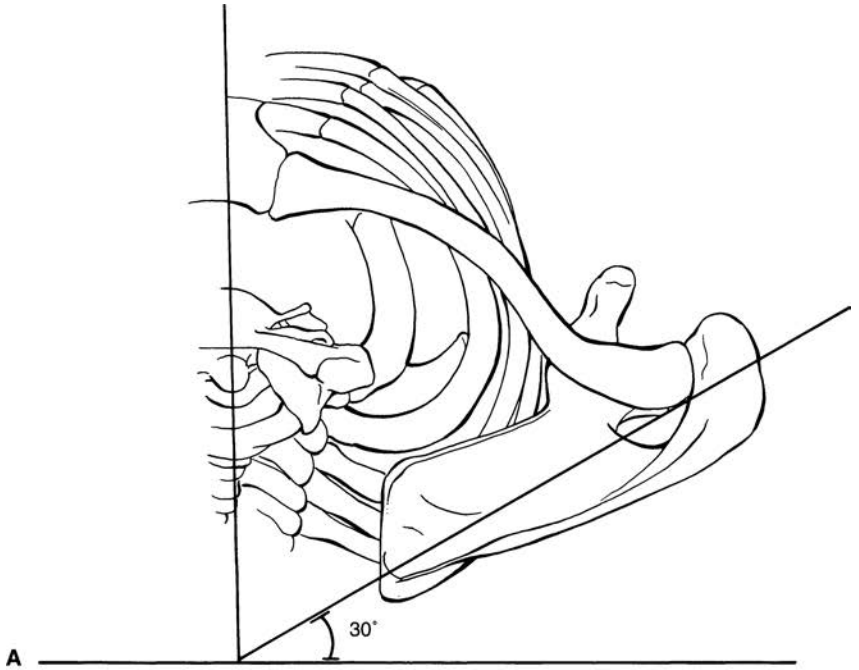
Protraction of the scapula is accomplished by the serratus anterior and pectoralis minor and major as these muscles move the scapula lateral and forward. Retraction of the scapula relies on the middle trapezius and rhomboideus major and minor (Fig. 34-6).

During high-intensity shoulder activities, such as throwing, the trapezius and rhomboideus major and minor have high electromyographic activity during the windup and cocking phases to maximally retract and stabilize the scapula. For acceleration and follow-through the scapula is protracted with serratus anterior and pectoralis minor and major. This is thought to allow the scapula to follow the humeral head, maintaining glenohumeral stability, and help dissipate the tremendous forces generated during the cocking and acceleration phases of throwing.[55,90]

Bursae About the Scapula

Scapulothoracic bursae allow for smooth, gliding scapulothoracic motion. Two major or anatomic bursae and four minor or adventitial bursae have been described for the scapulothoracic articulation (Table 34-2, Fig. 34-7). The first major bursa is found in the space between the serratus anterior muscle and the chest wall. The second major bursa is located between the subscapularis and the serratus anterior muscles.[23,95] These bursae are easily and reproducibly found.[95]

Clinical scapulothoracic bursitis seems to affect two areas of the scapulothoracic articulation, the superomedial angle and the inferior angle of the scapula. When symptomatic, these areas tend to develop inflamed bursae; however, these bursae are not reliably found, and



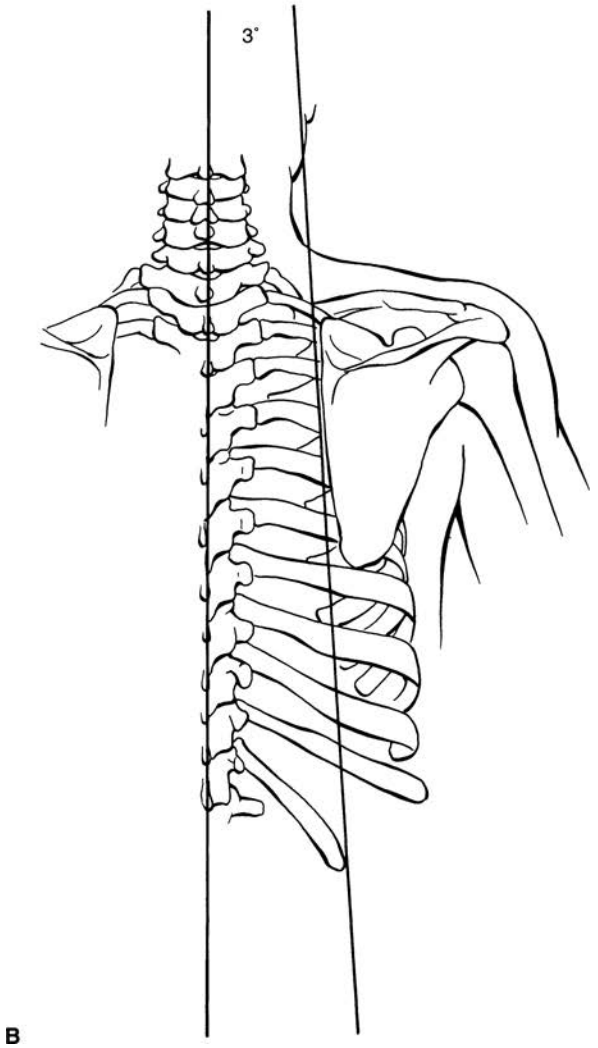


Figure 34-2 Normal resting position of the scapula on the thorax. The resting scapula is 30 degrees anterior to the coronal plane, with the medial border rotated 3 degrees from the spine.

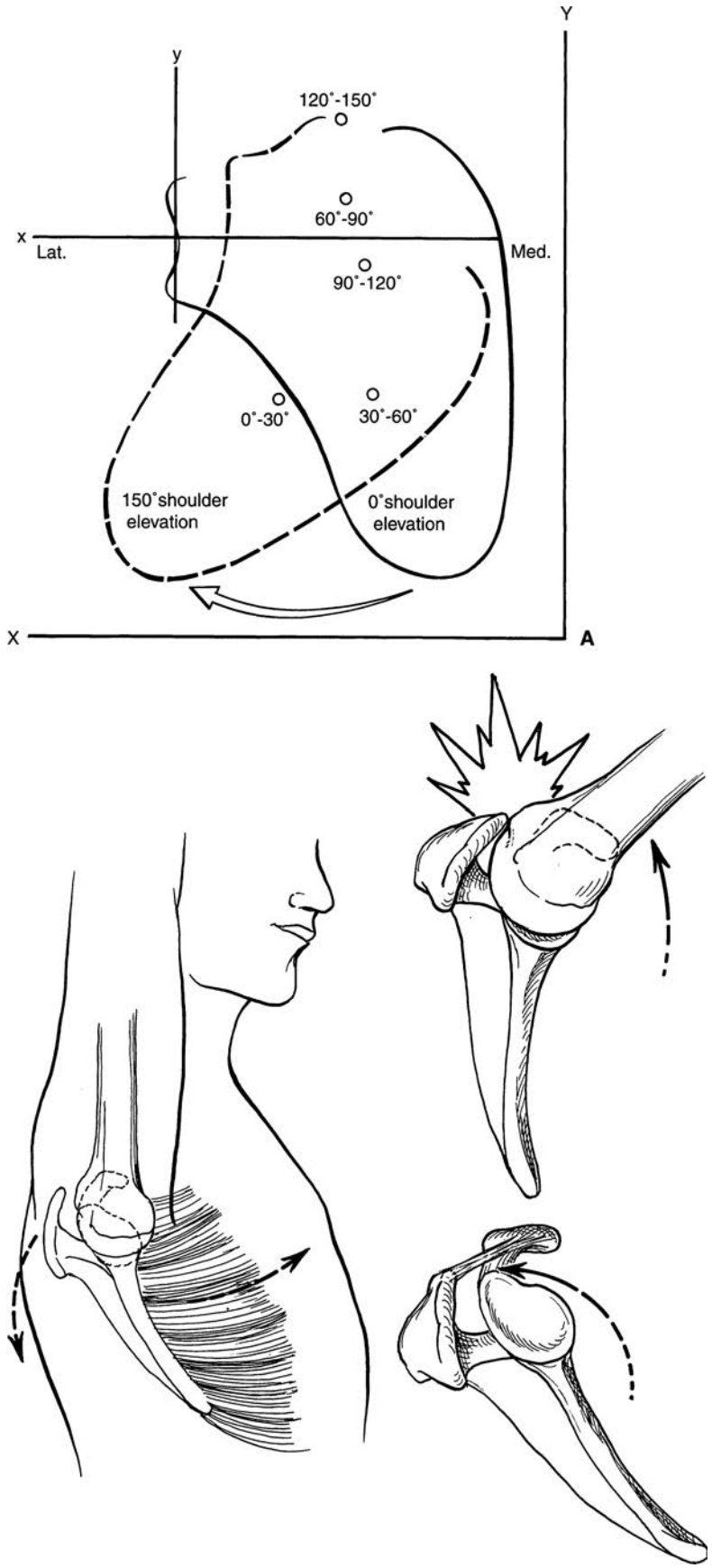


Figure 34-3 Changes in the center of rotation of the scapula with arm elevation. **(A)** demonstrates that the center of rotation moves superiorly during arm elevation, resulting in upward and medial displacement of the glenoid face, with lateral displacement of the inferior angle. (Reprinted with permission from Poppen NK, Walker PS. Normal and abnormal motion of the shoulder. *J Bone Joint Surg Am* 1976;58A:195–201.) **(B)** demonstrates that the scapula rotates with arm elevation clearing the acromion away from the impingement zone. (Reprinted with permission from Kuhn JE, Hawkins RJ. Evaluation and treatment of scapular disorders. In: Warner JP, Iannotti JP, Gerber C, eds. *Complex and revision problems in shoulder surgery.* Philadelphia: Lippincott-Raven Publishers, 1997:357–375.)

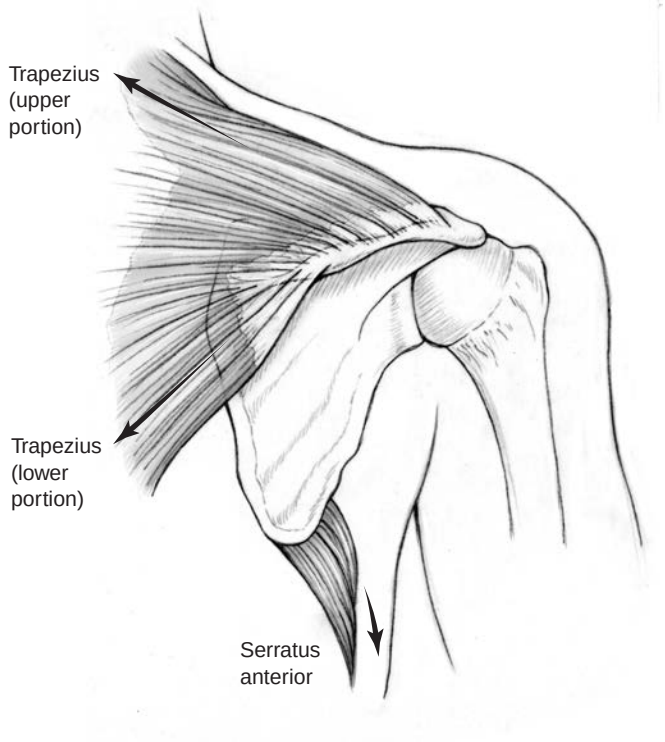


Figure 34-4 Upward rotation of the scapula. As the middle trapezius stabilizes the scapula, the serratus pulls the inferior angle and the upper trapezius elevates the lateral angle.

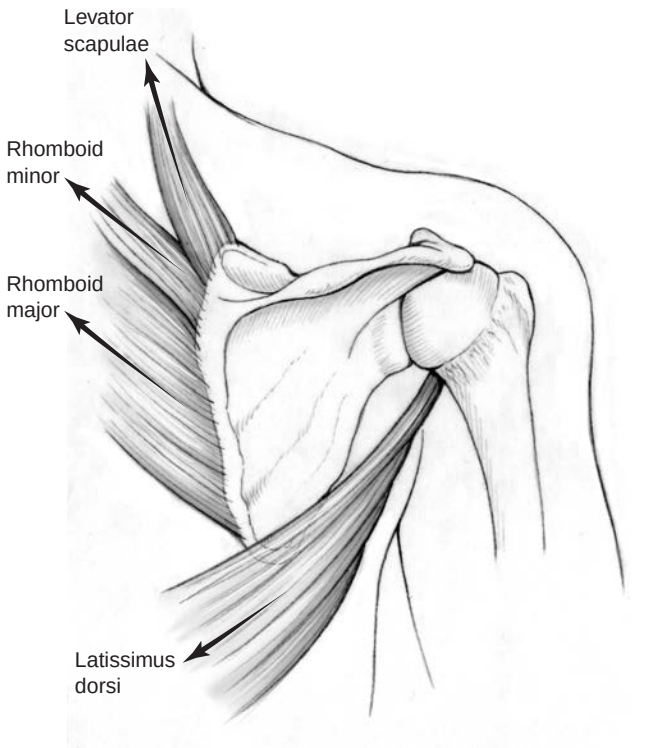


Figure 34-5 Downward rotation of the scapula. The rhomboidei and levator scapulae elevate the medial border of the scapula while the pectoralis minor, lower pectoralis major, and latissimus dorsi depress the lateral portion of the scapula.

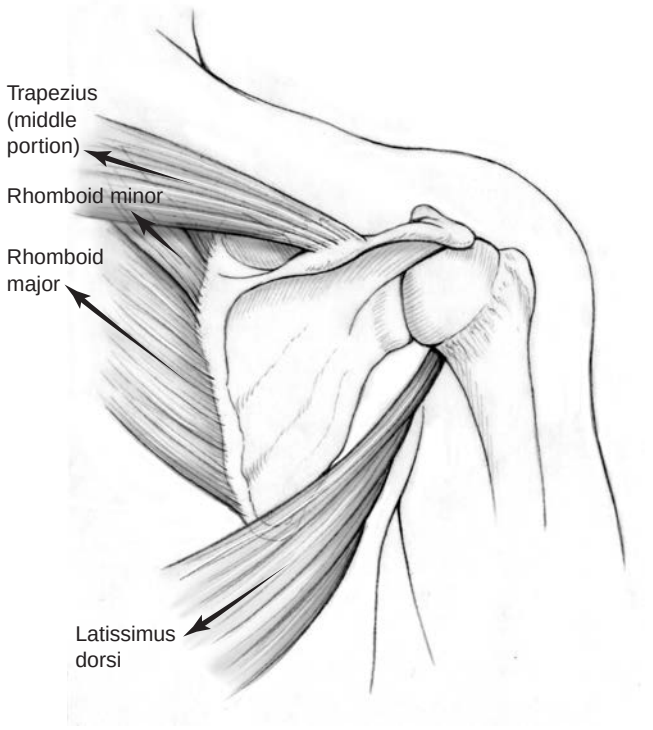


Figure 34-6 Retraction of the scapula. The middle portion of the trapezius and the rhomboidei serve to counter the effect of the serratus and pectoralis and retract the scapula toward the spine.

TABLE 34-2
BURSAE OF THE SCAPULA

Major/Anatomic Bursae

Infraserratus bursae—between the serratus anterior and chest wall

Supraserratus bursae—between the subscapularis and serratus anterior muscles

Minor/Adventitial Bursae

SUPEROMEDIAL ANGLE OF THE SCAPULA

Infraserratus bursae—between serratus anterior and chest wall

Supraserratus bursae—between subscapularis and serratus anterior

INFERIOR ANGLE OF THE SCAPULA

Infraserratus bursae—between serratus anterior and chest wall

SPINE OF SCAPULA

Trapezoid bursae—between medial spine of scapula and trapezius

The scapulothoracic articulation is associated with a number of bursae, many of which can become pathologic, causing symptomatic scapulothoracic bursitis. (Reprinted with permission from Kuhn JE, Hawkins RJ. Evaluation and treatment of scapular disorders. In: Warner JP, Iannotti JP, Gerber C, eds. *Complex and revision problems in shoulder surgery.* Philadelphia: Lippincott-Raven Publishers, 1997:357–375.)

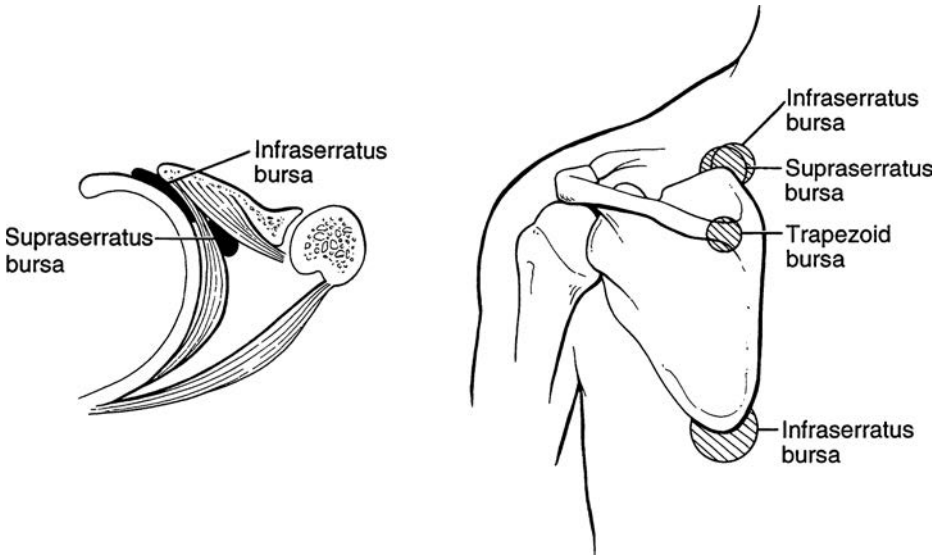


Figure 34-7 Bursae of the scapula. The location of both anatomic (black) and adventitial (hatched) bursae are displayed. (Reprinted with permission from Kuhn JE, Hawkins RJ. Evaluation and treatment of scapular disorders. In: Warner JP, Iannotti JP, Gerber C, eds. *Complex and revision problems in shoulder surgery.* Philadelphia: Lippincott-Raven Publishers, 1997:357–375.)

in fact may be adventitious.[24,95,136] With regard to the inferior angle of the scapula, most authors agree that the affected bursa lies between the serratus anterior muscle and the chest wall.[25,121,162] This bursa has been given many names including the infraserratus bursa[25] and the bursa mucosa serrata.[162,182] The second site of pathology lies at the superomedial angle of the scapula. Here Codman[25] believed this bursa is also an infraserratus bursa, lying between the upper and anterior portion of the scapula and the back of the first three ribs. Von Gruber,[182] on the other hand, identified a bursa in this region between the subscapularis and the serratus anticus muscles, which he called the bursa mucosa angulae superioris scapulae.

A third minor or adventitial bursa, which Codman believed was the site of painful crepitus in scapulothoracic crepitus, was called the trapezoid bursa and is found over the triangular surface at the medial base of the spine of the scapula under the trapezius muscle.[20] Some believe that these minor bursae are adventitial and develop in response to abnormal pathomechanics of the scapulothoracic articulation.[24,95,136] It would not be surprising, then, to find these bursae inconsistently or in different soft tissue planes.

WINGING OF THE SCAPULA

Scapular winging is one of the most common abnormalities of the scapulothoracic articulation and results from a variety of causes. Winging may be described as primary, secondary, or voluntary (Table 34-3). Primary scapular winging results from identifiable anatomic disorders that directly affect the scapulothoracic articulation. Secondary scapular winging usually is associated with some form of glenohumeral pathology. This type of winging will resolve as the glenohumeral pathology is addressed. Voluntary winging, which is quite rare, may have an underlying psychologic cause.

Primary Scapular Winging—Neurologic Disorders

The more common causes of primary scapular winging are attributable to neurologic disorders and include damage to the spinal accessory nerve causing trapezius palsy, damage

TABLE 34-3
WINGING OF THE SCAPULA

I. Primary Scapular Winging
  A. Neurologic
    1. Long thoracic nerve—trapezius palsy
    2. Spinal accessory nerve—serratus anterior palsy
    3. Dorsal scapular nerve—rhomboideus palsy
  B. Bony
    1. Osteochondromas
    2. Fracture malunions
  C. Soft Tissue
    1. Contractural winging
    2. Muscle avulsion/muscle agenesis
    3. Scapulothoracic bursitis
II. Secondary scapular winging
III. Voluntary scapular winging

Primary scapular winging results from anatomic disorders of the scapulothoracic articulation. Secondary scapular winging results from glenohumeral or subacromial pathology. Voluntary scapular winging is not pathologic and may have psychologic overtones. (Reprinted with permission from Kuhn JE, Hawkins RJ. Evaluation and treatment of scapular disorders. In: Warner JP, Iannotti JP, Gerber C, eds. *Complex and revision problems in shoulder surgery.* Philadelphia: Lippincott-Raven Publishers, 1997:357–375.)

to the long thoracic nerve causing serratus anterior palsy, and damage to the fifth cervical nerve root causing rhomboideus palsy. Primary scapular winging may also be bony in origin or may be due to scapulothoracic bursitis or disorders in periscapular musculature.

Spinal Accessory Nerve Palsy and Trapezius Winging

The spinal accessory nerve is the only nerve supplying the vast trapezius muscle.[75,147,174] The spinal accessory nerve is superficial, lying in the subcutaneous tissue on the floor of the posterior cervical triangle. Its superficial location makes it susceptible to injury[85,86] (Fig. 34-8). Injury to this nerve causes significant deformity as well as painfully disabling alterations in scapulothoracic function.[35,42,74,85,86,100,131,174,195–197] This nerve can be injured in a variety of ways including blunt trauma,[11,73,197] stretching of the nerve,[197] and penetrating trauma, which includes surgical biopsy of lymph nodes in the posterior cervical triangle[42,131,196,197] and radical neck dissection.[14,147,164]

Signs and Symptoms

Patients will attempt to compensate for a palsy of the trapezius by straining other muscles of the shoulder girdle including the levator scapulae and the rhomboid muscles. This strain may lead to pain and muscle spasm, which can be disabling.[11] Patients can also develop pain from adhesive capsulitis, shoulder subacromial impingement, and radiculitis from traction on the brachial plexus as the shoulder girdle droops.

Upon examination, patients will have difficulty when attempting to shrug their shoulder, and will have weakness in forward elevation and abduction of the arm. The patient will assume a position with the shoulder depressed and the scapula translated laterally with the inferior angle rotated laterally (Fig. 34-9).

Diagnosis and Imaging

The diagnosis is confirmed by electromyography. It is important to specify an examination of the trapezius and spinal accessory nerve on the requisition, as these areas may not be routinely evaluated in a diagnostic evaluation of the shoulder. Serial electromyography at 3-month intervals may be helpful to document the potential for recovery, which may take many months.

Natural History

In the majority of patients, spinal accessory nerve injuries are iatrogenic and are a complication of neck surgery, either radical dissections or lymph node biopsy. In these patients the nerve may be injured as a result of vigorous retraction, but in many patients the nerve is cut or resected. Patients develop symptoms quickly and note a perceived weakness in the shoulder and a feeling of discomfort sitting against the back of a chair. Clearly a transected nerve

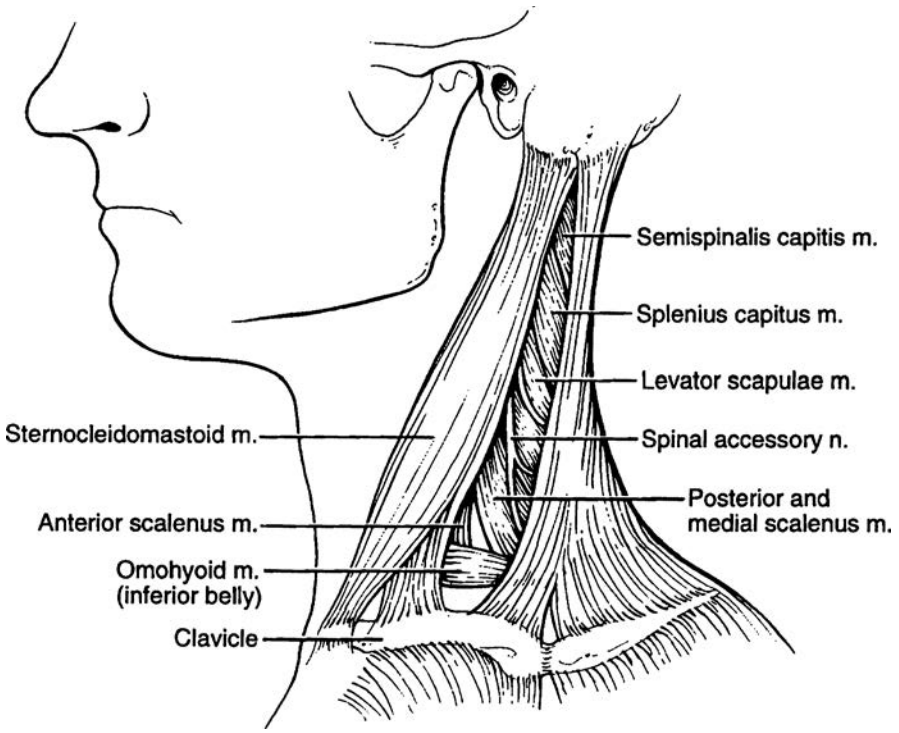


Figure 34-8 Location of the spinal accessory nerve. Its location in the posterior cervical triangle makes it susceptible to injury during surgical procedures in this area.

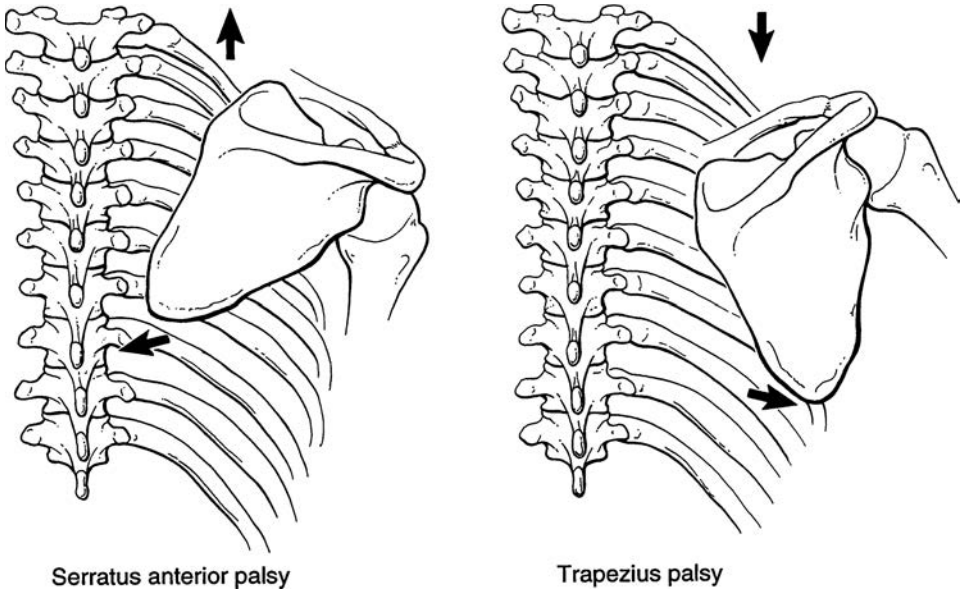


Figure 34-9 Resting location of the scapula with palsy of the serratus anterior and trapezius palsy. The unopposed muscles cause the scapula to drift into a typical position. (Reprinted with permission from Kuhn JE, Hawkins RJ. Evaluation and treatment of scapular disorders. In: Warner JP, Iannotti JP, Gerber C, eds. *Complex and revision problems in shoulder surgery.* Philadelphia: Lippincott-Raven Publishers, 1997: 357–375.)

has no potential for recovery. Nevertheless, it is possible the nerve injury occurred as a result of vigorous retraction and may recover. In all patients serial electromyography will be helpful in assessing the potential for recovery.

Treatment Options

Treatment considerations for patients with trapezius winging depend on the duration and severity of symptoms (Fig. 34-10). The initial treatment is nonoperative. The arm can be placed in a sling to rest the other periscapular muscles. Physical therapy is helpful to maintain glenohumeral motion, preventing stiffness.[137] In cases due to blunt trauma, serial electromyographic analysis should be performed at 4- to 6-week intervals to follow the returning function of the nerve. In cases due to penetrating trauma or when there is no evidence of nerve function on electromyographic analysis, neurolysis and/or nerve grafting can be performed.[1,42,64,130,195] The results of these procedures have been variable; however, the success rate seems to be improved if the neurolysis is performed before 6 months.[11]

Surgical Techniques

Patients who have had symptoms in excess of 1 year are unlikely to benefit from continued nonoperative treatment,[131,174] and surgery can be offered. Historically, a variety of procedures have been described for the treatment of trapezius winging.[35,68,76, 89] These can be divided into static stabilization, including scapulothoracic fusion[68] and any of the many described operations that tether the scapula to the thoracic spine,[35,89] or dynamic stabilization, which involves muscle transfers.[76]

The Dewar-Harris procedure[35] was once a popular operation for trapezius paralysis.[34,104,129,139] In this procedure the medial border of the scapula is stabilized by securing it to the T1 and T2 spinous processes with fascia lata strips, which attempt to compensate for the middle and lower thirds of the trapezius. In addition, the levator scapulae muscle is transferred laterally to the spine of the scapula to compensate for the upper third of the trapezius (Fig. 34-11). Following surgery, the arm is placed in a shoulder spica cast in 45 to 50 degrees of abduction for 6 to 8 weeks. Other operations have been described for trapezius winging that also employ fascial slings, all with the principle effect to compensate for the three portions of the trapezius.[36,40,70,89]

Of historical interest, a partial fusion for trapezius winging has been described by Spira.[165] In this technique, a hole is made in the inferior angle of the scapula. The sixth rib is cut, passed through the hole, and then reapproximated (Fig. 34-12). Complete scapulothoracic fusions can also be performed.[68] However, these are generally reserved for patients with generalized shoulder girdle weakness. Because scapulothoracic fusions limit motion significantly, and because fascial sling suspensions tend to fail, causing a recurrence of winging in 2 to 3 years,[11,76] dynamic muscle transfers have become the procedure of choice for persistent trapezius winging.[11,27,44,98–100,115]

In the Eden-Lange procedure,[44,98,99] the levator scapula, rhomboideus minor, and rhomboideus major muscles are transferred laterally (Fig. 34-13). The levator scapula substitutes for the upper third of the trapezius, the rhomboid major substitutes for the middle third of the trapezius, and the rhomboid minor substitutes for the lower third of the trapezius. By moving these muscle insertions laterally, their

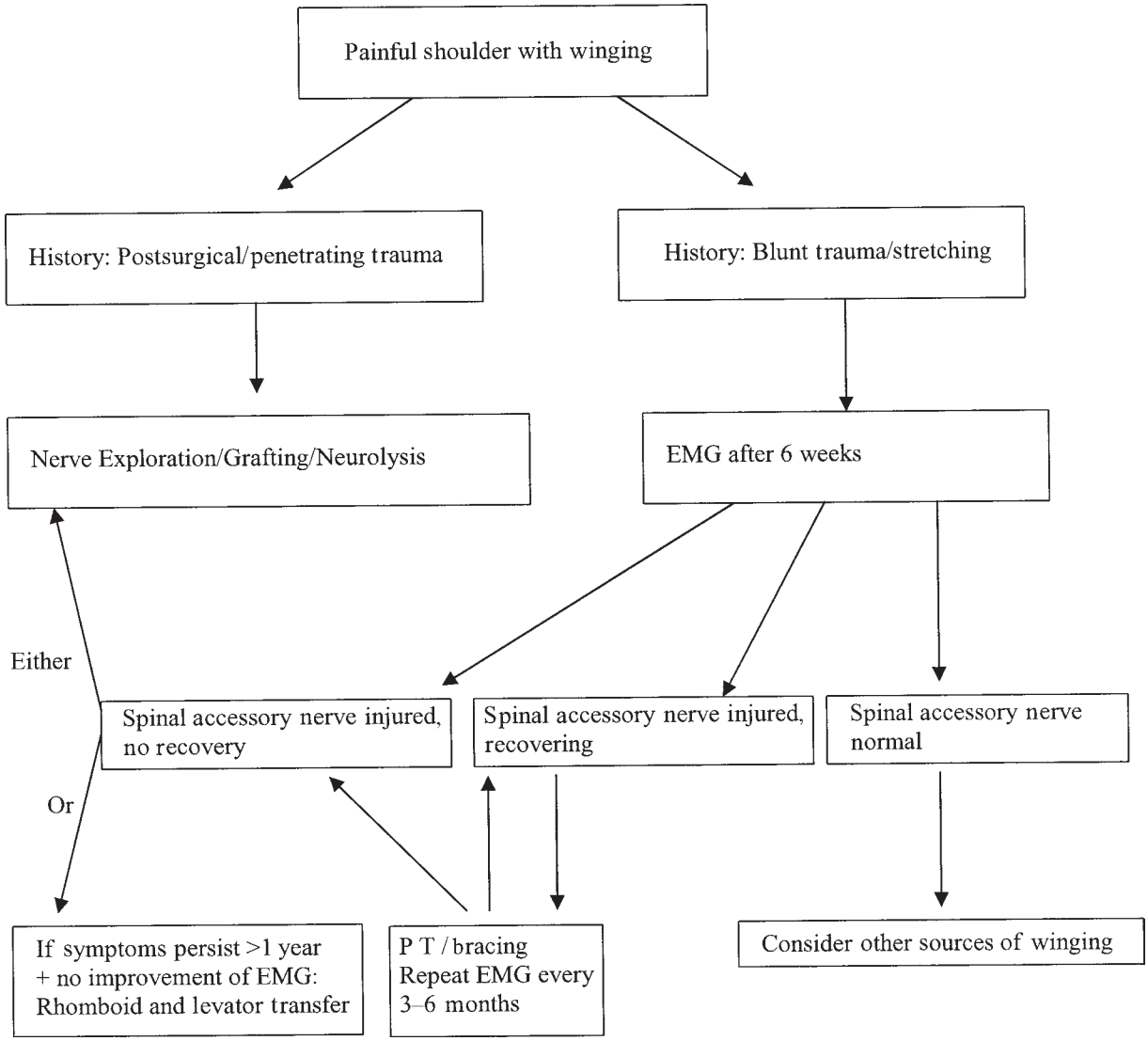


Figure 34-10 Algorithm for treatment of trapezius palsy. EMG, electromyography; PT, physical therapy.

mechanical advantage is improved and winging is eliminated. The surgical technique involves two incisions. The first is along the medial scapular border, and the second is over the spine of the scapula. The levator scapula, rhomboideus minor, and rhomboideus major are detached from the medial scapula taking a small portion of insertional bone. The rhomboideus muscles are advanced laterally under the infraspinatus and are secured with suture, which is passed through drill holes placed 5 cm lateral to the medial border of the scapula. The levator scapula is passed subcutaneously to the second incision and is sutured to the scapular spine through drill holes (see Fig. 34-13).

Postoperative Considerations

Patients are kept in an abduction cast or foam abduction pillow for 4 to 6 weeks and then passive and active range of motion is begun. Resisted exercises should begin after approximately 10 to 12 weeks. Full return to activities as tolerated usually occurs 4 to 6 months after surgery.

Results of Treatment

Bigliani et al. recently reported their results using this procedure on 23 patients with trapezius scapular winging and had 87% excellent and good results.[11] Significant improvement in pain was seen in 91% of these patients and 87% had significant improvement in function.[11]

Complications

Reported complications are rare. Failure occurs in approximately 15% of patients who have poor function, possibly related to poor healing or continued weakness of the trans-

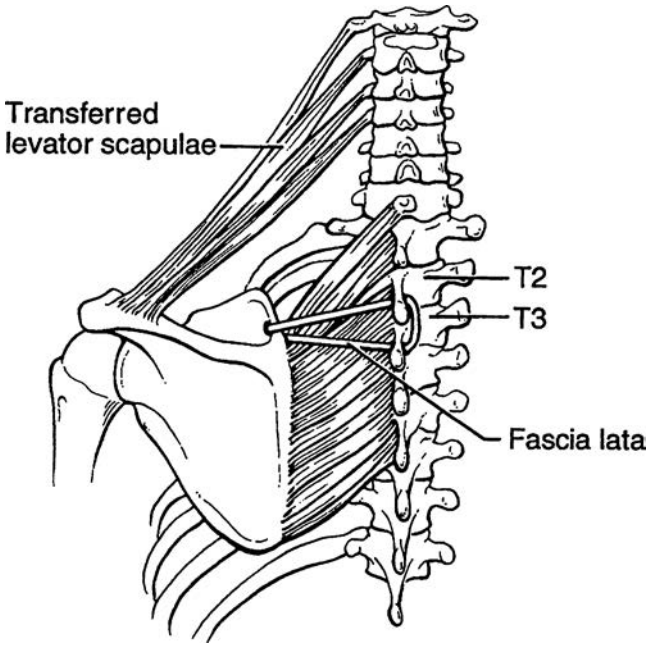


Figure 34-11 Dewar-Harris operation for trapezius palsy. This procedure combines the use of fascial slings to compensate for the middle and lower trapezius, and transfer of the levator scapulae to compensate for the upper trapezius.

ferred muscles. Scapulothoracic crepitus and bursitis may develop after this surgery as well.

Long Thoracic Nerve Injury and Serratus Anterior Palsy

Palsy of the serratus anterior muscle can also cause painful, disabling winging. The long thoracic nerve, which powers

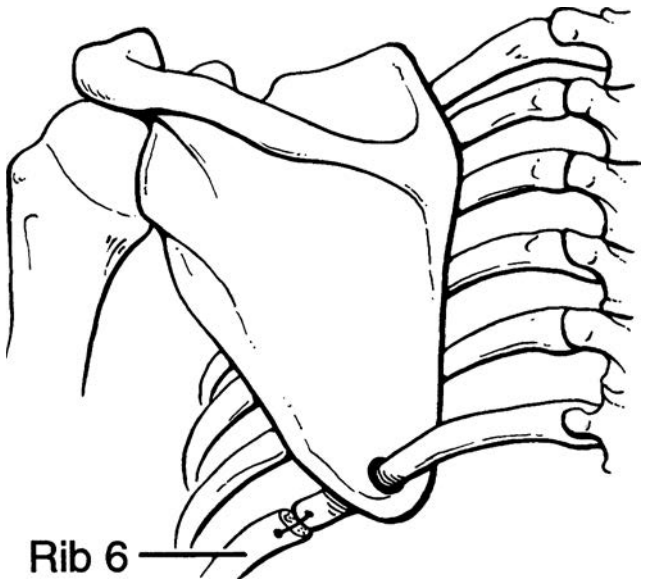


Figure 34-12 Spira's partial fusion for trapezius palsy. In this procedure a hole is made in the inferior angle of the scapula, and the sixth rib is passed through the scapula, stabilizing it.

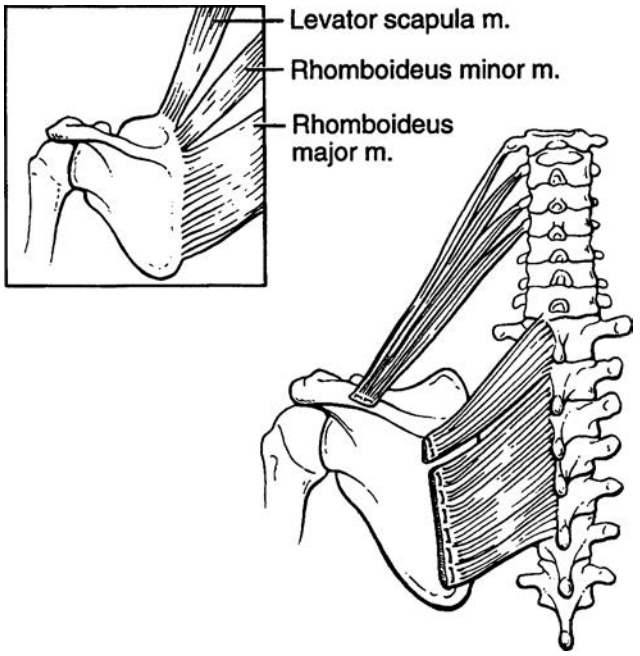


Figure 34-13 Eden-Lange procedure for trapezius palsy. In this procedure, the levator scapula is transferred laterally to function as the upper trapezius, while advancement of the rhomboid major and minor compensate for the loss of the middle and lower trapezius.

the serratus anterior muscle, originates from the ventral rami of the C5, C6, and C7 cervical nerves and travels beneath the brachial plexus and clavicle and over the first rib. It then travels superficially along the lateral aspect of the chest wall, which makes the nerve susceptible to injury (Fig. 34-14). Recently, a fascial band from the inferior aspect of the brachial plexus superior to the middle scalene muscle and inserting on the first rib was found to possibly be a source of nerve compression as the nerve has been shown to "bowstring" across this band with abduction and external rotation.[72]

Signs and Symptoms

Blunt trauma or stretching of this nerve is particularly common in athletics and has been observed in tennis players, golfers, swimmers, gymnasts, soccer players, bowlers, weight lifters, ice hockey players, wrestlers, archers, basketball players, and football players.[48,60,102,179] Repetitive industrial use of the shoulder has also been implicated as a cause of serratus paralysis.[132,163] Penetrating trauma will rarely cause injury to this nerve, yet surgical procedures such as radical mastectomy, first rib resection, and transaxillary sympathectomy have all been identified as potential sources of injury to the long thoracic nerve.[102] This nerve can also be affected by nontraumatic events, including positioning during anesthesia,[132,163] the sequelae of viral illness[45,143] and inoculations,[6] and neuritis affecting the brachial plexus or long thoracic nerve

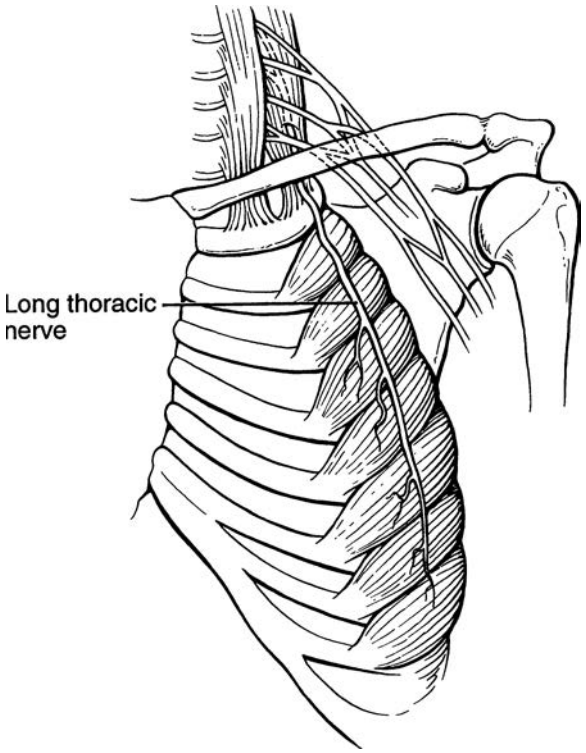


Figure 34-14 Location of the long thoracic nerve. Its superficial location along the chest wall makes it susceptible to injury. (Reprinted with permission from Kuhn JE, Hawkins RJ. Evaluation and treatment of scapular disorders. In: Warner JP, Iannotti JP, Gerber C, eds. *Complex and revision problems in shoulder surgery.* Philadelphia: Lippincott-Raven Publishers, 1997:357–375.)

alone.[78,79,134] Prolonged bed rest has also been reported to trigger a dysfunction of the long thoracic nerve, particularly if the arm is abducted, for example, propping up the head to read.[104,132,140,142] Interestingly, because the long thoracic nerve has a significant C7 component, patients with a C7 radiculopathy may also present with serratus anterior weakness and scapular winging.[110]

Patients with serratus anterior palsies will complain of pain as the other periscapular muscles try to compensate. More severe pain may indicate an acute brachial plexus neuritis[137] or Parsonage-Turner syndrome, which may affect the long thoracic nerve alone.[134] With an injury to the long thoracic nerve and dysfunction of the serratus anterior muscle, the scapula assumes a position of superior elevation and medial translation, and the inferior pole is rotated medially (see Fig. 34-9). The patient will have difficulty with arm elevation above 120 degrees, which will magnify the degree of winging.[51,84] Pain may be increased with this maneuver and when the head is tilted toward the contralateral side.[137]

Diagnosis and Imaging

Electromyography is recommended to confirm the diagnosis and follow the recovery of the injured long thoracic nerve. When requesting an electromyographic analysis, it is important to specify an evaluation of the long thoracic nerve and serratus anterior muscle. Because the majority of long thoracic nerve palsies will recover spontaneously, regular electromyographic examinations at 1- to 3-month intervals have been recommended to follow nerve recovery.[80,104]

Natural History

Most injuries to the long thoracic nerve are neurapraxic injuries from stretching the nerve. Patients may be able to identify the source of the injury, but frequently the winging is noted without a profound injury. The patient loses arm function, feels like the shoulder is weak, and has pain. These injuries typically do recover, although slowly. Most long thoracic nerve injuries recover spontaneously within 1 year,[9,41,47,51,52,59,60,66,78,81,84,104,132,140,142] although maximal recovery may take up to 2 years.[51,57,103]

Treatment Options

Treatment options are offered as an algorithm in Fig. 34-15. Nonoperative treatment should be implemented immediately upon diagnosis and should include range-of-motion exercises to prevent glenohumeral stiffness. Many types of braces and orthotics have been developed, but their use is controversial.[80,116] In general, these braces attempt to hold the scapula against the chest wall, and may have some role if their cumbersome nature is overshadowed by their symptom relief.[52,84,104,192] Some have recommended bracing to limit continued traction on the nerve in hopes of accelerating nerve regeneration.

Surgical Techniques

There are little data in the literature regarding the results of neurolysis, nerve grafting, or repair of an injured long thoracic nerve.[163] Nevertheless, penetrating injuries should undergo nerve exploration and early repair. Neurorrhaphy may be indicated in circumstances when the lesion can be localized.[104] As mentioned above, the lesion is frequently supraclavicular and neurolysis in this region has been performed in a few patients with good success.[38] Many patients with persistent impairment of the serratus anterior are able to compensate and would not elect to have a surgical reconstruction.[104] In patients with symptomatic serratus winging that persists for more than 1 year, surgical intervention may alleviate pain and improve function. Historically, the operations for serratus paralysis can be classified into three types: scapulothoracic fusions[68]; fascial sling suspensions[36,176,189]; and muscle transfers that have utilized pectoralis minor,[22,79,144] pectoralis major,[155] the sternocostal head of pectoralis major,[42,177] the clavicular head of pectoralis major,[43] teres major,[65] the rhomboideus muscles,[71] and combinations.[167,198]

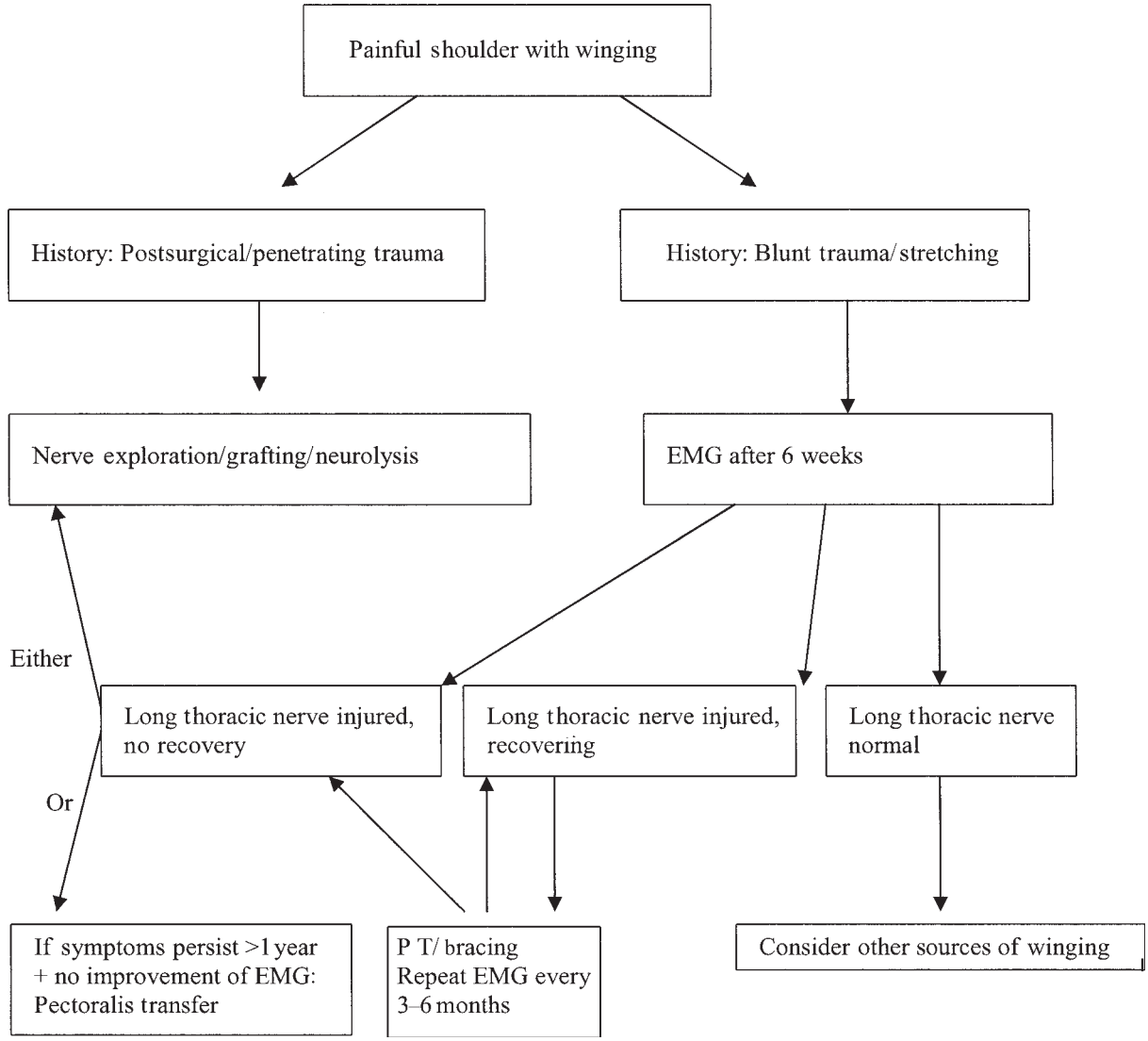


Figure 34-15 Algorithm for treatment of serratus palsy. EMG, electromyography; PT, physical therapy.

Scapulothoracic fusions as a first operation for serratus winging have been discouraged by some,[12,78] primarily for their inherent loss of motion. Pain relief, however, is a reasonable expectation.[68] Complications of scapulothoracic fusions are many and include nonunions and pneumothorax.[68] For these reasons, and because of the limited expectations with regard to motion, scapulothoracic fusions have been generally reserved for salvage operations after failure of other techniques, or for patients with paralysis of other shoulder girdle muscles in addition to the serratus anterior.[80] Another possible indication for primary scapulothoracic fusion for serratus winging is in the laborer who places heavy demand on the shoulder.[103]

Fascial sling suspensions have had their proponents as well.[5,34,36,176,189] However, significant concern exists with regard to fascial sling failure and recurrence of winging.[76] For these reasons, muscle transfers for dynamic scapular stabilization have gained wide acceptance. Although a variety of muscle transfers have been described, transferring the sternocostal head of the pectoralis major with a fascia lata graft extension[112] has become the most popular[47,56,59,80,104,116,129,135,141,171] (Fig. 34-16).

In this technique,[59,112,116,138] the patient is placed in the lateral decubitus position using a bean bag and the involved arm and forequarter are prepared and draped. An incision is made crossing the axilla from the pectoralis major muscle anteriorly to the inferior tip of the scapula. Alternatively, two incisions can be used, with the pectoralis tendon and graft tunneled along the chest wall subcutaneously. The sternocostal head of the pectoralis major is released from its insertion on the bicipital groove of the humerus. A graft of fascia lata measuring 7″ × 2″ is harvested from the ipsilateral

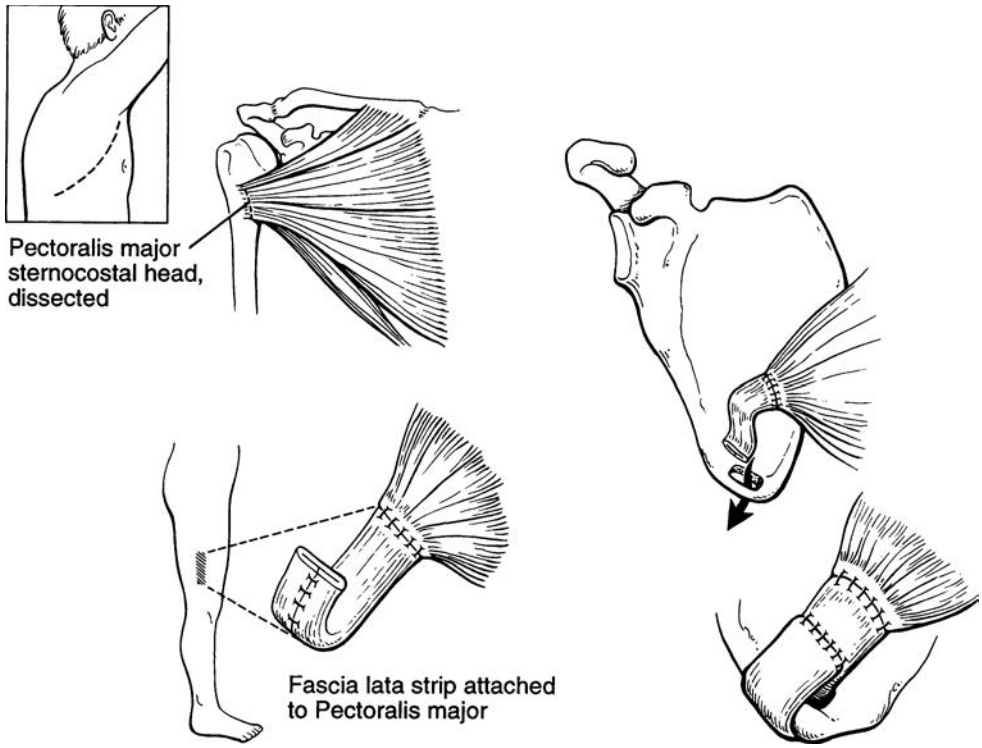


Figure 34-16 Pectoralis major transfer for scapular winging. As described by Marmor and Bechtol,[103] the sternocostal head of the pectoralis major is sutured to a tubularized fascia lata graft and woven through a foramen made in the inferior angle of the scapula. (Reprinted with permission from Kuhn JE, Hawkins RJ. Evaluation and treatment of scapular disorders. In: Warner JP, Iannotti JP, Gerber C, eds. *Complex and revision problems in shoulder surgery.* Philadelphia: Lippincott-Raven Publishers, 1997:357–375.)

leg and sutured into a tube, 7″ long. Alternatively, hamstring tendon may be used. This graft is then sutured to the distal portion of the freed pectoralis tendon. After the inferior border of the scapula has been exposed, a foramen is made in the inferior angle. The graft is inserted through this defect and sutured to itself under moderate tension. It is important to position the graft such that the end of the pectoralis tendon meets the scapula, and the fascia lata graft is used as augmentation. To prevent stretching of the transferred tendon, it has been recommended to have the pectoralis tendon reach the scapula, using augmentation only when necessary.[141] The wounds are closed in layers.

Postoperative Considerations

Postoperatively the arm is placed in a sling or abduction pillow for comfort. Early passive motion is started immediately. Active motion is begun at 6 weeks and strengthening at 12 weeks. Overaggressive therapy may stretch the graft and should be avoided. A scapulothoracic brace may be worn to prevent winging during healing.

Results of Treatment

Although there are few large series in the literature, results using this technique have been encouraging, with 70% to 91% success rates as determined by normal shoulder motion and a significant reduction in pain and winging.[59,80,102,171]

Complications

Surgical complications of this procedure are few but include pneumothorax, and postoperative chest radiographs should always be taken. Late complications include fracture of the inferior border of the scapula or failure of the graft, both of which will manifest as a recurrence of winging.[138] Management of severe complications can be treated with a scapulothoracic fusion.

Rhomboideus Major and Minor Palsy

Weakness of the rhomboid major and rhomboid minor muscles is a rarely identified cause of scapular winging. These muscles receive innervation from the dorsal scapular nerve, which takes its origin from the C5 nerve root. The dorsal scapular nerve passes deep to or through the levator scapulae on its way to the rhomboids. Scapulothoracic winging may result from a C5 radiculopathy or an injury to the dorsal scapular nerve.[152]

Signs and Symptoms

Patients with rhomboid weakness may complain of pain along the medial border of the scapula. The winging produced by rhomboideus palsy at rest is usually minimal, but may appear similar to trapezius winging with the shoulder slightly depressed, the scapula laterally translated, and the inferior angle rotated laterally. In addition, atrophy along the medial border of the scapula may be evident. During arm elevation the inferior angle of the scapula is pulled laterally and downward by the unopposed serratus anterior muscle.[34] With rhomboid weakness, the winging is accentuated by slowly lowering the arm from the forward-elevated position, during which time the inferior angle of the scapula is pulled laterally and dorsally.[152] Patients will have difficulty pushing the elbows backwards against resistance with the hands on the hips.[67]

Diagnosis and Imaging

Electromyography and nerve conduction studies will help differentiate this type of winging from winging caused by other neurologic injury.

Natural History

Unlike other forms of neurapraxic winging, rhomboid winging is not likely going to improve spontaneously. However, the winging is generally not as severe as other forms of winging and may be less symptomatic.

Treatment Options

A treatment algorithm for rhomboid winging is offered in Fig. 34-17. In patients with C5 radiculopathies, addressing

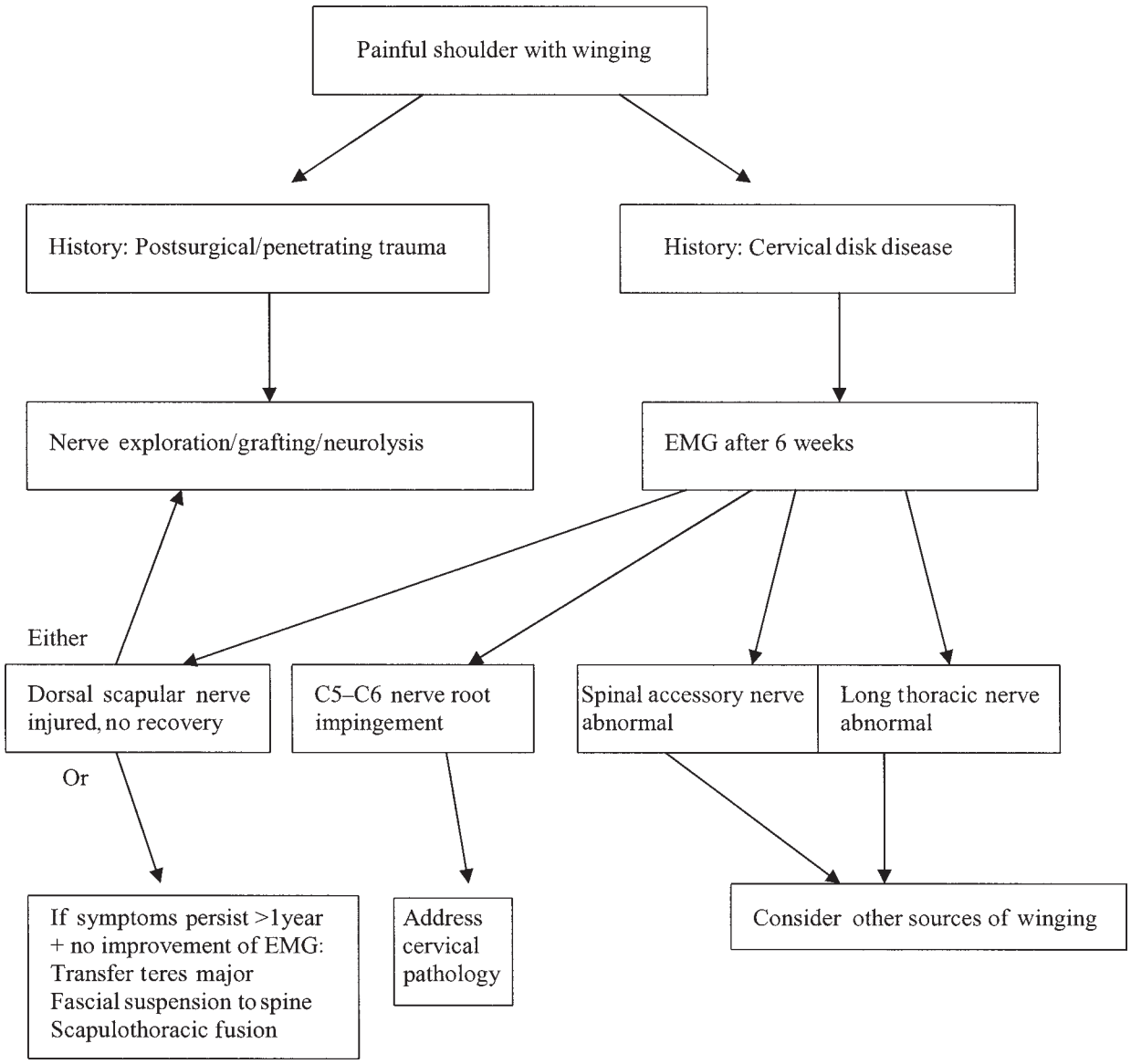


Figure 34-17 Algorithm for treatment of rhomboid palsy. EMG, electromyography.

the source of the radiculopathy may provide some benefit. In general, the treatment of rhomboideus winging consists of trapezius strengthening exercises.

Surgical Techniques

The patient who has significant symptoms and fails nonoperative treatment may be helped by a fascial sling operation as described by Dickson.[34,36] In this operation two fascia lata grafts are tubularized and used to connect the lower vertebral border of the scapula to the spinal muscles and the inferior angle of the scapula to the fibers of the latissimus dorsi. Alternatively, hamstring tendons may be used. This procedure is thought to be useful in stabilizing the scapula and partially arresting the high thoracic scoliosis that may occur with rhomboid and levator scapulae paralysis.[34] There is some concern that fascial sling suspensions may elongate and fail in time.[76] Alternatively, creative muscle transfers may be entertained, such as a transfer of the teres major where the insertion is taken from the humerus, the muscle is flipped on itself, and the insertion is secured to the spinous processes. Finally scapulothoracic fusion may be considered as well.

Postoperative Considerations

The postoperative management of these patients may be difficult. For surgeries designed to stabilize the scapula, either fascial slings or fusions, limiting scapular motion for 8 to 12 weeks is ideal. This can be done with the help of an abduction pillow and/or a scapular brace.

Results of Treatment

There are very few series in the literature to gauge the results of treatment for this type of winging.

Complications

Any surgery around the scapulothoracic articulation, in particular scapulothoracic fusions, is at risk for the development of pneumothoraces. Fascial sling suspensions have been described as having a risk of late failure, after which a fusion may be warranted.

Primary Scapular Winging—Muscle Disorders

Muscle abnormalities that cause winging have included traumatic ruptures and congenital absence of periscapular muscles. In the cases of serratus anterior muscle avulsion, significant trauma has occurred that pulls the muscle insertion from the medial border of the scapula.[50,69,119,132,187] Fiddian and King reported a case of serratus anterior division during thoracotomy, which was a source of symptomatic winging.[48] In these cases, nerve conduction studies are likely to be normal and computed tomography (CT) or magnetic resonance imaging (MRI) should be considered to assist in the diagnosis. Surgical reattachment is recommended in all patients and excellent results can be expected.[50,69,132,187] Advanced age with systemic disease may, however, be a contraindication to surgery.[119] Congenital absence of the serratus anterior,[26,106] the trapezius,[77,88,157,158] and the rhomboideus major and trapezius muscles[159] have all been reported. Patients with these congenital anomalies, however, seem to function very well without treatment.[193]

Fascioscapulohumeral Dystrophy

Fascioscapulohumeral dystrophy is a genetic neuromuscular dystrophy[46] characterized by deletion of the subtelomeric region of chromosome 4q35 and affects the muscles of the face, shoulder girdle, and upper limbs, producing a profound scapular winging of a muscular origin.[83,146]

Signs and Symptoms

A notable feature of fascioscapulohumeral muscular dystrophy is substantial weakness of the shoulder girdle muscles, scapulothoracic instability, and impairment of shoulder flexion and abduction[61]; however, the deltoid seems to be spared. As the scapular stabilizing muscles are affected, patients with this disorder have difficulty with abduction and forward elevation.[61] However, if stability of the scapulothoracic articulation is achieved, the deltoid is able to elevate the arm and improve function.

Diagnosis and Imaging

The diagnosis is clinical and is made with genetic testing.

Natural History

This disorder typically presents in the second decade of life and has a slow progression.[17] However, dysfunction of the upper extremities makes employment and activities of daily living difficult.[188]

Treatment Options

Because this disorder is characterized by multiple muscle involvement, muscle transfers are generally not available, and this form of muscle-origin scapular winging is treated best with a scapulothoracic fusion.[10,18,30,82,89,105]

Surgical Techniques

When performing surgery, it is advisable to use a double lumen endotracheal tube to allow for selective deflation of the ipsilateral lung during wire placement around the ribs.

The patient is positioned prone. The entire arm, scapula thorax, and ipsilateral posterior iliac crest are prepared and draped in the operating field.

An incision is made along the medial border of the scapula from just superior to the scapular spine to the inferior angle. The trapezius muscle is dissected from the spine of the scapula and retracted medially. The rhomboid muscles are dissected from the medial scapula with care to protect the fascial insertional tissue for later reattachment. The medial scapula border can then be retracted laterally and posteriorly to gain access to the posterior thorax.

The subscapularis origin is dissected from the undersurface of the medial border of the scapula approximately one-third of the width of the scapula. The ventral surface of the scapula bone is decorticated with a burr. This should be a minimal decortication to prevent weakening of the scapula and potential later fracture.

At this point the scapula is reduced to the rib cage in the position of interest. For facioscapulohumeral dystrophy, the inferior angle of the scapula should lie over the seventh rib. The vertebral side of the spine of the scapula should lie between the third and fourth rib and should be angled 15 to 20 degrees from the midline.[109] The vertebral border should rest 5 to 7 cm from the spinous processes.[30] This position is thought to prevent neurovascular complications. For other conditions the position may be altered. For example, for problems with associated glenohumeral joint instability, a position of increased angulation of 35 to 40 degrees has been recommended.[97] At this point the ribs corresponding to the decorticated ventral surface of the scapula are identified (typically ribs three to six). The scapula is retracted again, and the ribs are prepared by incising the periosteum and then decorticating lightly with a burr. It is important to remove all soft tissue between the ribs and scapula to allow for bony fusion.

The involved lung is deflated, and cerclage wires (1.5 mm) are passed around the ribs using periosteal dissectors. The location of the wires should correspond to the medial border of the scapula. At this point a one-third semitubular, five- or six-hole, large fragment plate is aligned with the medial border of the scapula on the dorsal side. A 3-mm burr is used to make holes through the scapula at the medial border and the wires are passed through the scapula and through the holes on the plate.

Cancellous bone harvested from the posterior iliac crest is used between the ribs and the scapula, and the wires are then tightened over the plate, compressing the scapula to the ribs. The lung is reinflated, and irrigation is used to search for a pneumothorax. Pneumothorax is very common and a chest tube is placed at this time. The chest tube is also placed to drain any reactive pleural effusion or blood.

Closure is performed in layers. First the rhomboids are reattached to the medial border of the scapula, and then the trapezius may be reattached to the spine. The subcutaneous tissue and skin are closed routinely.

Postoperative Considerations

The patient is placed in a gunslinger-type brace and admitted for observation. A chest radiograph is obtained to evaluate pneumothorax or hemothorax. The chest tube is typically removed after 1 to 2 days.

Patients are discharged with the gunslinger brace. Immobilization is used for 12 weeks. At 12 weeks, physical therapy can begin starting with gentle passive range of motion, emphasizing forward elevation and external rotation. At 16 weeks patients can progress to active range of motion. Strengthening should begin at approximately 18 to 20 weeks after surgery.

Outcomes of Treatment

Humeral function after applying manual compression of the scapula on the chest wall can be useful to predict a successful surgical outcome.[184] Most authors report successful outcomes of long duration[30] with shoulder flexion increasing at least 33 to 56 degrees and abduction increasing at least 25 to 44 degrees.[2,105,178] Although some have performed surgery on both scapulae at the same time,[94] the length of the surgery, the substantial postoperative pain, and the lengthy period of immobilization usually direct the surgeon and patient to having one side fused first and the second side fused at a later date.

Complications

Reported complications include brachial plexopathy,[191] vascular compromise,[109] pleuritic chest pain, pneumothorax, rib stress fractures, and irritation from surgical hardware.[184] Pseudarthrosis is a common complication occurring in approximately 25% of cases.[97]

Primary Scapular Winging—Bony Abnormalities

Bone abnormalities that may present as scapular winging include osteochondromas, the most common tumor of the scapula, which can be a cause of "pseudowinging"[28,32,48,108,135] (Fig. 34-18). Rib osteochondromas may also cause scapular winging.[48] Malunions of fractures of the scapula and clavicle have also been implicated as a source of primary winging of the scapula.[48,118] Because the muscle function would not be expected to be impaired, these patients may not be symptomatic.

Signs and Symptoms

This type of scapular winging is structural and may be associated with scapular crepitus. Patients will have static scapular winging, which may not change with varying the position of the arm.

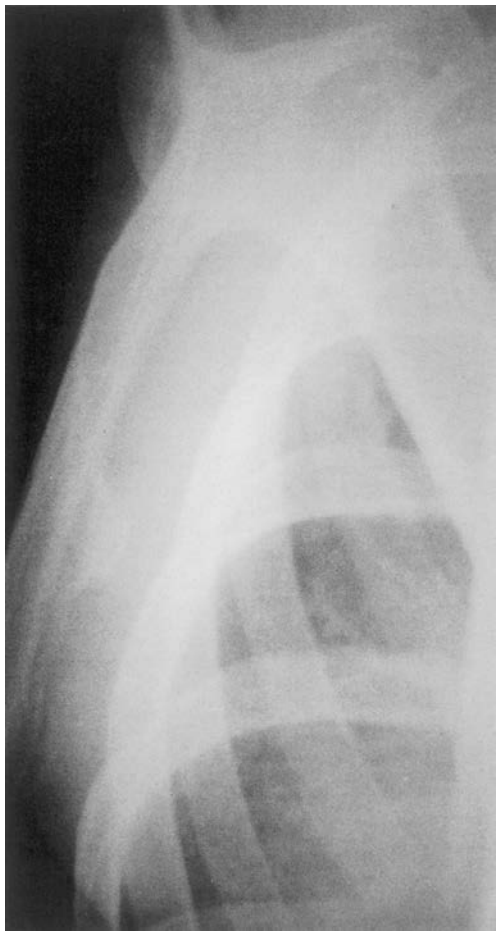


Figure 34-18 Radiograph of a scapular osteochondroma causing scapular winging and crepitus. The patient's symptoms resolved completely with resection of the osteochondroma. (Reprinted with permission from Kuhn JE, Hawkins RJ. Evaluation and treatment of scapular disorders. In: Warner JP, Iannotti JP, Gerber C, eds. *Complex and revision problems in shoulder surgery.* Philadelphia: Lippincott-Raven Publishers, 1997:357–375.)

Diagnosis and Imaging

In this instance, electromyography studies will be normal, and the osteochondroma is identified by radiographs tangential to the plane of the scapula, or computed tomography.

Treatment Options

Winging and scapulothoracic crepitus are alleviated with resection of the bony pathology.

Complications

Few complications of resection of bony pathology of the scapula have been reported.

Primary Scapular Winging—Bursal Disorders

The articulation between the scapula and the thorax has bursae, that, in rare circumstances, may become inflamed. This is one source of scapular crepitance and pain. Winging has been identified in 50% of patients with a snapping scapula and no bony abnormalities.[136] This is presumably related to the subscapular bursitis, and with treatment of the bursitis, either by nonoperative means or surgical bursectomy, the winging resolves.

Secondary Scapular Winging

Secondary scapular winging originates from disorders of the glenohumeral joint, which produce abnormal scapulothoracic dynamics (Fig. 34-19). This phenomenon has not been thoroughly investigated in the literature. A thorough evaluation of the patient with secondary scapular winging usually, but not always, identifies the source of the glenohumeral pathology.[170] The physical examination of every patient with a shoulder condition should include looking for secondary scapular winging at rest with dynamic forward elevation and with resisted forward elevation. A characteristic of secondary scapular winging is a normal electromyographic examination of the long thoracic nerve and serratus anterior, the spinal accessory nerve and trapezius, and the dorsal scapular nerve and rhomboids.

Contractural winging[4] is one example in which contractures about the glenohumeral joint can produce secondary scapular winging. Patients with upper root brachial plexus injuries develop contractures due to unbalanced muscle forces with the humerus abducted and internally rotated relative to the scapula. When the arm is forcibly adducted to the chest wall and externally rotated, the superior corner of the scapula projects away from the chest wall, producing the "scapular sign of Putti,"[156] otherwise known as contractural

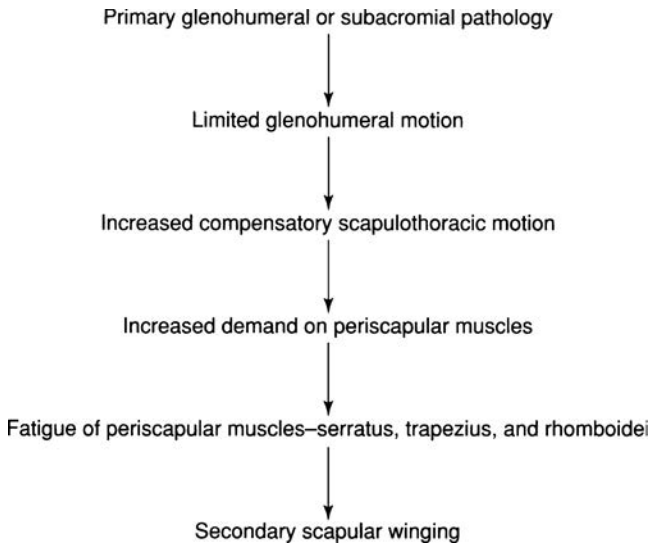


Figure 34-19 Sequence of events leading to secondary scapular winging. (Reprinted with permission from Kuhn JE, Hawkins RJ. Evaluation and treatment of scapular disorders. In: Warner JP, Iannotti JP, Gerber C, eds. *Complex and revision problems in shoulder surgery.* Philadelphia: Lippincott-Raven Publishers, 1997:357–375.)

winging.[4] Contractural winging can also occur with fibrosis of the deltoid muscle in which the winging decreases with arm elevation and increases with lowering of the arm.[175] This condition is thought to be either congenital[148,190] or related to a history of injections into the deltoid muscle[62,124] and is almost always associated with winging of the scapula.[124]

Other more common disorders involving the glenohumeral joint can be a source of secondary scapular winging. This is thought to occur by reflex muscle spasm provoked by some painful condition in the glenohumeral or subacromial areas.[175] This has been associated with rotator cuff tears,[175] nonunion of acromion fractures,[60] malunion of clavicle fractures,[48] fractures of the glenoid,[48] avascular necrosis of the humeral head,[60] acromegalic arthropathy of the shoulder,[48] acromioclavicular joint disorders,[48,170] and shoulder instability.[48,49,154,170] In our practice we have observed secondary scapular winging in patients with adhesive capsulitis, the impingement syndrome, and anterior shoulder instability, and in throwing athletes with secondary impingement due to subtle shoulder instability. We suspect that patients with painful shoulders reflexively limit glenohumeral motion. This forces the periscapular muscles to work in excess as scapulothoracic motion must increase to compensate for the limited glenohumeral motion. With fatigue of the periscapular muscles, particularly the serratus anterior, trapezius, and rhomboids, scapular winging occurs (see Fig. 34-19). As has been shown,[170,175] treatment of the primary glenohumeral pathology will alleviate the scapular winging, and conversely the scapular winging is unlikely to improve until the primary source of pathology is addressed.[170] A scapular rehabilitation program should be added to the treatment of the primary glenohumeral pathology to facilitate recovery in every patient with secondary scapular winging.[127,133]

Voluntary Scapular Winging

Voluntary scapular winging is also very rare.[48,60,149,170] Rowe reported on four patients with voluntary scapular winging. These patients were assured and instructed on the normal muscle firing patterns of the shoulder with "instructions not to tighten or contract their shoulder muscles when elevating the arm."[149] All four patients recovered after this coaching. Gregg et al. described asymptomatic bilateral voluntary scapular winging in an orthopaedic resident.[60] It is important to appreciate that patients with voluntary scapular winging who seek medical attention, like patients with voluntary subluxation of the shoulder, may have unaddressed psychologic issues complicating their care.[150]

SCAPULAR DYSKINESIA

Although static scapulothoracic abnormalities have been recognized in the past, dynamic scapulothoracic dyskinesia was an underappreciated phenomenon until recently. Investigations in normal and athletic patients have demonstrated significant alterations in the dynamic motion of the scapulothoracic articulation when accompanied by shoulder pathology. It is uncertain if the scapular dyskinesis is a primary problem or a secondary problem that develops as a consequence of other glenohumeral joint pathology. A variety of pathologies have been associated with scapular dyskinesis, as scapular motion also determines the location of the glenohumeral joint in space and is important not only to optimize glenoid placement relative to the humeral head to maintain glenohumeral joint stability, but also to elevate the acromion to allow motion without impingement.

Rotator Cuff Pathology

In their study evaluating scapulothoracic motion using Moire topographic analysis, Warner et al. found static scapulothoracic asymmetry present in 32% of patients with glenohumeral instability and 57% of patients with subacromial impingement.[186] Dynamic testing was more sensitive with asymmetric scapulothoracic motion in 64% of patients with glenohumeral instability and all patients with impingement.[186]

The pectoralis minor tendon is thought to be contracted, and painful, which has been shown to influence scapulothoracic kinematics and produce subacromial impingement.[16] Somewhat related, thoracic and scapular posture has profound influences on shoulder range of motion and the range of shoulder motion before impingement symptoms develop.[107]

Glenohumeral Instability

As mentioned above, in their study evaluating scapulothoracic motion using Moire topographic analysis, Warner et al. found static scapulothoracic asymmetry present in 32% of patients with glenohumeral instability.[186] Scapular position seems to be altered in patients with multidirectional instability, which is thought to be causative of failure of humeral head centering.[181]

The Dysfunctional Athlete's Shoulder

Throwing athletes with anterior shoulder instability have altered scapulothoracic mechanics, with the serratus anterior demonstrating a marked reduction in activity during throwing.[55] A consistent firing pattern of the scapular stabilizing muscles has been identified by electromyographic studies in the overhand athlete.[37,93] These studies have determined that throwing and other overhand athletic motions are dependent upon synchronized, coordinated scapulothoracic motion. A coordinated firing pattern of the posterior scapular stabilizing muscles helps to stabilize

the shoulder joint distributing the substantial forces in throwing to the thorax.[93] Alterations in this coordinated activity are thought to uncouple the force relationships required for throwing and lead to decreased efficiency and/or an increased risk for injury. The position of the scapula during the throwing motion will determine the muscle length–tension relationships and allow for maximum muscle force generation and efficiency.[90] Finally, periscapular muscles may be important in providing power and accepting eccentric load, as the scapula is maximally retracted during windup and cocking and maximally protracted during acceleration and follow-through.[55,90,93] Restricted scapulothoracic retraction may limit the power stored in the windup and cocking phases of throwing. In addition, limited protraction may cause the eccentric loading to be transferred to the posterior capsule and posterior rotator cuff tendons of the glenohumeral joint during the follow-through phase of overhead throwing. These excessive repetitive stresses may lead to rotator cuff injury or glenohumeral instability. Overhand throwing athletes often present with abnormal scapular function in the form of winging, poor retraction, poor protraction, poor positioning, or some other form of dyskinesia.

Myers and colleagues studied this concept by measuring scapulothoracic motion in a population of throwing athletes and compared this to scapulothoracic motion in a control population.[128] They showed that throwing athletes demonstrated significantly increased upward rotation, internal rotation, and retraction of the scapula during humeral elevation, implying that throwing athletes may develop these adaptations for more efficient performance of the throwing motion. Su et al. have demonstrated that scapular kinematics may be altered in symptomatic swimmers, an effect that is magnified with fatigue associated with a practice.[173]

Signs and Symptoms

Patients with scapulothoracic dyskinesis will not typically direct the physician toward the scapula, and will complain of pains in the glenohumeral joint. Inspection of the scapulae from the back will demonstrate asymmetry at rest, with the affected shoulder frequently depressed and the scapula protracted and tilted forward. Mild scapular winging may be present with the posterior angle and the medial border of the scapula prominent. Patients will frequently have pain to palpation at the medial coracoid, the insertion of the pectoralis minor. Asking the patient to elevate the arm in the frontal plane and in the scapula plane will reveal asymmetry in scapulothoracic motion. In the presence of rotator cuff pathology, this may be related to decreased firing of the middle and lower trapezius.[29]

Kibler has described the scapular lateral slide test to evaluate patients with abnormal scapulothoracic function.[90] This test evaluates the patient's ability to stabilize the medial border of the scapula during different positions and loads. To perform this test, the arms are held in three different positions and the distance between the medial border of the scapula and the thoracic spinous processes is measured bilaterally. These positions include (a) relative rest in neutral rotation, (b) the hands on the hips with the thumbs pointing posteriorly, and (c) the arms abducted to 90 degrees with maximal internal humeral rotation. In the asymptomatic athlete, symmetry is the general rule, with less than 1 cm side-to-side difference. In symptomatic throwers, more than 1 cm side-to-side difference in position b or c is statistically associated with pain and decreased function.[91]

The clinical implications of this abnormal lateral protraction of the scapula include increased anteversion of the glenoid, opening the anterior half of the glenohumeral articulation. This may create a situation predisposing the patient to increased instability and glenoid labral damage.[90,153] Loss of retraction may prevent optimal energy storage during the windup and cocking phases of throwing, with reduced power. Finally, abnormal scapulothoracic dynamics may limit acromial elevation, producing rotator cuff impingement (see Fig. 34-3).

Burkhart et al. recently described a condition known as the SICK scapula.[19] The acronym SICK stands for Scapula malposition, Inferior medial border prominence, Coracoid pain and malposition, and dysKinesis of scapular movement. The scapula assumes an abnormal position at rest, characterized by a position that is inferior, protracted, and tilted anteriorly. Tenderness is typically found on the medial edge of the coracoid, and the pectoralis minor is thought to be in spasm. The authors recognized this pattern in throwing athletes with shoulder pain.

Diagnosis and Imaging

The diagnosis of scapular dyskinesis is made clinically during the physical examination. While examining patients with shoulder complaints, the back of the shoulder should be inspected at rest and with elevation of the arm. Pain in the rotator cuff can frequently be ameliorated using the scapular assistance test, where the examiner supports the medial border of the scapula and assists its rotation and protraction during arm elevation.[92]

Natural History

As the concept of scapular dyskinesis is relatively new, there are little data on the natural history if left untreated. Although the association between scapular dyskinesis and glenohumeral joint pathology is apparent, it is unclear if the scapular dyskinesis is the primary problem creating

glenohumeral pathology, or if the glenohumeral pathology leads to dyskinesis of the scapula.

Treatment Options

When evaluating patients with shoulder disorders for rehabilitation, it is important to consider and treat abnormal scapulothoracic function, as the scapula is an essential component to most upper-extremity activities. In treating these disorders, rehabilitation begins at the base of the kinetic chain, the legs, hips, and back, and moves distally to the shoulder when the foundation is functioning adequately.[90] Periscapular muscle-strengthening exercises should be an integral component to the complete shoulder rehabilitation program. Strengthening exercises for the rotator cuff should generally be avoided until the scapulothoracic articulation is functioning normally. Kinetic chain-based rehabilitation programs have been recommended,[92,151] as many of the patients with scapulothoracic kinematic abnormalities will have weakness in the core stabilizers of the trunk.

In general, treatment for athletes is nonoperative and requires postural exercises designed to prevent sloping of the shoulders, massage and stretching for a tight pectoralis minor, and hip- and trunk-strengthening exercises.[19,151] Despite these recommendations, there are no series in the literature evaluating the treatment or complications of treatment for scapular dyskinesis. Clearly more work is needed to gain a complete understanding of scapulothoracic problems in the athlete.

SCAPULOTHORACIC CREPITUS

Symptomatic scapulothoracic crepitus has been given a variety of names through the years, including the snapping scapula,[121] the washboard syndrome,[26] the scapulothoracic syndrome,[126] the rolling scapula,[24] the grating scapula,[129] and the scapulocostal syndrome.[161] While Codman himself stated that he was able to make his own scapula "sound about the room without the slightest pain,"[25] Boinet[15] was the first to describe this disorder in 1867. Thirty-seven years later, Mauclaire[114] classified scapulothoracic crepitus into three groups: *froissement* was described as a gentle friction sound and was thought to be physiologic, *frottement* was a louder sound with grating and was usually pathologic, and *craquement* was a loud snapping sound and was always pathologic. These scapular noises are thought to occur from two sources, either anatomic changes in the tissue interposed between the scapula and the chest wall or an incongruence in the scapulothoracic articulation (Table 34-4). Extrapolating from Milch,[121] *frottement* may suggest soft tissue pathology or bursitis, while *craquement* may suggest bony pathology as the source of symptomatic scapulothoracic crepitus. These noises are amplified by the air-filled thoracic cavity, which acts as a resonance chamber, much like a string instrument.[7]

There are a number of described abnormal structures that lay between the scapula and the chest wall that give rise to scapulothoracic crepitus. Pathologic conditions affecting muscle in the scapulothoracic articulation include atrophied muscle,[121] fibrotic muscle,[121,122,187] and anomalous muscle insertions.[166]

The most common bony pathology in the scapulothoracic space that may give rise to scapulothoracic crepitus is the osteochondroma, arising either from the ribs[33] or the scapula[122,123,135] (see Fig. 34-18). Malunited fractures of the ribs or scapula are also capable of creating painful crepitus.[121,122,168] Abnormalities of the superomedial angle of the scapula, including a hooked superomedial angle[123,145] and a Luschka's tubercle (which originally was described as an osteochondroma, but has subsequently come to mean any prominence of bone at the superomedial angle[121,168,183]), have also been implicated as sources for scapulothoracic crepitus. Others[7,148,172] implicate reactive spurs of bone that are created by the microtrauma of chronic, repeated periscapular muscle avulsions.

Certainly, any bony pathology that causes scapulothoracic crepitus is capable of forming a reactive bursa around the

TABLE 34-4
CAUSES OF SCAPULOTHORACIC CREPITUS

Interposed Tissue

MUSCLE
- Atrophy[91,112]
- Fibrosis[112,113,172]
- Anatomic variation[91,160]

BONE
- Rib osteochondroma[26]
- Scapular osteochondroma[113,114,125]
- Rib fracture[112,113]
- Scapular fracture[162]
- Hooked superomedial angle of scapula[112,135]
- Luschka's tubercle[112,173]
- Reactive bone spurs from muscle avulsion[5,165]

OTHER SOFT TISSUE
- Bursitis[2,21,107,108]
- Tuberculosis[112]
- Syphilitic lues[112]

Abnormalities in Scapulothoracic Congruence

SCOLIOSIS[52,172]

THORACIC KYPHOSIS[19]

A variety of structures interposed between the scapula and thorax may cause crepitus. Similarly, spinal curvatures can cause crepitus by altering the scapulothoracic congruence. (Reprinted with permission from Kuhn JE, Hawkins RJ. Evaluation and treatment of scapular disorders. In: Warner JP, Iannotti JP, Gerber C, eds. *Complex and revision problems in shoulder surgery.* Philadelphia: Lippincott-Raven Publishers, 1997:357–375.)

area of pathology.[31,160] In fact, at the time of resection of bony pathology, a bursa is frequently seen. Bursae can become inflamed and painful in the absence of bony pathology, and may become a source of crepitus on their own right.

Other soft tissue pathology that has been implicated in scapulothoracic crepitus includes tuberculosis lesions in the scapulothoracic region and syphilitic lues.[121] Abnormalities in congruence of the scapulothoracic articulation are the other source of scapulothoracic crepitus. Both scoliosis[58,180] and thoracic kyphosis[24] have been implicated as sources of scapulothoracic crepitus.

Signs and Symptoms

The patient with symptomatic scapulothoracic crepitus may be able to identify the location of the crepitus. A history of overhead activity such as sporting activities[136] or paper hanging may be present.[24] Some suspect there is a familial tendency toward developing symptoms.[24] Patients may relate a history of trauma that precipitates symptoms,[3] and scapulothoracic crepitus may be bilateral in some patients.[23] Upon inspection of the scapula, fullness or winging may suggest a space-occupying lesion in the scapulothoracic space. Palpation or auscultation while the shoulder goes through a range of motion may help to identify the source of the periscapular crepitus.[3,23] A palpable mass, crepitus, prominence at rest, and normal scapulothoracic motion help delineate scapular winging due to a physical mass from neurologic scapular winging. In patients with scapulothoracic crepitus the neuromuscular examination is frequently normal, although some patients may have symptoms of thoracic outlet syndrome with scapulothoracic crepitus.[194]

Diagnosis and Imaging

Supplemental radiographs, which include tangential views of the lateral scapula, CT or MRI scans, may be helpful in identifying anatomic pathology.

Natural History

It is important to realize that scapulothoracic crepitus is not necessarily a pathologic condition. Scapular crepitus has been found in 35% of normal asymptomatic people.[63] As a result of this, patients with hidden agendas or psychiatric conditions may not respond to treatment as well as other patients. However, if the scapulothoracic crepitus is truly associated with pain, winging, or other disorders of the scapulothoracic articulation, then the crepitus is considered to be pathologic.

Treatment Options

A treatment algorithm is offered in Fig. 34-20. Patients with clearly defined bony pathology such as an osteochondroma are unlikely to improve with conservative treatment.[121] Resection of the bony pathology is usually necessary to alleviate symptoms with a high likelihood of success.[122,125,135] Other patients should undergo a trial of conservative treatment, with surgery reserved for those who fail.[111] Conservative treatment seems to be most beneficial if soft tissue disorders are the source of scapulothoracic crepitus.[23,121] These include postural exercises designed to prevent sloping of the shoulders.[23,120] A figure-eight harness may be a useful tool to remind patients to maintain upright posture. Exercises to strengthen periscapular muscles are also thought to be important.[23,121,136] Systemic nonsteroidal antiinflammatory drugs as well as local modalities, such as heat, massage, phonophoresis and ultrasound, and the application of ethyl chloride to trigger points, may also prove useful.[23,121,136] Injections of local anesthetics and corticosteroids into the painful area has also been recommended.[20,24,116,121,136] Caution must be used as there is a risk for creating a pneumothorax.[20] Using these means, most patients are expected to improve significantly[116,136]; however, for those who fail, a number of operations have been described.

Muscle plasty operations include those described by Mauclaire, who reflected a flap of the rhomboids or trapezius and sutured it to the undersurface of the scapula.[114] This is thought to be inadequate, however, because the muscle flap may atrophy with time and symptoms can recur.[121] Rockwood has excised a rhomboid muscle avulsion flap with the elimination of snapping and pain.[20] Partial scapulectomy has also been popular as a means of treating symptomatic scapulothoracic crepitus, including resection of the medial border of the scapula[21] or the superomedial angle.[3,24,96,121,145,172,193]

Surgical Techniques

The surgical technique for the resection of the superomedial border of the scapula begins with the patient in the prone position (Fig. 34-21). An incision based over the medial spine of the scapula is made and the soft tissue dissected down to the spine of the scapula. The periosteum over the spine is incised and a plane is developed between the superficial trapezius and the underlying scapula. Next, the supraspinatus, rhomboids, and levator scapulae muscles are dissected free of the scapula in a subperiosteal plane, starting at the spine of the scapula. The superomedial angle of the scapula is resected with an oscillating saw. Caution is warranted as the resection is carried laterally to avoid injury to the dorsal scapular artery and the suprascapular nerve in the suprascapular notch. After resecting the bone, the reflected muscles fall back into place, and the periosteum is reapproximated back to the spine of the scapula using suture through drill holes.

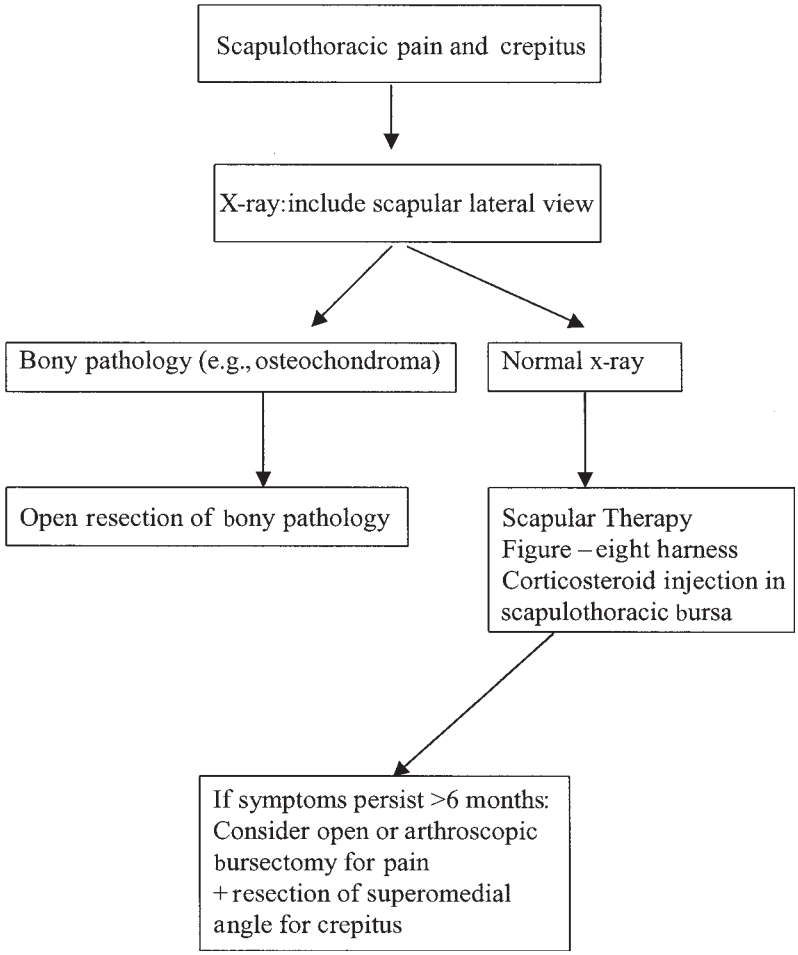


Figure 34-20 Algorithm for treatment of scapulothoracic crepitus.

Postoperative Considerations

Postoperatively, the patient is placed in a sling and begins passive motion immediately. Active motion is begun at 8 weeks, and resistance exercises follow at 12 weeks.

Results of Treatment

Although some authors report mixed results after partial scapulectomy for symptomatic scapulothoracic crepitus,[20] others suggest that this is an excellent option.[24,96,121,145] However most of these reports are case reports or very small series. Arntz and Matsen[3] describe their results after a resection of the superomedial border of the scapula in 14 shoulders in 12 patients with 42 months follow-up. In this relatively large series, 86% of patients obtained complete relief of pain and crepitus. It is important to note that the bone resected appears normal and shows no pathology, which has prompted some to perform bursectomies and avoid a partial scapulectomy.[117,162]

Complications

Complications associated with partial scapulectomy include pneumothorax, postoperative hematoma, and, in younger patients, regrowth of bone. However, this rarely produces symptoms.

SCAPULOTHORACIC BURSITIS

Codman, in his classic text, *The Shoulder,* was clearly one of the first physicians with an interest in scapulothoracic bursitis. He described the bursa of the scapulothoracic articulation and identified and named those bursae in the superomedial border of the scapula that he thought were the cause of symptomatic scapulothoracic bursitis.[25]

Two major or anatomic bursae and four minor or adventitial bursae have been described for the scapulothoracic articulation (Table 34-2, see Fig. 34-7). The first major bursa is found in the space between the serratus anterior muscle and the chest wall. The second major bursa is located between the subscapularis and the serratus anterior muscles.[23,95] These bursae are easily and reproducibly found.[95]

Clinical scapulothoracic bursitis seems to affect two areas of the scapulothoracic articulation: the superomedial angle and the inferior angle of the scapula. When symptomatic, these areas tend to develop inflamed bursae; however, these bursae are not reliably found, and in fact may

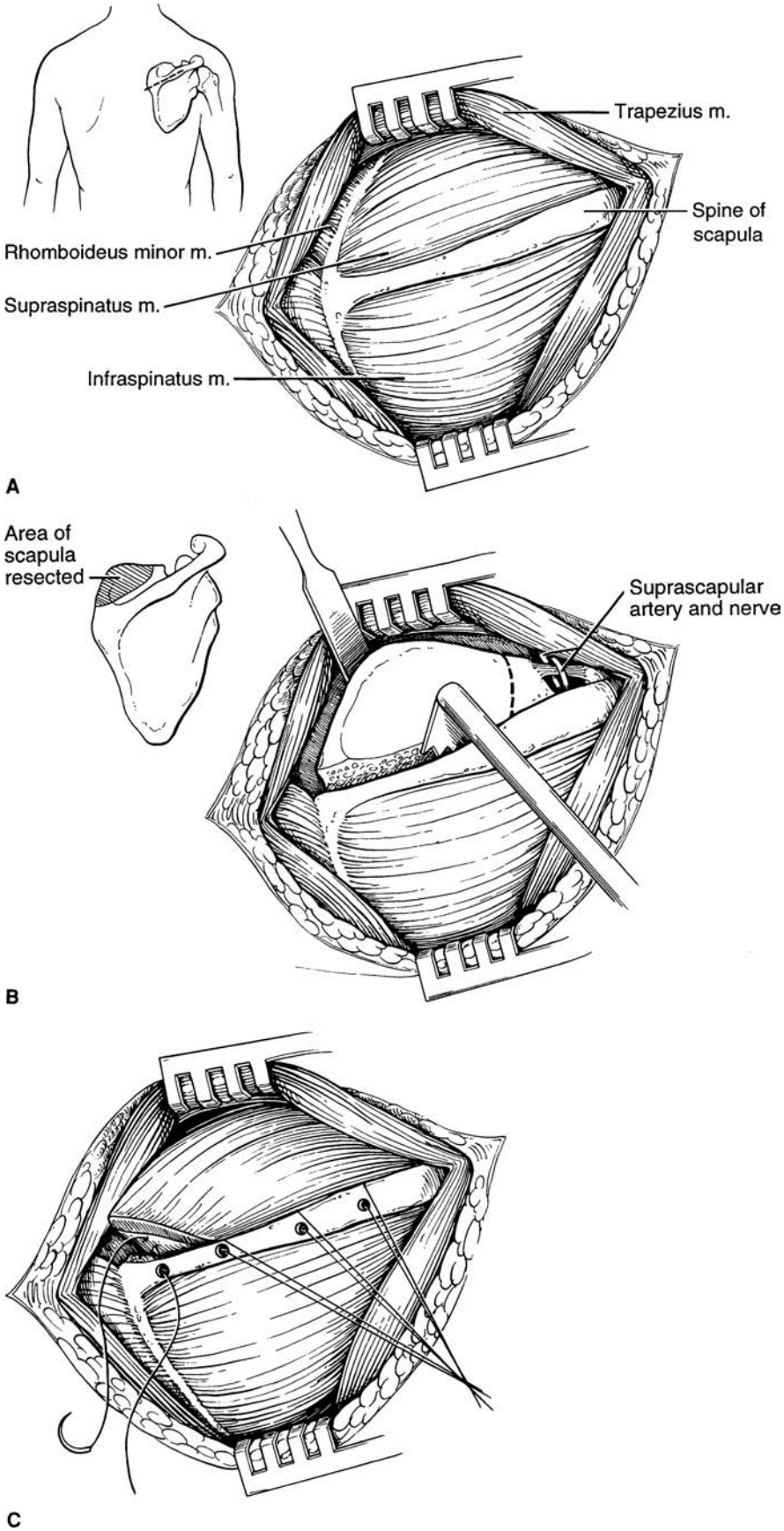


Figure 34-21 Technique for resection of the superomedial border of the scapula for symptomatic scapulothoracic crepitus. **(A)** The trapezius is elevated from the spine of the scapula. **(B)** The supraspinatus, rhomboids, and serratus are elevated in a subperiosteal plane from the medial border, and the superomedial scapula is resected while protecting the suprascapular nerve and artery. **(C)** The supraspinatus is sutured back to the spine of the scapula. (Reprinted with permission from Kuhn JE, Hawkins RJ. Evaluation and treatment of scapular disorders. In: Warner JP, Iannotti JP, Gerber C, eds. *Complex and revision problems in shoulder surgery.* Philadelphia: Lippincott-Raven Publishers, 1997:357–375.)

be adventitious.[24,95,136] With regard to the inferior angle of the scapula, most authors agree that the affected bursa lies between the serratus anterior muscle and the chest wall.[25,121,162] This bursa has been given many names, including the infraserratus bursa[25] and the bursa mucosa.[121,182] The second site of pathology lies at the superomedial angle of the scapula. Here Codman[25] believed this bursa is also an infraserratus bursa, lying between the upper and anterior portion of the scapula and the back of the first three ribs. Von Gruber,[182] on the other hand, identified a bursa in this region between the subscapularis and the serratus anticus muscles, which he called the bursa mucosa angulae superioris scapulae.

A third minor or adventitial bursa, which Codman believed was the site of painful crepitus in scapulothoracic crepitus, was called the trapezoid bursa and is found over the triangular surface at the medial base of the spine of the scapula under the trapezius muscle.[25] Some believe that these minor bursae are adventitial and develop in response to abnormal pathomechanics of the scapulothoracic articulation.[24,95,136] It would not be surprising, then, to find these bursae inconsistently or in different soft tissue planes.

Signs and Symptoms

Scapulothoracic bursitis may accompany painful scapular crepitus or may exist as a separate entity. Patients generally complain of pain with activity and may have audible and palpable crepitus of the scapulothoracic articulation. Usually the scapular crepitus associated with bursitis is of a much lesser quality and nature than that described with bony pathology. Patients relate a history of trauma[3,117] or overuse due to sport or work,[116,117,162] which produces repetitive or constant movement of the scapula on the posterior thorax. This may irritate soft tissues until a chronic bursitis and inflammation develops. The bursa then undergoes scarring and fibrosis, with crepitus and pain to follow.

Diagnosis and Imaging

Unless bony pathology exists, radiographs will be of little help. In severe cases, MRI will demonstrate fluid-filled regions, typically between the serratus anterior and chest wall, with slightly high signal on T1 and high signal on T2 images.[87] However, in many patients, the MRI may appear normal.

Natural History

It is important to note that scapulothoracic crepitus may not be pathologic and may not be associated with pain; however, pain in the periscapular region may be caused by scapulothoracic bursitis, even without accompanying crepitus. The symptom severity seems to be activity related. There are few reports in the literature describing the natural history of untreated scapulothoracic bursitis.

Treatment Options

The initial treatment of scapulothoracic bursitis regardless of its location is conservative, beginning with rest, analgesics, and nonsteroidal antiinflammatory drugs. Physical therapy to improve posture, heat, and local steroid injections has also been recommended.[116,162] Efforts to strengthen periscapular muscles and stretching are frequently added.[116,162] For patients who continue to have symptoms despite conservative treatment, surgery may be beneficial.

Open Surgical Techniques

Sisto and Jobe[162] described an open procedure for resecting a bursa at the inferior angle of the scapula in four Major League baseball pitchers. All pitchers had pain during the early and late cocking phases as well as during acceleration, and could no longer pitch (Fig. 34-22). Only one of the four patients presented with scapulothoracic crepitus, but all had a palpable bursal sac ranging in size from 1 to 2 cm, best seen with the arm abducted to 60 degrees and elevated forward 30 degrees. All four pitchers failed conservative therapy and underwent a bursal excision via an oblique incision just distal to the inferior angle of the scapula. The trapezius muscle and then the latissimus dorsi muscle were split in line with their fibers, exposing the bursa. The bursa was sharply excised and any osteophytes on the inferior pole of the scapula or ribs were removed. The wounds were closed routinely over a drain, and a compression dressing was applied. Physical therapy stressing motion was begun after 1 week and progressed to allow gentle throwing at 6 weeks. This progressed as symptoms permitted return to full-speed throwing. After this procedure, all four pitchers were able to return to their former level of pitching.

Similarly, McCluskey and Bigliani[116,117] performed an open excision of a symptomatic superomedial scapulothoracic bursa in nine patients noting a thickened, abnormal bursa between the serratus anterior and the chest wall at the time of surgery. Their surgical technique involved making a vertical incision medial to the vertebral border of the scapula. The trapezius is dissected free and a subperiosteal dissection is used to free the levator scapulae and rhomboids from the medial border of the scapula. A plane is developed between the serratus anterior and the chest wall. The thickened bursa is resected and any bony projections removed. The medial periscapular muscles and trapezius are reapproximated to the scapula. The skin is closed in a routine fashion.

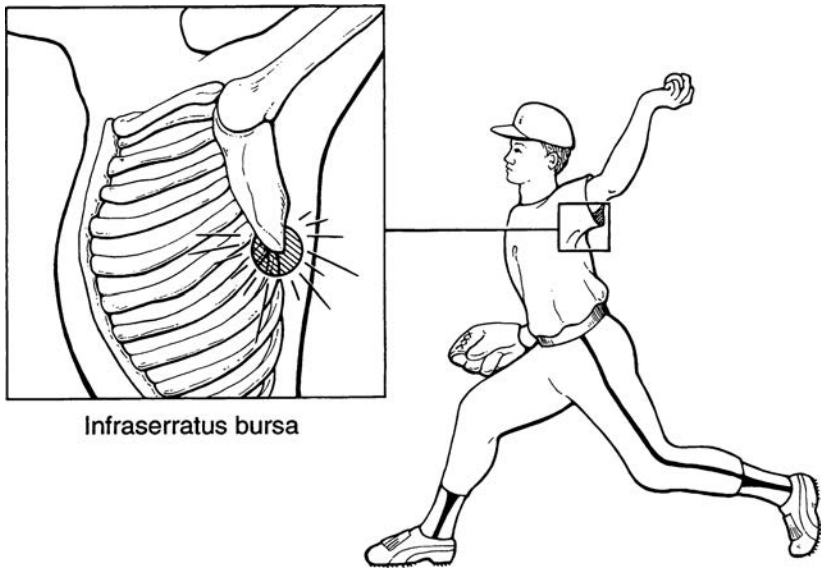


Figure 34-22 Bursae of the inferior angle of the scapula. This bursa has been described in baseball pitchers, and its removal has allowed a return to pitching. (Reprinted with permission from Kuhn JE, Hawkins RJ. Evaluation and treatment of scapular disorders. In: Warner JP, Iannotti JP, Gerber C, eds. *Complex and revision problems in shoulder surgery.* Philadelphia: Lippincott-Raven Publishers, 1997:357–375.)

Endoscopic Surgical Technique

Resection of the symptomatic scapulothoracic bursa has been performed endoscopically as well.[13,23,54,95,113] Ciullo and Jones [23] have the largest endoscopic series to date with 13 patients who underwent subscapular endoscopy after failing a conservative treatment program for symptomatic scapulothoracic bursitis. Débridement was performed for fibrous adhesions found in the bursa between the subscapularis and serratus muscles as well as the bursa between the serratus and chest wall. In addition, débridement or scapuloplasty of changes at the superomedial angle or inferior angle were performed. All 13 patients returned to their preinjury activity level, except for physician-imposed restrictions in a few patients, limiting the assembly line use of vibrating tools.[23]

The technique for scapulothoracic endoscopy has been described by Matthews and colleagues.[113] Patients can be placed in the prone or lateral position; however, the lateral position is preferred as it allows for arthroscopic evaluation of the glenohumeral joint and the subacromial space. In addition, if the arm is extended and maximally internally rotated, the scapula will fall away from the thorax, improving access to the bursae.

Three portals are used, which are placed at least 2 cm from the medial border of the scapula in the region between the scapular spine and the inferior angle. For the middle portal, a spinal needle is inserted into the bursa between the serratus anterior and the chest wall. This needle should be inserted midway between the scapular spine and the inferior angle, at least three fingerbreadths medial to the medial border of the scapula to avoid injury to the dorsal scapular artery and nerve. The bursa under the serratus anterior can be distended with fluid before a stab wound is made in the skin and the blunt obturator and endoscope are inserted. Deep penetration may traverse the serratus entering the axillary space and should be avoided.

Once this initial middle portal has been established, a superior portal placed three fingerbreadths medial to the vertebral border of the scapula just below the spine will penetrate the interval between the rhomboideus major and rhomboideus minor. This portal will allow access to the superomedial angle of the scapula. Portals placed superior to the scapular spine jeopardize the dorsal scapular nerve and artery, the spinal accessory nerve, and the transverse cervical artery and should be avoided. A third inferior portal can be made in a similar fashion at the inferior angle of the scapula.

In the bursa between the serratus anterior and chest wall, landmarks are generally absent except the ribs. A motorized shaver and electrocautery are required to perform the bursectomy and obtain hemostasis. The arthroscopic pump should be kept at low pressure throughout the procedure. After completing the bursectomy, the portals are closed in a standard fashion and the patient is placed in a sling for comfort. Physical therapy beginning with active range of motion is initiated as tolerated by the patient.

Postoperative Considerations

With an open resection, the patient uses a sling for comfort and begins passive motion and pendulum exercises immediately. At 3 weeks active motion is allowed, with strengthening begun at 12 weeks.

Results of Treatment

In general, most patients do well with open resection of painful scapulothoracic bursectomy. With this technique, 88% of patients with symptomatic scapulothoracic bursitis

had good or excellent results. One patient with a fair result also required muscle transfers for trapezius winging.[117] Arthroscopic treatment also reports high rates of success.[13,23,54,95,113]

Complications

Complications associated with open or arthroscopic bursectomy include pneumothorax, postoperative hematoma, potential long thoracic nerve injury, and recurrence of the bursitis. Recurrence is more likely if the patient had bony pathology with crepitus and the bony pathology was not addressed at the time of bursectomy.

CONCLUSIONS

The importance of the scapulothoracic articulation has gained more attention as of late. A variety of pathologic conditions may affect the scapulothoracic articulation. It is not surprising that some are intimately related to problems that affect the glenohumeral joint. A comprehensive evaluation of the shoulder must include an assessment of the glenohumeral joint to look for winging, crepitus, bursitis, and dyskinesis.

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Injuries of the Brachial Plexus

35

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ANATOMY

Brachial Plexus

The origin of the brachial plexus is from the fifth through eighth cervical (C5 through C8) and the first thoracic (T1) spinal nerves.[85,127] Small contributions may originate from the fourth cervical (C4) and second thoracic (T2) nerves. The dorsal and ventral rootlets (six to eight rootlets per level) exit the spinal cord, merge to form the spinal nerves, which leave the intervertebral foramina, and quickly divide into dorsal and ventral rami (Fig. 35-1). The small dorsal rami travel posterior to innervate the skin and muscles of the neck and upper back and are not part of the brachial plexus. The ventral rami emerge between the anterior and middle scalene muscles and are designated as the nerve roots of the brachial plexus (Fig. 35-2). The upper roots (C5 through C8) descend toward the first rib, whereas the lower T1 root must ascend over the first rib to form the brachial plexus. Sympathetic fibers join the nerve roots as they traverse between the scalene muscles. C5 and C6 receive fibers from the middle sympathetic cervical ganglion, and C7, C8, and T1 acquire fibers from the cervicothoracic ganglion. These sympathetic fibers control blood-vessel smooth-muscle contraction (vasoconstriction) and sweat gland activity.

The motor cell bodies of the nerve roots for the brachial plexus are located within the ventral horn of the spinal cord gray matter (see Fig. 35-1). In contrast, the sensory cell bodies are positioned outside the spinal cord within the dorsal root ganglia. The dorsal root ganglia transfer afferent fibers to the spinal cord through the dorsal rootlets. The knowledge of the difference in anatomic location of cell bodies between motor and sensory fibers is

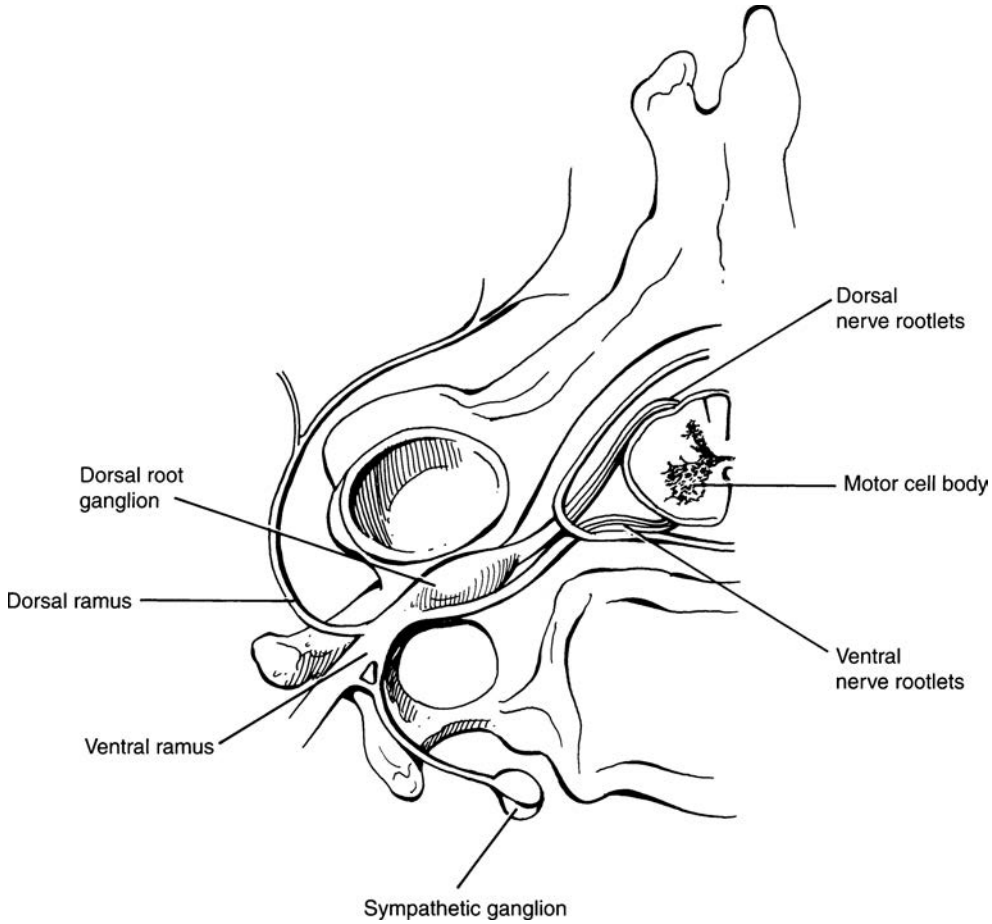

Figure 35-1 Artist's schematic of spinal cord with motor and sensory (dorsal root ganglion) cell bodies.

important for the accurate diagnosis and treatment of proximal brachial plexus injuries.

The ventral rami of C5 and C6 combine to form the superior trunk, the C7 ramus continues alone as the middle trunk, and C8 and T1 unite to form the lower trunk (see Fig. 35-2). The trunks are located in the posterior triangle of the neck, enclosed by the posterior border of the sternocleidomastoid muscle, anterior border of the upper trapezius, and clavicle. The spinal accessory nerve (cranial nerve XI) crosses the posterior triangle to innervate the trapezius muscle. This nerve divides the posterior triangle into nearly equivalent upper and lower parts. The lower portion of the triangle contains the brachial plexus, with the upper and middle trunks superior to the omohyoid muscle and the lower trunk inferior. Each trunk divides into anterior and posterior divisions and proceeds behind the clavicle. The divisions then merge into three cords named in relation to the axillary artery. The anterior divisions of the upper and middle trunks combine to form the lateral cord. The three posterior divisions of the upper, middle, and inferior trunks converge to form the posterior cord. The anterior division of the lower trunk continues as the medial cord.

The cords proceed behind the pectoralis minor muscle into the axilla. Each cord divides into two terminal branches (Fig. 35-3). The lateral cord terminates as the musculocutaneous nerve and a branch to the median nerve. The musculocutaneous nerve perforates and supplies the coracobrachialis muscle and then becomes the principal motor nerve of the flexor compartment of the arm. The posterior cord divides into the axillary and radial nerves. At the level of the glenohumeral joint, the axillary nerve, along with the posterior humeral circumflex vessels, travels inferior to the subscapularis muscle and across the upper border of the teres major to enter the quadrangular space for innervation of the deltoid and teres minor muscles. Compression of the nerve or artery can occur at the quadrangular space from hypertrophy or anomalies of the bordering muscles. The radial nerve is the largest branch of the brachial plexus and passes inferior to the teres major muscle to enter the posterior arm between the long head of the triceps and humerus. The medial cord continues as the ulnar nerve and a branch to the median

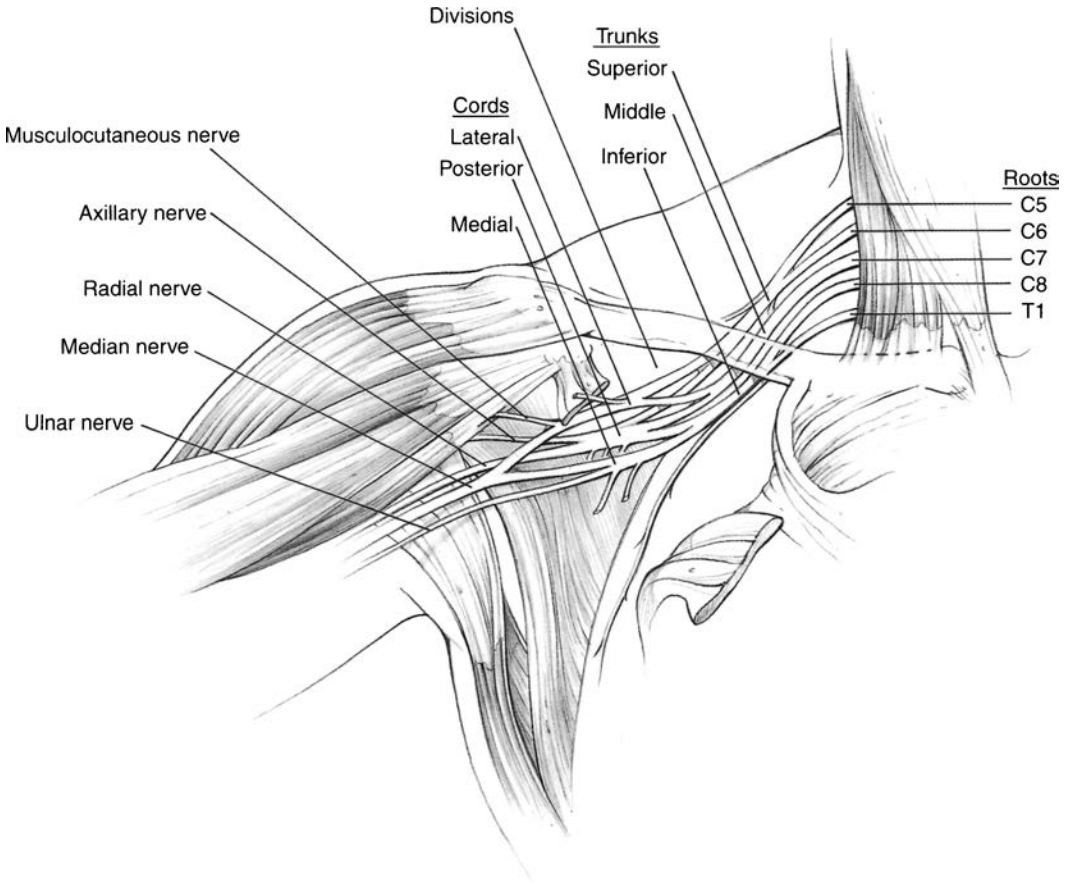

Figure 35-2 Artist's schematic of brachial plexus from roots to branches.

nerve. The ulnar nerve travels down the arm medial to the brachial artery, pierces the medial intermuscular septum, and enters the cubital tunnel. The median nerve forms anterior to the axillary artery from the union of the medial and lateral cord branches and descends into the arm.

Several branches arise from the roots, trunks, and cords of the brachial plexus (see Fig. 35-2). The status of these intermediate nerves provides valuable information about the location of nerve injury. At the root level, the dorsal scapular nerve arises from C5, pierces the middle scalene to enter the posterior triangle of the neck, and innervates the levator scapulae and rhomboid muscles. The long thoracic nerve arises from C5, C6, and C7 just distal to the intervertebral foramina and travels behind the brachial plexus along the chest wall to supply the serratus anterior muscle. The phrenic nerve originates at the root level from C3, C4, and C5 and crosses the anterior scalene muscle to enter the thorax. The phrenic nerve may be injured in nerve root injuries, which results in hemidiaphragm paralysis.

At the trunk level, the suprascapular nerve arises from the superior trunk to travel across the posterior cervical triangle to the suprascapular notch. The suprascapular nerve advances through this notch to innervate the supraspinatus and infraspinatus muscles.

There are no branches from the plexus at the division level. At the cord level, multiple branches are present. From the lateral cord, the lateral pectoral nerve arises to pass anterior to the axillary artery, to perforate the clavipectoral fascia, and to innervate the clavicular part of the pectoralis major. The posterior cord supplies three branches: the upper subscapular, thoracodorsal, and lower subscapular nerves. The upper subscapular nerve innervates the upper portion of the subscapularis muscle. The lower subscapular nerve innervates the lower subscapularis and the teres major muscle. The thoracodorsal nerve originates between the upper and lower subscapular nerves, passes behind the axillary artery, and supplies the latissimus dorsi muscle. The medial cord provides one motor and two sensory branches. The medial pectoral nerve traverses and innervates the pectoralis minor muscle and then continues to supply the sternocostal portion of the pectoralis major muscle. The medial brachial and medial antebrachial cutaneous nerves are the only sensory branches to arise directly from the plexus and supply the arm and forearm, respectively.

The vascular anatomy of the brachial plexus centers about the subclavian and axillary vessels. The subclavian artery originates from the arch of the aorta on the left side and from the brachiocephalic artery on the right. The

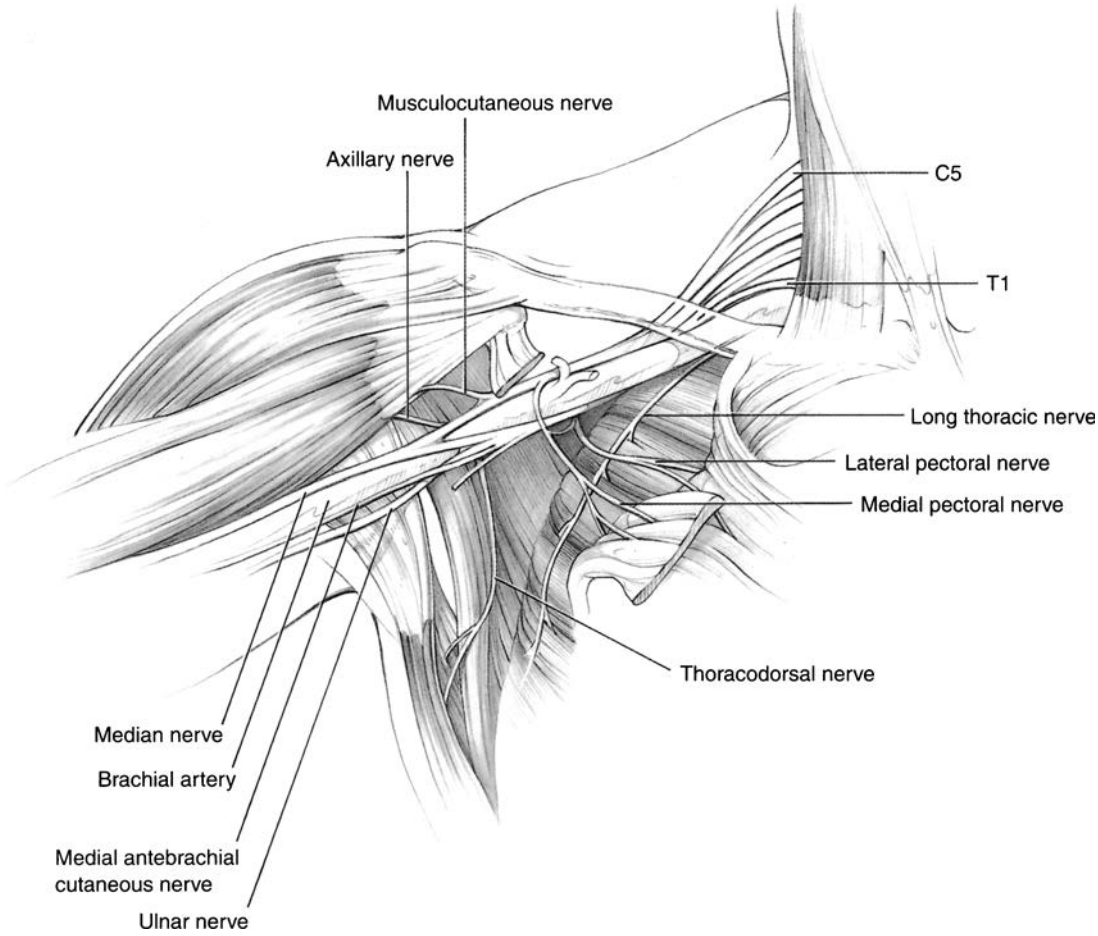

Figure 35-3 Artist's schematic of brachial plexus behind the clavicle pectoralis minor muscle.

subclavian artery ascends over the first rib to reside between the anterior and middle scalene muscles, with the roots and trunks of the brachial plexus. In contrast, the subclavian vein is located anterior to the anterior scalene muscle. The subclavian vessels cross the first rib and become the axillary vessels with the vein medial to the artery. The axillary vessels accompany the brachial plexus behind the pectoralis minor muscle to enter the axilla. The axillary vessels become the brachial vessels beyond the axilla.

There is variability and asymmetry in the neural and vascular anatomy of the brachial plexus.[85,127,190] The plexus is termed *prefixed* when there is a relatively large contribution from C4 and a small allotment from T1. Similarly, a *postfixed* plexus has substantial contribution from T2 with little from C5. There are also variations in cord separation and peripheral branchings of nerves that may or may not affect segmental innervation. Branches may occasionally arise from divisions that usually originate from trunks or cords. The vascular relation to the plexus can be altered, with the axillary artery or vein shifted in position or even piercing a nerve.[115] The subclavian vein can travel with the artery and brachial plexus posterior to the anterior scalene muscle.[128] Anomalies in nerve and/or vascular anatomy should be considered when clinical, diagnostic, and surgical findings do not correspond.

Thoracic Outlet Anatomy

The thoracic outlet begins just distal to the intervertebral foramina and extends to the coracoid process. The outlet is surrounded by anatomic constraints that encompass the brachial plexus and associated vessels (subclavian and axillary). These structures include muscles (anterior and middle scalene muscles), skeleton (first rib, cervical ribs, clavicle, and coracoid), and fascia or fibrous bands. The most common sites of compression in thoracic outlet syndrome are at the superior thoracic outlet, the scalene interval or triangle, the costoclavicular space, or the subcoracoid area (Fig. 35-4; Table 35-1).[6,130,143,153] This compression can be static or dynamic (i.e., dependent on posture and activity).[98]

The anterior border of the superior thoracic outlet is the sternum; lateral boundary in the first rib and the posterior border in the thoracic vertebrae.[203] The inferior trunk must

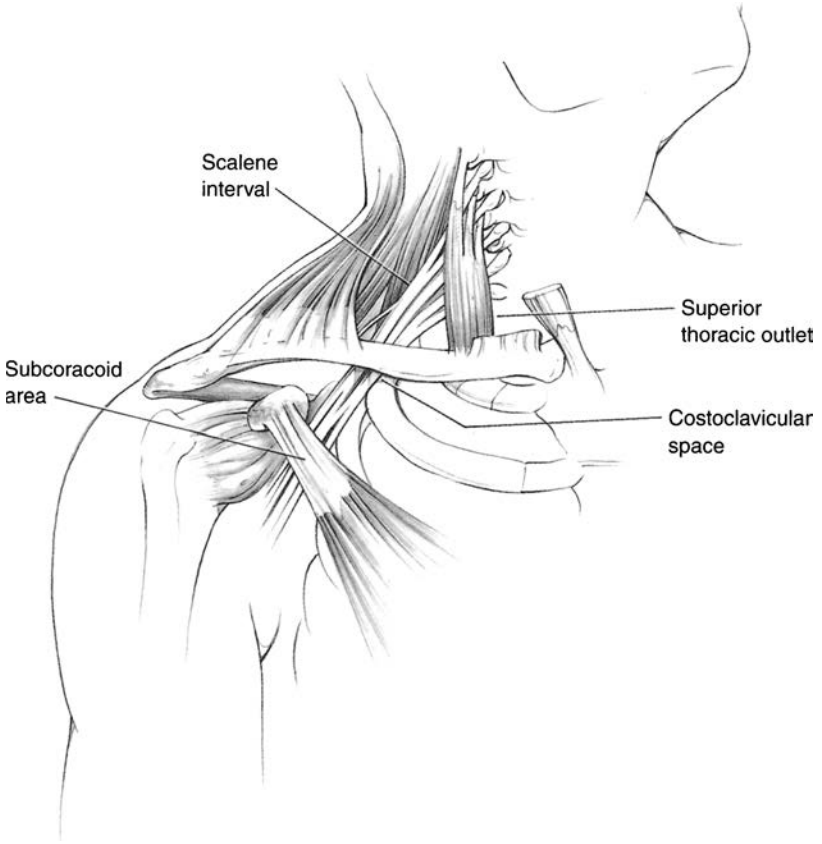

Figure 35-4 Artist's schematic of thoracic outlet compression sites.

ascend from the intervertebral foramina to navigate over the first rib. A postfixed brachial plexus must climb even higher to exit from the thorax. The inferior trunk can be compressed or stretched over the first rib in this area.

The scalene triangle is formed by the anterior and middle scalene attachments on the first rib. These muscles originate from similar transverse processes of the upper and middle cervical vertebrae and diverge to their insertion sites. The scalene triangle has a narrow base (approximately 1 to 2 cm) and elongated sides.[43,87,143] The upper roots (C5 through C7) descend, whereas the inferior roots (C8 and T1) and subclavian artery must ascend to pass through the scalene triangle. The presence of a cervical rib or a fibrous band extending from an incomplete cervical rib to the first rib can reduce the dimensions of the triangle by elevation of its base. This forces the inferior roots and subclavian artery to further ascend to enter the scalene triangle. Cervical ribs are present in 0.5% to 1% of individuals and occur bilaterally 50% to 80% of the time.[6,98,141,169] The width of the scalene triangle can be narrowed by anterior or middle scalene muscle abnormalities, which can precipitate thoracic outlet compression.[43,81,179]

TABLE 35-1
SITES OF COMPRESSION IN THORACIC OUTLET SYNDROME

Site	Principal Cause
Superior thoracic outlet	First rib or cervical rib
Scalene interval or triangle	Scalene muscles or fibrous bands
Costoclavicular space	Narrow clavicle–first rib distance
Subcoracoid area	Coracoid process

The costoclavicular interval is between the clavicle and first rib. Depression of the clavicle reduces this space and can compress the brachial plexus and subclavian vessels. A hypertrophied subclavius muscle or a clavicle with abundant callus formation can narrow the costoclavicular space. The subclavian vein is also susceptible to compression within the costoclavicular interval.

The subcoracoid area can compress the brachial plexus. The coracoid provides a fulcrum across the plexus during abduction and external rotation of the arm. The pectoralis minor and conjoined tendon (short head of the biceps and coracobrachialis) prevents slippage of the plexus from behind the coracoid. Excessive arm elevation can lead to a traction or compressive neuropathy along the coracoid process.[175]

ADULT BRACHIAL PLEXUS INJURIES

Adult brachial plexus injuries may be secondary to vehicular trauma, athletic endeavors, domestic violence, or systemic disease. The causes can be divided into trauma (penetrating and nonpenetrating), entrapment, and infection (Table 35-2). There are other less common causes of brachial plexopathy related to tumors, neuropathies, and iatrogenic causes.[6,69,79,109]

Pathophysiology

The most common cause of brachial plexus injury is traction.[175] The mechanism that yields traction is variable and may be from pulling the extremity, forcible head rotation from the shoulder, or direct depression of the shoulder girdle.[175] Traction causes injury via stretching or straining of the nerve beyond its physiologic limits. There is a direct correlation between strain and reduction of intraneural blood flow. A 15% elongation reduces blood flow approximately 80% to 100%.[35,102,200] The extrinsic vascular system (longitudinal vessels in the epineurium) appears to be more susceptible to stretch than the intrinsic circulation (endoneurial vascular network).[89] Continued elongation will cause overt ischemia and disruption of nerve metabolism. Persisted traction will ultimately disrupt nerve fiber and/or sheath continuity. Therefore, traction can cause a spectrum of nerve injury ranging from a temporary disruption in nerve fiber conduction to functional discontinuity of the nerve fibers and sheath.

TABLE 35-2
CAUSES OF BRACHIAL PLEXUS INJURIES

Trauma
Nonpenetrating (traction)
Penetrating (knife, gunshot wound)
Nerve entrapment
Thoracic outlet syndrome
Infection
Viral plexopathy (Parsonage-Turner syndrome)
Radiation
Fibrosis
Malignant degeneration after radiation
Tumors
Primary (schwannomas or neurofibromas)
Secondary (pulmonary apices)
Neuropathies
Iatrogenic
Axillary or scalene anesthesia
Surgical biopsy
Intraoperative positioning
Median sternotomy
Inadvertent traction

Various factors determine the portion of the plexus injured and the extent of damage incurred. These determinants include the local anatomy, magnitude and duration of the force applied, and the position of the extremity and head at time of accident.[175] The upper roots descend beneath the clavicle, whereas the lower roots ascend over the first rib. Therefore, the upper plexus is taut with the arm dangling at the side, and the lower plexus is taut with the arm abducted and elevated. As a result, the upper roots and trunk of the plexus are most susceptible to traction that increases the distance between the head and neck. In contrast, the medial, lateral, and posterior cords pass beneath the coracoid, which act as a pulley and can direct the force incurred. Therefore, excessive arm abduction elevates the coracoid pulley and places preferential tension across the cords of the brachial plexus. As a result, disruptive forces in the form of traction often direct maximal strain across specific segments within the plexus, which leads to variable injury across the plexus.

Nonpenetrating Trauma

Nonpenetrating trauma is the leading cause of upper brachial plexus injuries. Most brachial plexus injuries result from motor vehicle accidents, especially motorcycle misadventures.[124,156] The injury is more frequently seen in areas where motorcycles are the principal mode of transportation.[29] The incidence of brachial plexus injury following a motorcycle accident has been approximated to be 2%, with a prevalence in young males.[124,125] Interestingly, the incidence has recently increased and is probably related to mandatory helmet regulations and better transportation of trauma patients who previously would have succumbed to their injuries.[125] Associated injuries are common, including head trauma and fractures or dislocations of the cervical spine, shoulder, forearm, and hand.[124,156]

Automobile, bicycle, sporting, and pedestrian accidents are much less common causes of brachial plexus injuries, as are shoulder dislocations or fractures (proximal humerus, clavicle).[73,112] Minor stretch injuries of the plexus (i.e., burners or stingers) are common in contact sports, especially football, because tackling drives the shoulder downward and forcibly flexes the neck toward the contralateral side.[73] The athlete experiences burning pain and paresthesias that radiate from the supraclavicular area into the arm. Transient weakness and sensory abnormalities are present. The vast majority of burners are upper plexus traction injuries. There should not be concomitant neck pain or restricted neck mobility. The signs and symptoms usually resolve spontaneously over a few minutes. Incomplete neurologic recovery or restricted neck motion requires further diagnostic evaluation as the impact could have caused a cervical spine fracture–dislocation or a severe brachial plexus injury. Failure to make a timely diagnosis of cervical spine injury can result in subsequent quadriplegia.

Shoulder dislocations are often related to sporting accidents and can produce nerve injuries of the axillary, suprascapular, or musculocutaneous nerves.[4] These are usually (80% of the time) temporary lesions that resolve over 4 to 6 months (80%) and are covered in Chapter 36.

Penetrating Trauma

Penetrating trauma to the brachial plexus is often secondary to gunshot wounds or stabbings. Gunshot wounds occur at time of war and during domestic violence.[91,133] Bullet injuries act as projectile missiles that cause neural injury by means of a blast effect or less commonly direct nerve transection.[18,91,133] The shock wave (temporary cavity) reverberates, causing a variable degree of nerve injury. The extent of blast correlates with the deformation, fragmentation, orientation, and velocity of the bullet. The part of the plexus injured depends upon the entrance and exit passageway.

In contrast to gunshot wounds, stab wounds transect portions of the plexus or cause vascular injury with secondary nerve compression by expanding hematoma.[18,47] Most stab wounds to the supraclavicular area involve the upper and middle plexus as the clavicle protects the lower portion.[47]

Infection

Infection is an uncommon cause of brachial plexopathy. The exact cause remains unclear, with both viral pathogens and secondary immunologic factors proposed as possibilities. This brachial neuritis, also known as Parsonage-Turner syndrome, presents with acute onset of intense pain about the shoulder girdle without antecedent trauma.[136] Weakness of the muscles innervated by the affected nerves develops after the initial pain subsides. The suprascapular or long thoracic nerve is most commonly affected (Fig. 35-5).

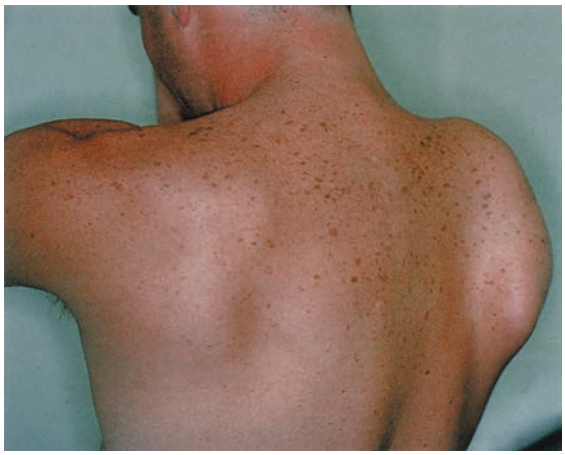

Figure 35-5 A 20-year-old man who awoke with intense shoulder pain and subsequent scapula winging from isolated long thoracic nerve involvement in Parsonage-Turner syndrome.

Electromyographic abnormalities of the denervated muscles are apparent 3 to 4 weeks after the initial pain response, with positive sharp waves and fibrillation potentials. The treatment is conservative management with observation and serial examinations. The natural history of brachial neuritis is resolution of the pain followed by improvement in motor function. However, prolonged recovery time (1 to 2 years) and persistent weakness is not uncommon years after the brachial neuritis.[73,105,120,183]

Classification of Brachial Plexus Injury

The lesion in brachial plexus injury is classified according to the anatomic location and extent of nerve involvement. Supraclavicular lesions affect the roots, trunks, and divisions, whereas infraclavicular injuries involve the cords and branches. A supraclavicular injury can disrupt the rootlet connection with the spinal cord and is called an *avulsion* injury. An avulsion is usually secondary to traction along the affected root(s) and separates the motor cell body in the spinal cord from its axons. The sensory cell body, however, is located in the dorsal root ganglion and remains connected to its axons (see Fig. 35-1). Therefore, the motor portion of the nerve undergoes wallerian degeneration, with degradation of the axons and myelin sheaths. The sensory fibers are spared from wallerian degeneration, but have been irrevocably detached from the spinal cord. The injury will cause a clinical motor and sensory loss, whereas electrodiagnostic studies will reveal abnormal motor findings with intact sensory conduction. In rare instances, the rupture can cause isolated disruption of only the motor or sensory rootlets.

The injury can also interrupt nerve continuity at the trunk level and is called a *rupture.* Ruptures of the plexus separate both motor and sensory cell bodies from their axons and wallerian degeneration occurs across all fibers. The differentiation between avulsion and rupture is a decisive element in the treatment algorithm of brachial plexus traction injuries. Discontinuity by rupture can be treated by various surgical techniques to reestablish nerve continuity. In contrast, avulsion injuries are irreparable and require alternative techniques to restore function. The diagnosis can be difficult, as traction can cause ruptures and avulsions at different levels, which complicates accurate diagnosis.

Supraclavicular lesions account for most brachial plexus injuries (approximately 75%) and are also subdivided into groups according to the pattern of involvement (Table 35-3).[5,119] The Erb-Duchenne palsy involves C5 and C6, or the upper trunk, and is characterized by loss of elbow flexion and weakness of shoulder abduction and external rotation.[46,53] Sensory deficit is apparent in the corresponding dermatome (radial side of forearm and thumb). A C7 injury can accompany an Erb's palsy (a.k.a., extended upper brachial plexus lesion) and adds paralysis

TABLE 35-3
PATTERNS OF BRACHIAL PLEXUS INJURIES

Pattern	Roots Involved	Primary Deficiency
1. Upper brachial plexus (Erb-Duchenne)	C5 and C6	Shoulder abduction and external rotation Elbow flexion
2. Extended upper brachial plexus	C5, C6, and C7	Above plus Elbow and digital extension
3. Lower brachial plexus (Dejerine-Klumpke)	C8 and T1	Hand intrinsic muscles Finger flexors
4. Total brachial plexus lesion	C5, C6, C7, C8, and T1	Entire plexus
5. Peripheral brachial plexus lesion		Variable

of elbow extension, wrist extension (extensor carpi radialis brevis), and finger extension (extensor digitorum communis and propius). The Dejerine-Klumpke palsy involves C8 and T1, or lower trunk, and is characterized by absent intrinsic hand musculature and finger flexors (flexor digitorum profundus and superficialis) with intact shoulder, elbow, and wrist function.[92] Sensory deficit is situated over the ulnar side of the forearm and hand. This isolated lower plexus palsy is uncommon in both adults and children.[57,72] Lastly, the injury can include the entire plexus (C5, C6, C7, C8, and T1), which causes a flail and anesthetic arm.

Supraclavicular lesions can also be isolated to peripheral branches, such as the suprascapular or long thoracic nerve. This can be secondary to trauma, infection, and surgical positioning, or can be iatrogenic.

Infraclavicular lesions are less common (approximately 25%) and usually represent stretch injuries from an associated shoulder dislocation or fracture.[4] These injuries represent peripheral nerve lesions of the plexus. The axillary nerve is particularly susceptible to traction because it is securely anchored as it traverses the quadrangular space. However, injury to the musculocutaneous nerve and other elements of the brachial plexus can occur after severe trauma. These injuries are covered in Chapter 36.

Supraclavicular and infraclavicular nerve injuries can also be characterized by their severity, regardless of the location of injury and extent of plexus involvement. The gradation of nerve injury begins with neurapraxia, extends to axonotmesis, and culminates in neurotmesis (Table 35-4).[163] *A neurapraxia* is a segmental demyelination with maintenance of intact nerve fibers and axonal sheath. A temporary conduction block follows, without axonal damage and wallerian degeneration. Complete recovery occurs over the ensuing days to weeks as remyelinization is completed. Electrodiagnostic studies demonstrate a decrease in nerve conduction without electromyographic changes of denervation within the muscle.[21] An *axonotmesis* is a disruption of nerve fiber integrity with preservation of

TABLE 35-4
SEDDON'S CLASSIFICATION OF NERVE INJURY

Type	Definition	Outcome
Neurapraxia	Interruption of nerve conduction; some segmental demyelination; axon continuity intact	Reversible
Axonotmesis	Axon continuity disrupted; neural tube intact	Wallerian degeneration; incomplete recovery
Neurotmesis	Complete disruption of nerve continuity; loss of axons and neural tubes	No spontaneous recovery; surgery required

Adapted from Seddon HJ. *Surgical disorders of peripheral nerve injuries*, 2nd ed. Edinburgh, Churchill-Livingstone, 1972.

the axonal sheath and framework. Wallerian degeneration and nerve fiber regeneration are necessary for recovery. Electrodiagnostic studies exhibit a decrease in nerve conduction and electromyographic changes of muscle denervation (insertional activity, fibrillations, and positive sharp waves).[21] Wallerian degeneration is characterized by the proliferation of Schwann cells that phagocytose myelin and axon debris. The axons distal to the injury degrade from lack of nutrition and loss of blood supply. The regeneration rate is approximately 1 mm/day or 1 in./month. This slow regeneration delays recovery and means that distal nerve injuries have a better prognosis because the extent of wallerian degeneration is decreased and the proximity to the motor endplates is increased. In addition, prolonged denervation of longer than 18 to 24 months results in irreversible motor endplate degradation and muscle fibrosis. In contrast, the encapsulated sensory receptors retain their capacity for reinnervation for many years. These factors lend the overall prognosis for axonotmesis as variable and guarded.

A *neurotmesis* is a disruption of the nerve fiber and axonal sheath. Transection is the classic example of this injury, but severe traction or contusion can produce a similar injury with irreversible intraneural scarring. The prognosis is bleak without surgical resection of the intervening scar and nerve coaptation by direct repair or graft interposition to allow for nerve regeneration. A severe brachial plexus injury often represents a combined lesion, with elements of neurapraxia, axonotmesis, and neurotmesis. This combination injury complicates accurate diagnosis and predictions for recovery.

Evaluation of Adult Brachial Plexus Injury

The clinical evaluation of the patient with a brachial plexus injury begins with a careful history and detailed examination. The examination should include the head and neck, thorax, injured extremity, and neurovascular systems. Knowledge of brachial plexus anatomy and concomitant muscle innervation is a prerequisite to accurate diagnosis. A thorough physical examination is the foundation that will dictate the treatment algorithm. Imaging studies and electrodiagnostic tests provide supplemental information to further clarify the extent of injury. The goal of this evaluation is to precisely define the location and extent of nerve injury. This information will direct treatment, which may range from continued observation to prompt surgical intervention.

Adult brachial plexus injuries are usually not evaluated in the acute setting, but rather referred after treatment of any life-threatening injuries. An inquiry into the mechanism of injury and degree of energy involved is important. The force applied (direction, magnitude, and duration), position of extremity, and concomitant injuries (fractures, dislocations, visceral damage, head trauma, and vascular damage) are important details. Unfortunately, the particulars of the accident are often obscured by loss of consciousness, associated injuries, or amnesia.[125] In sharp penetrating trauma, the timing of neurologic deficit is important. An instantaneous deficit implies nerve laceration, whereas a delayed onset indicates a compressive neuropathy by an expanding hematoma or false aneurysm.[18,47] Important past medical history includes overall general health before the accident, time interval from injury, hand dominance, occupation, and hobbies.

The physical examination begins with observation. The posture of the extremity and the manner by which the patient uses the extremity are important indicators of the segment of the brachial plexus involved. The motor and sensory deficits determined by the physical examination should be correlated with the ancillary studies to define the extent and pattern of injury to the plexus. The basis of the evaluation is to perform a detailed examination to determine the specific motor and sensory deficits. An inventory of the muscles innervated by the brachial plexus is imperative to accurately define the injury and provides a baseline to assess recovery. Physical findings should be recorded on a data sheet, including gradation of muscle strength according to the international muscle scoring system and the presence or absence of sensory deficits (Table 35-5).[2] Sensibility is assessed using two-point discrimination or Semmes Weinstein monofilament testing. This documentation method eliminates inadvertent omission of important elements of the examination and allows serial examinations to be performed by different individuals.

Careful examination of the muscles innervated by the proximal branches from the brachial plexus will help define the proximity of the plexus lesion to the spinal cord (Table 35-6). Disruption of the dorsal rami (paraspinal muscles), dorsal scapular (rhomboids and levator scapulae), and long thoracic (serratus anterior) nerves are suggestive of an avulsion injury. The presence of a Horner's syndrome results in a drooped eyelid, constricted pupil, sunken globe, and sweating deficiency along the affected side of the face and usually implies an avulsion injury at C8 and T1 (Fig. 35-6). However, the false-positive response rate for a Horner's syndrome is 10% and the false-negative figure is 28%.[71] Percussion of the supraclavicular and infraclavicular plexus is performed. A Tinel's sign is indicative of a postganglionic injury (e.g., rupture), for this sign will be absent in a preganglionic lesion (e.g., avulsion) because the link to the spinal cord and brain has been disrupted.

Postganglionic injuries are further localized by examination of the intermediate and terminal branches. The status of the suprascapular (spinati), thoracodorsal (latissimus dorsi), subscapular (subscapularis and teres major), and pectoral (pectoralis major and minor) will further define the injury. Examination of the pectoralis major muscle is particularly helpful because of its dual segmental innervation from the lateral pectoral (C5, C6, C7) and medial pectoral (C8, T1) nerves from the lateral and medial cords,

TABLE 35-5
BRACHIAL PLEXUS EXAMINATION SHEET

Muscle Tested	R	L	R	L	R	L
Trapezius (C3,C4,X1)						
Levator scapulae (C3,C4,C5)						
Rhomboids (C4,C5)						
Supraspinatus (C5,C6)						
Infraspinatus (C5,C6)						
Serratus anterior (C5,C6,C7)						
Teres major (C5,C6)						
Subscapularis (C5,C6,C7)						
Pectoralis major clavicle (C5,C6,C7)						
Pectoralis major sternocostal (C6,C7,C8 T1)						
Latissimus dorsi (C6,C7,C8)						
Biceps and brachialis (C5,C6)						
Deltoid (C5,C6)						
Teres minor (C5,C6)						
Pronator quadratus (C7,C8,T1)						
Pronator teres (C6,C7)						
Flexor carpi radialis (C6,C7)						
Flexor digitorum profundus II, III (C7,C8,T1)						
Flexor digitorum superficialis (C7,C8,T1)						
Flexor pollicis longus (C7,C8,T1)						
Abductor pollicis brevis (C6,C7,C8,T1)						
Opponens pollicis (C8,T1)						
Lumbricals (C8,T1)						
Triceps (C6,C7,C8)						
Supinator (C5,C6)						
Brachioradialis (C5,C6)						
Extensor carpi radialis longus (C6,C7)						
Extensor carpi radialis brevis (C6,C7,C8)						
Extensor carpi ulnaris (C7,C8)						
Extensor digitorum communis (C7,C8)						
Extensor digiti minimi (C7,C8)						
Extensor indicis (C7,C8)						
Extensor pollicis longus (C7,C8)						
Extensor pollicis brevis (C6,C7)						
Abductor pollicis longus (C6,C7)						
Flexor carpi ulnaris (C7,C8,T1)						
Flexor digitorum prof. IV, V (C7,C8,T1)						
Abductor digiti minimi (C8,T1)						
Abductor pollicis (C8,T1)						
Opponens digiti (C8,T1)						
Interossei (C8,T1)						

Muscle Grading Chart

Muscle Grade	Description
5, Normal	Full range of motion against gravity with full resistance
4, Good	Full range of motion against gravity with some resistance
3, Fair	Full range of motion against gravity
2, Poor	Full range of motion with gravity eliminated
1, Trace	Slight contraction without joint motion
0, Zero	No evidence of contraction

respectively. Selective atrophy of the clavicular head (lateral pectoral) or sternocostal head (medial pectoral) facilitates diagnosis, whereas complete atrophy implies a global injury. A peripheral vascular examination with palpation of the radial and ulnar pulses is a fundamental component of the evaluation, for damage to the axillary or subclavian vessels can occur at the time of initial injury. Decreased or absent peripheral pulses, a delayed neurologic deficit (expanding hematoma), and penetrating trauma warrant magnetic resonance angiography or arteriography.[18,47,55,176]

TABLE 35-6
INDICATORS OF AVULSION INJURIES AND POOR PROGNOSIS FOR RECOVERY

Finding	Implication
Denervation paraspinal muscles	Dorsal rami injury
Denervation rhomboid muscles	Dorsal scapular (C5) injury
Scapular winging	Long thoracic (C5, C7, C8) injury
Horner's syndrome	Cervicothoracic sympathetic injury
Absent Tinel's sign	Preganglionic separation from cord
Sensory impairment neck	Cervical plexus injury
Hemidiaphragm paralysis	Phrenic nerve injury
Cervical transverse process fx	Avulsion fracture with root injury
Pseudomeningocele	Dura and arachnoid avulsion injury
Anesthesia and intact conduction velocity	Dorsal ganglion intact, but avulsion from cord

Imaging Studies

Imaging studies are an important part of the evaluation. The information garnered provides valuable information about the level of injury. The radiology tests utilized are plain x-ray films, fluoroscopy, myelography, computed tomography (CT), and magnetic resonance imaging (MRI).

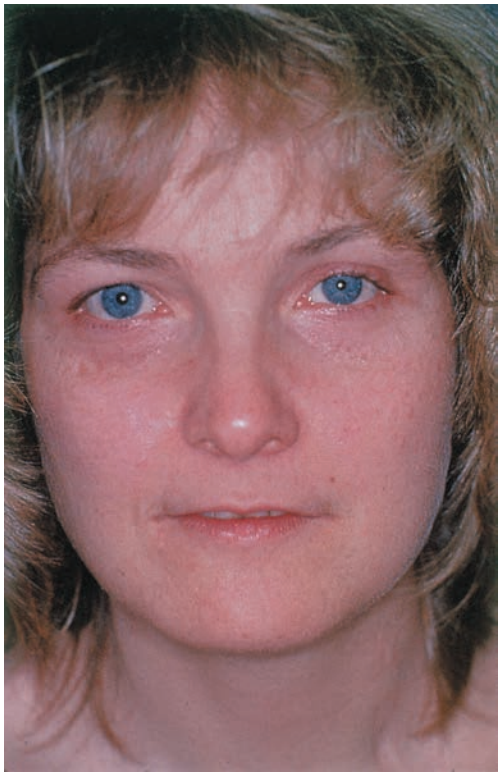

Figure 35-6 A 30-year-old woman involved in a motor vehicle accident with avulsions of C8 and T1 and ruptures of C5, C6, and C7. Persistent subtle left Horner's syndrome years after injury.

The particular studies employed vary with the type of injury, physical examination, imaging strategy, and expertise of the radiologist.

Plain x-ray films of the cervical spine, clavicle, shoulder, and chest are routine after a brachial plexus injury. Various findings are suggestive of particular levels of plexus injury (Table 35-7). The bone and/or ligament injury often represents a failure of the supporting and protective structures about the brachial plexus. For example, transverse process fracture or scapulothoracic dissociation implies a preganglionic avulsion injury while a shoulder dislocation infers a postganglionic nerve injury. Scapulothoracic dissociation is a devastating injury that can cause rapid exsanguination from an avulsion of the subclavian artery and brachial plexus. If the patient survives the injury, the nerve damage is usually extensive with avulsions across the plexus. The prognosis for return of nerve function is bleak, and extensive methods of nerve reconstruction are often required to restore nerve continuity.[49]

Hemidiaphragm paralysis from injury to the adjacent phrenic nerve may occur at the time of brachial plexus injury. Hemidiaphragm paralysis may not be apparent on a static chest x-ray film. Fluoroscopic evaluation during inspiration is required for diagnosis of phrenic nerve injury.

Angiography is indicated if there is any question of the vascular status of the extremity or integrity of the subclavian or axillary vessels. Palpable radial or ulnar pulses may be present (secondary to collateral flow) despite proximal injury.[18] Penetrating trauma or an expanding hematoma also requires angiography.[47,55,176]

Myelography is used to define the presence or absence of a pseudomeningocele, a meningeal pouch filled with cerebrospinal fluid that extends through the intervertebral foramen into the paraspinal area (Fig. 35-7). This pouch represents an extraction of the dural and arachnoidal sleeve through the intervertebral foramen that often occurs during a root avulsion injury. The inability to visualize the

TABLE 35-7
RADIOGRAPHIC FINDINGS IN INJURIES TO THE BRACHIAL PLEXUS

Plain x-ray Film	Findings	Significance
Chest	Elevated hemidiaphragm	Phrenic injury, proximal plexus, and possible preganglionic avulsion
	First-rib fracture	Subclavian or axillary artery injury, lower-trunk injury
C-spine	Fracture or dislocation	Cervical spine injury
	Transverse process fx	Preganglionic avulsion injury
Clavicle	Fracture	Possible traction injury to plexus or pseudoparalysis
Shoulder	Glenohumeral dislocation	Infraclavicular injury
	Scapulothoracic dissociation	Severe neurovascular injury

nerve root on the myelogram further supports the diagnosis of an avulsion injury. However, false-positive pseudomeningoceles have been found in patients in whom the rootlets were intact with isolated dura rupture, and false-negative results have been reported during surgical exploration.[34,57,72] Methods recommended to improve the diagnostic accuracy of myelography are to delay the test for 4 to 6 weeks after injury to allow for resolution of local swelling and intradural blood clots and using CT myelography to visualize small pseudomeningoceles.[125,151]

MRI has become the primary imaging modality in some institutions, based on the scanner available and specialization of the radiologist.[132,134] The MRI provides multiplanar imaging to assess the various components of the brachial plexus.[132] Strong magnetic gradients (greater than or equal to one tesla) and flexible surface coils improve image quality. Varying the pulse sequence will provide high-resolution images that will facilitate identification of plexus pathology. Similar criteria for the diagnosis of root avulsion (pseudomeningocele and nonvisualization of a nerve root)

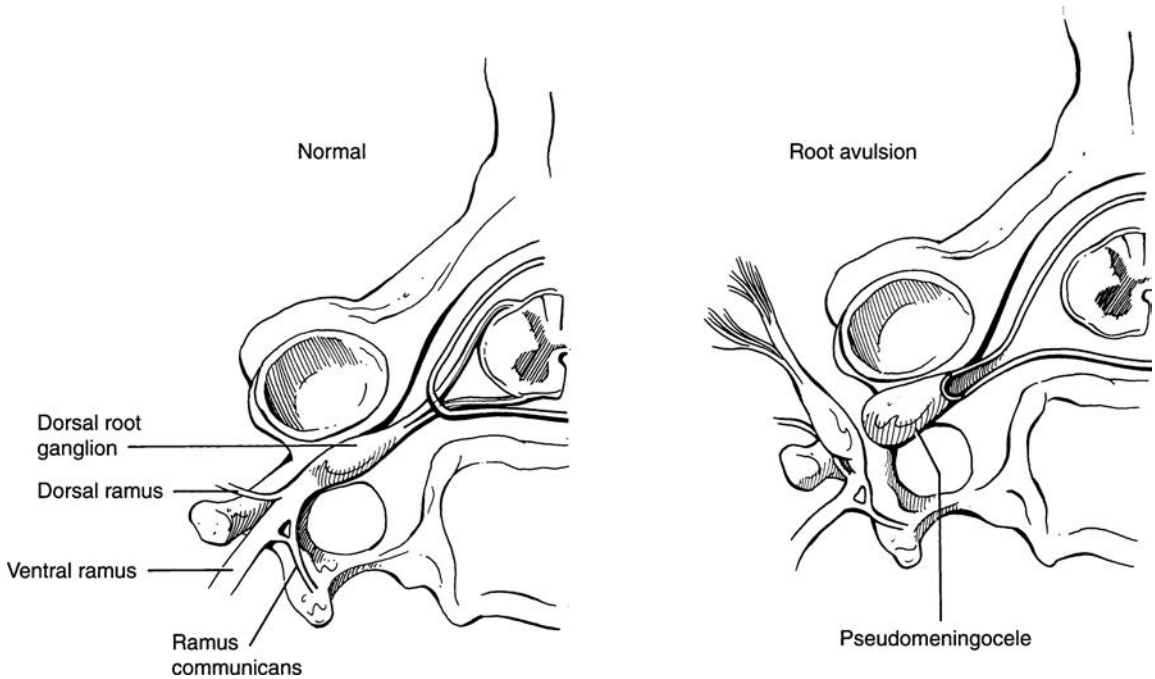

Figure 35-7 Artist's schematic of pseudomeningocele associated with avulsion injuries of the brachial plexus.

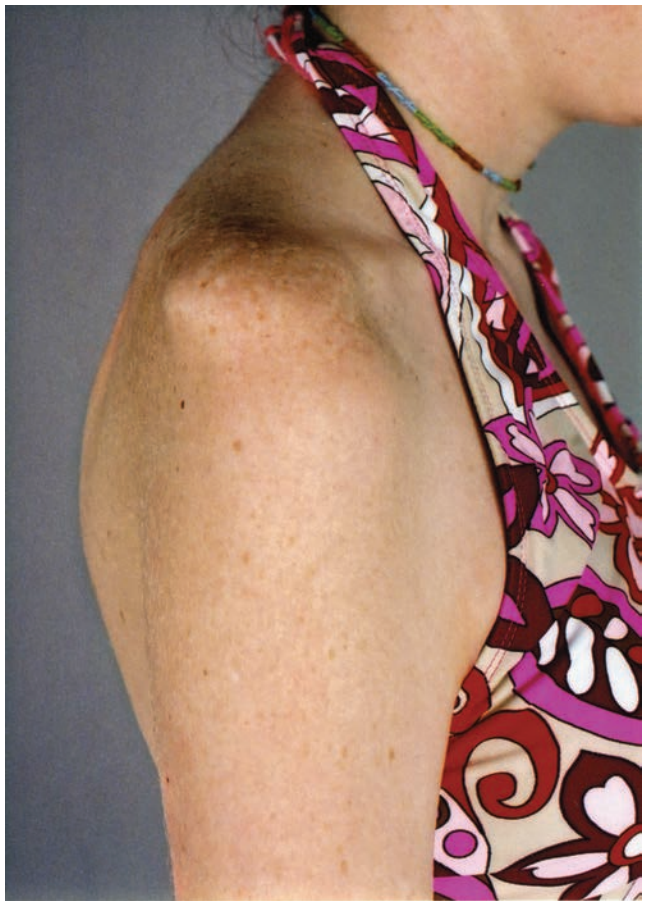

Figure 35-8 A 16-year-old female injured snowboarding with subluxation of shoulder and no active motion. (Courtesy of Shriners Hospital for Children, Philadelphia.)

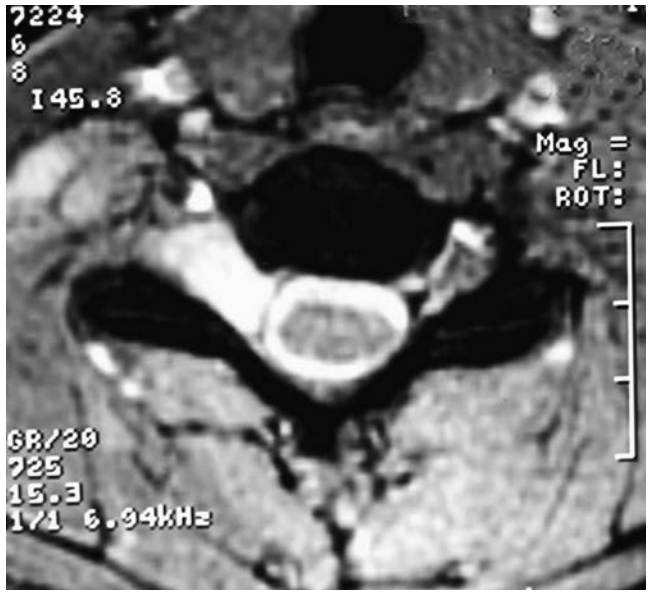

Figure 35-9 Magnetic resonance imaging reveals pseudomeningocele at C5 indicative of root avulsion. (Courtesy of Shriners Hospital for Children, Philadelphia.)

are used with MRI. MRI, however, offers better evaluation of the trunks and cords, with potential identification of a neuroma (Fig. 35-8 and 35-9).[132] MRI is also the modality of choice to evaluate neoplasms of the brachial plexus.

Electrodiagnostic Studies

Electrodiagnostic testing plays an integral part in the diagnosis and treatment of brachial plexus lesions.[44] The surgeon and neurophysiologist must have a dependable relationship with reciprocal communication. The results of neurophysiologic testing performed preoperatively, intraoperatively, and postoperatively may directly affect the decision-making process.[122] Methods employed to evaluate brachial plexus injuries are electromyography, nerve conduction velocity measurements, somatosensory-evoked potentials, and nerve action potentials.

Electromyography

Electromyography (EMG) records the electrical activity of muscle fibers at rest and during activation. This signal is recorded by the insertion of EMG needles into the muscle. The normal muscle is silent at rest and active during contraction with progressive recruitment of motor units. A denervated muscle will exhibit spontaneous electrical discharge (fibrillations and positive sharp wave) when the EMG needle is inserted.[21,44] These findings are not present until 2 to 4 weeks after denervation. A reinnervated muscle will begin to show reinnervation or nascent potentials (polyphasic low-amplitude recordings). These electrical changes of muscle regeneration will precede the clinical detection of muscle activity. Therefore, the EMG examination of the muscles innervated by the brachial plexus can provide valuable information about the degree of injury and early recovery. The dilemma with EMG interpretation, however, is the inability to quantify electrical recordings with extent of recovery. In other words, return of electrical activity may not correlate with return of active motion.

An EMG evaluation of the more proximal branches from the brachial plexus can help differentiate an avulsion injury from a rupture (see Table 35-6). For example, fibrillations and positive sharp waves of the paraspinal muscles (innervated by the dorsal rami from the spinal nerve) or serratus anterior (innervated by the long thoracic nerve) imply an avulsion injury, whereas preservation of a normal electrical signal suggests a more distal lesion. The EMG is also useful to differentiate the degree of intraneural injury (neurapraxia, axonotmesis, and neurotmesis) and to follow the progress after injury. A neurapraxia can be differentiated from a more severe nerve injury by the absence of fibrillation potentials and positive sharp waves. An axonotmesis or neurotmesis will exhibit these denervation changes. Serial EMG evaluations can infer an axonotmesis

injury by the spontaneous return of nascent units or reinnervation potentials that will precede clinical recovery of function. A neurotmesis injury will not exhibit EMG signs of spontaneous recovery.

Conduction Velocity

The integrity of the peripheral nerve is determined by the measurement of the conduction velocity. The motor or sensory latency can be measured depending on the recording of the compound motor action potential (CMAP) or the sensory nerve action potential (SNAP). The *conduction velocity* (CV) is the distance between two sites of stimulation divided by the time for the nerve action potential to travel from proximal to distal.[21] A severed nerve will lose the capability to conduct an action potential distal to the lesion as the nerve degenerates. However, the distal portion of the nerve may be able to conduct for several days after injury prior to degeneration. Therefore, early measurement of conduction velocities can produce a false-positive result for nerve continuity. Electrodiagnostic testing is often delayed for 3 weeks to allow time for loss of conduction and denervation changes in muscle.

The status of the SNAP and corresponding sensory nerve conduction velocity is helpful in differentiating preganglionic avulsion injuries from postganglionic lesions. The presence of a normal sensory CV in an anesthetic part of the arm indicates a preganglionic injury. The sensory nerve is not separated from its cell body, which is located in the dorsal root ganglion. The sensory nerve is separated from the spinal cord and sensation cannot be processed within the central nervous system. In contrast, the CMAP or motor CV is absent in both preganglionic and postganglionic injuries and is not a distinguishing factor.

Somatosensory-Evoked Potentials

Somatosensory-evoked potentials (SEPs) are electrical responses of the brain and spinal cord to stimulation of peripheral sensory fibers. Recordings of the conduction from the stimulating electrode to the central nervous system can be useful for the detection of lesions within the sensory system. This technique can be employed to assess conduction across the brachial plexus and during surgery to define irreparable nerve root avulsions. The absence of SEPs recorded over the spinal cord or contralateral sensorimotor cortex on nerve root stimulation indicates a root avulsion and is a contraindication for nerve grafting.[96,122]

Nerve Action Potentials

Nerve action potentials (NAPs) are used intraoperatively to assess lesions in continuity. Stimulating and recording of a nerve proximal and distal to a neuroma can identify the presence or absence of axonal continuity.[91] The presence of NAPs indicates propagation of an action potential along viable nerve fibers. This finding can help the surgeon decide between neurolysis or excision and interposition grafting.[91] This method is useful, but drawbacks include the inability to provide quantitative data and the incapacity to distinguish motor from sensory axons. Technical modifications to record the CMAP in innervated muscles or tetanic stimulation may provide better guidelines for surgical decision making.[44,91,97]

Nonoperative Treatment

The vast majority of adult brachial plexus injuries are initially treated without surgery. Brachial plexus injuries are often associated with other injuries that take precedence. Trauma resuscitation, repair of visceral damage, reconstitution of vascular flow, and stabilization of fractures dominate the acute management period. Brachial plexus injuries with concomitant upper extremity fractures are best treated by fracture fixation because of the high nonunion rate (almost 50%) and the need for early mobilization to prevent contracture, edema, and stiffness.[16] The initial goal of the postinjury period is to precisely define the location and extent of brachial plexus injury. This task is accomplished by a thorough examination and supplemented by the imaging studies previously described.

Following stabilization of the patient, the brachial plexus injury has certain features that require specific management (Fig. 35-10). Range-of-motion exercises and antiedema measures are required to prevent swelling and stiffness that will develop in the flail limb. The passive motion will also maintain supple joint structure and avoid contracture that will limit functional recovery. A sling is often applied to support the shoulder and to prevent additional traction across the plexus. This sling, however, must be removed for therapy as soon as the patient is comfortable. Electrical stimulation to diminish muscle wasting during nerve regeneration may be implemented, but its long-term efficacy is unclear.

Brachial plexus injury may result in neurogenic pain, which may be present after preganglionic and postganglionic injuries.[19] Preganglionic pain may be severe and constant, with a burning or crushing sensation. In most patients, this debilitating pain gradually subsides to a tolerable level. Postganglionic pain is from afferent signals that can still travel to the central nervous system. This pain can also be severe, but it usually dissipates over time to a bearable intensity. Pain management is accomplished by pharmacologic treatment, transcutaneous electrical stimulation, and other analgesic measures. Unfortunately, 5% of patients will exhibit persistent intractable pain that requires valiant attempts for pain relief, including ablation of the dorsal root entry zone (DREZ) or central nervous stimulators.[19,160] Amputation of the affected arm, however, will not relieve recalcitrant neurogenic pain.[196]

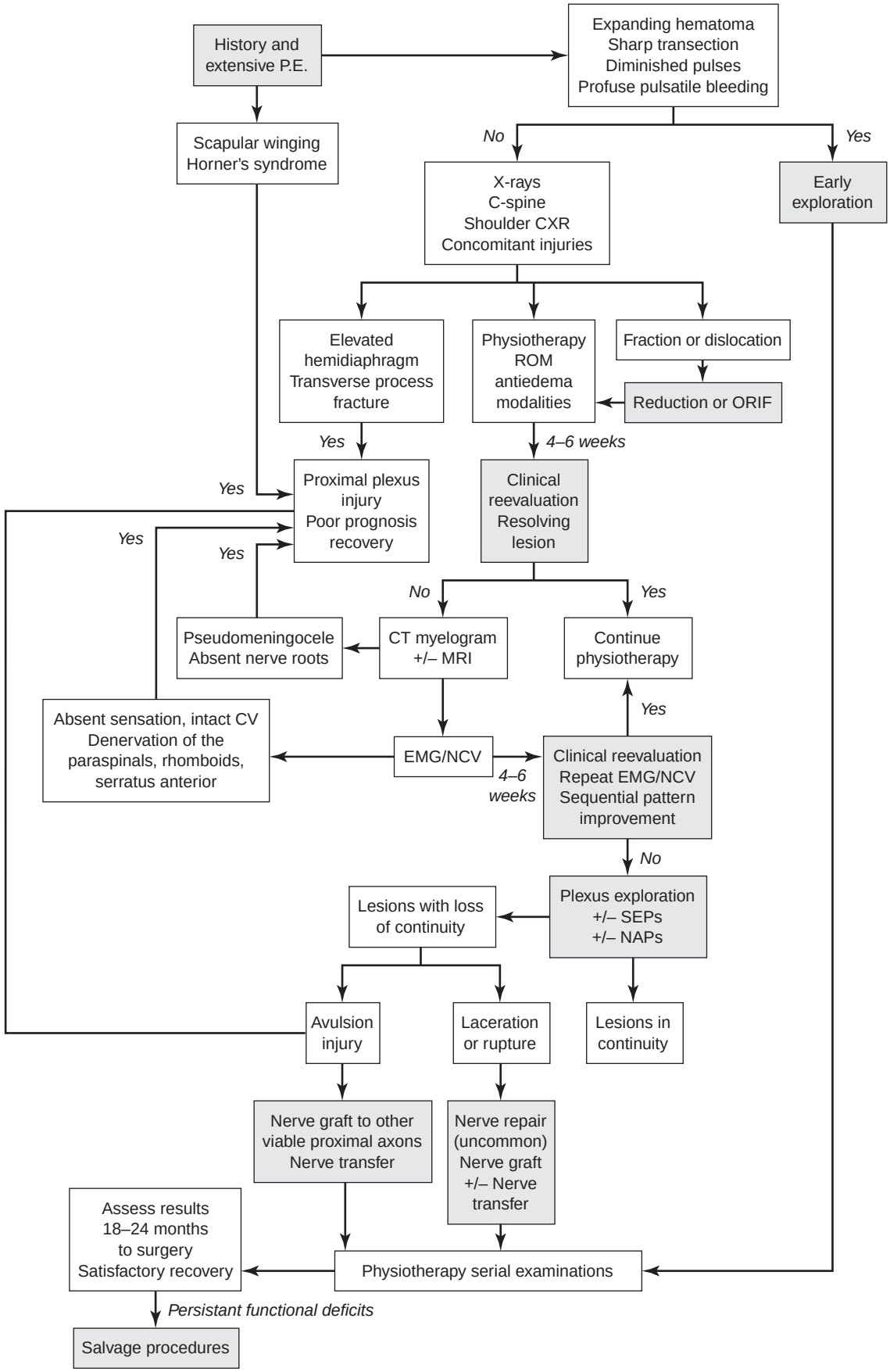

Figure 35-10 Treatment algorithm for adult brachial plexus injuries.

Serial examinations are performed to assess and further define the injury and to evaluate for signs of early recovery. X-rays of the chest, cervical spine, and shoulder girdle are evaluated for radiographic signs associated with a brachial plexus injury (see Table 35-7). Persistent deficits 3 to 4 weeks after injury warrant additional study. Options include advanced imaging studies (e.g., CT myelogram or MRI) and electrodiagnostic testing. Baseline electrodiagnostic studies are performed to assess for muscle denervation and the possibility of nerve rootlet avulsions (see Table 35-6).

Nonoperative management is usually continued for 3 months. Therapy is necessary to maintain passive motion and supple joints. Serial examinations are performed at monthly intervals. Repeat electrodiagnostic tests are often performed 3 months after injury. At this time, the clinical information and the ancillary studies are combined to define the level and extent of injury. The prognosis for recovery is also calculated. This observation period of 3 months is often sufficient time to adequately assess the injury and to decide whether spontaneous recovery is possible. The surgeon must also decide if the injury is amenable to surgical repair or reconstruction. Progressive recovery that follows a sequential pattern warrants continued observation and negates the need for exploration.

In contrast, a neurotmesis injury with loss of continuity (avulsion, rupture, laceration) or severe intraneural damage will not exhibit evidence of improvement at 3 months. In those reparable lesions, surgery is recommended between 3 and 6 months after injury. This time frame for surgery is important to ensure the viability of motor endplates to sprouting axons. A further delay in surgery may jeopardize the integrity of the motor endplates and prevent functional recovery, despite satisfactory nerve repair, grafting, or transfer. There is a trend toward earlier surgery if a nonrecoverable and reparable lesion can be identified to ensure endplate viability.

Infraclavicular lesions from fracture or dislocation are treated by prompt reduction of the fracture or dislocation. The nerve injury is often an isolated peripheral lesion and treated by observation. Neurapraxia and axonotmesis injuries are customary, and the prognosis for recovery is good. Exploration is reserved for those cases without spontaneous recovery.[4,125,126]

Surgical Management

Indications

Immediate surgical intervention for brachial plexus injuries is uncommon and is primarily performed for concomitant vascular injuries to the axillary or subclavian vessels (see Fig. 35-10). Simultaneous inspection of the plexus can be performed with identification of the injured segments. Immediate exploration is often frustrating, as the vascular repair cannot be adequately mobilized for fear of disruption. Once the injured nerve segments are identified, the treatment depends upon the status of the damaged segments. Sharp transections can be managed by direct repair or graft interposition. Primary neurorrhaphy requires approximation of the cut ends without excessive tension.[47,117,164] Primary neurorrhaphy is not recommended for traction or gunshot injuries. Delayed management is preferred to allow demarcation of the zone of injury and extent of nerve damage. Fortunately, most penetrating trauma by missile preserves nerve continuity.[18,91,133]

Delayed surgical management is usually performed 3 to 6 months after injury. The decision to operate is based on locating a repairable lesion that would not experience spontaneous recovery. The compilation of serial examination determines the level and extent of nerve injury. The time frame ensures the viability of motor endplates to sprouting axons via nerve grafting or transfer. Unfortunately, disruptions of the lower trunk (C8, T1) do not respond to nerve grafting in adults and are not an indication for surgery. This poor outcome reflects the distance required for the axons to travel to the motor endplates before irreversible changes.

Surgical Approach

The surgical exploration for brachial plexus injuries requires an extensile exposure of the supraclavicular and/or infraclavicular plexus. The procedure is performed with the patient supine, and ample preparation of the injured extremity, neck, hemithorax, and lower leg(s) for sural nerve grafts. Electrophysiologic testing apparatus may be used during surgery. This requires placement of scalp or cervical spine electrodes for SEPs and bipolar probes for intraoperative stimulation to record nerve action potentials.

The skin incision varies with the extent of plexus exposure. Supraclavicular exposure begins with an incision across the upper border of the clavicle. This incision may be extended in a proximal direction parallel to the posterior border of the sternocleidomastoid muscle (Fig. 35-11). Skin flaps are elevated and supraclavicular sensory nerves are mobilized. The subcutaneous platysma muscle is divided. The external jugular vein, which descends across the posterior border of the sternomastoid to pierce the fascia, is usually ligated. The sternocleidomastoid muscle is partially released from the clavicle to widen the medial exposure. Similarly, the trapezius muscle is partially released from the lateral clavicle to enlarge the lateral exposure. The omohyoid muscle is the "door" to the supraclavicular plexus and must be identified. The omohyoid is divided at its intermediate tendon to expose the scalene muscles and plexus (upper and middle trunks). The transverse cervical artery, which crosses from anterior to posterior across the operative field just cephalad to the suprascapular artery, is mobilized and ligated. The phrenic nerve

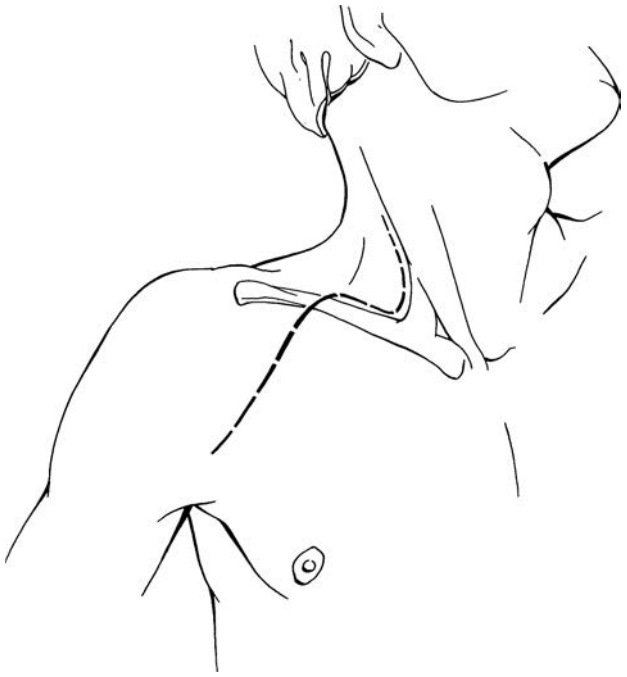

Figure 35-11 Artist's schematic of incision for approach to brachial plexus.

travels on the anterior scalene just anterior to the posterior triangle. The phrenic nerve must be protected throughout the dissection (Fig. 35-12). The upper trunk and C5 and C6 roots are isolated between the anterior and middle scalene muscles. The middle trunk and C7 is inferior and posterior to the upper trunk. The lower trunk is difficult to isolate via the supraclavicular approach, as the subclavian

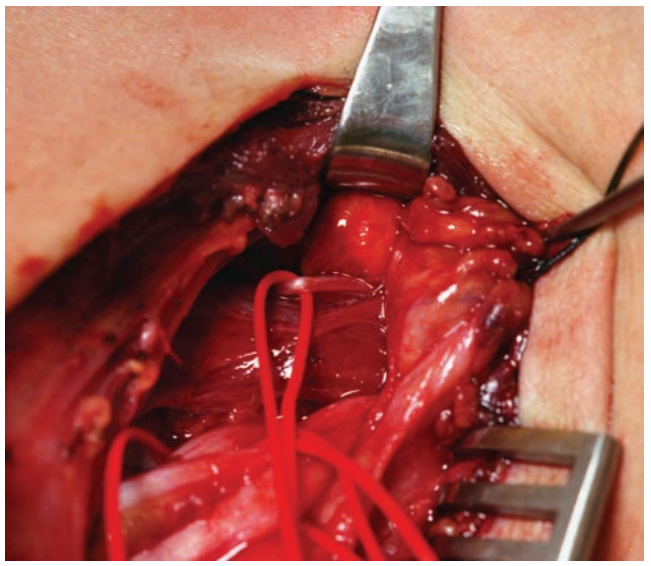

Figure 35-12 Phrenic nerve is isolated and protected. Nerve runs along anterior aspect of the anterior scalene. Patient is supine and head is to the right. (Courtesy of Shriners Hospital for Children, Philadelphia.)

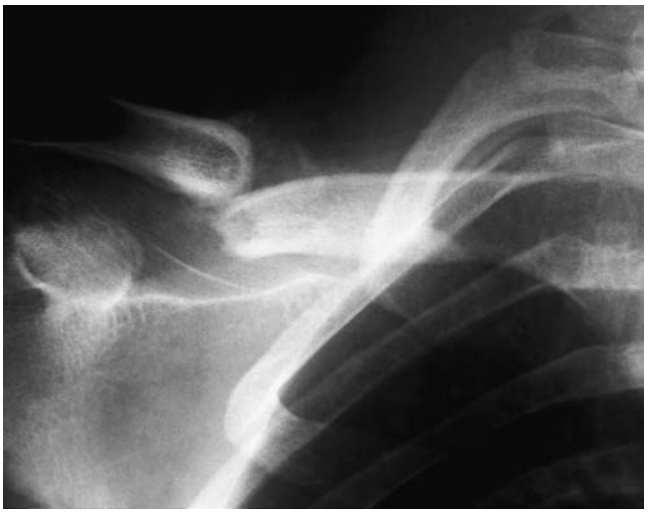

Figure 35-13 X-ray of 17-year-old male with right brachial plexus injury and clavicle malunion. (Courtesy of Shriners Hospital for Children, Philadelphia.)

vessels and clavicle impede inferior dissection. Occasionally, the lower trunk and C8 and T1 can be visualized. Osteotomy of the clavicle and/or infraclavicular exposure will enhance access to the lower trunk. Osteotomy of a clavicle malunion is often necessary to increase exposure of the underlying plexus (Fig. 35-13–35-17).

Combined supraclavicular and infraclavicular exposure necessitates extension of the lateral skin incision downward into the deltopectoral interval (see Fig. 35-11). Isolated infraclavicular brachial plexus exposure requires only a deltopectoral incision. The deltoid muscle is retracted in a lateral direction and the pectoralis major in a medial direction. The coracoid process is identified. The pectoralis minor tendon is the "door" to the infraclavicular plexus. The tendon is tagged with a suture and divided. The infraclavicular plexus

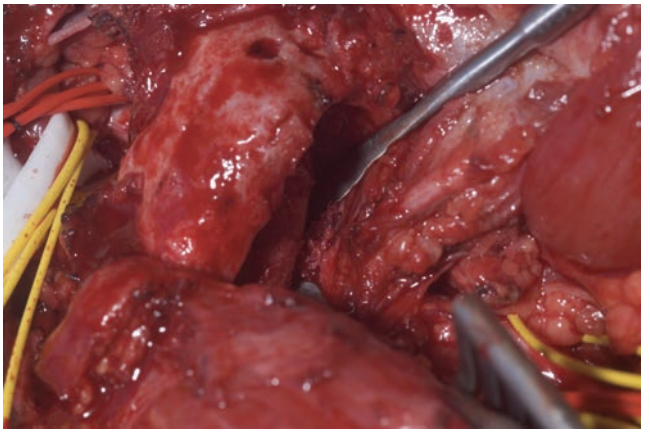

Figure 35-14 Exposure of clavicle with abundant callus. Clavicle prohibits adequate isolation of underlying brachial plexus. (Courtesy of Shriners Hospital for Children, Philadelphia.)

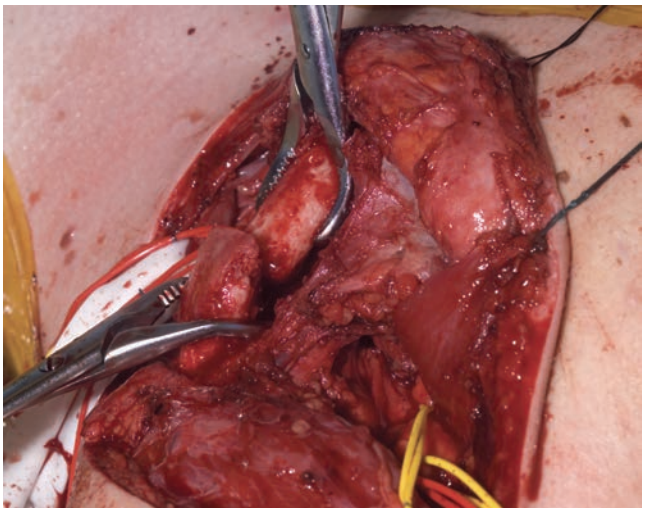

Figure 35-15 Clavicle malunion isolated and bone edges mobilized for better exposure of plexus. (Courtesy of Shriners Hospital for Children, Philadelphia.)

and axillary artery are isolated. The lateral cord (i.e., musculocutaneous and median nerves) is usually the first structure encountered. Identification if the posterior cord (i.e., axillary and radial nerves) and medial cord (i.e., ulnar nerve) requires mobilization of the axillary artery.

Surgical Strategy

The surgery for brachial plexus injury is tedious, as scar envelopes the injured neural elements. Following exposure of the brachial plexus, the injured neural elements are identified. The normal anatomy is often distorted and intraoperative nerve stimulation is helpful to identify specific nerve elements. Direct stimulation of the nerve and observation of any corresponding muscle contraction assists in proper nerve identification. The focus of the

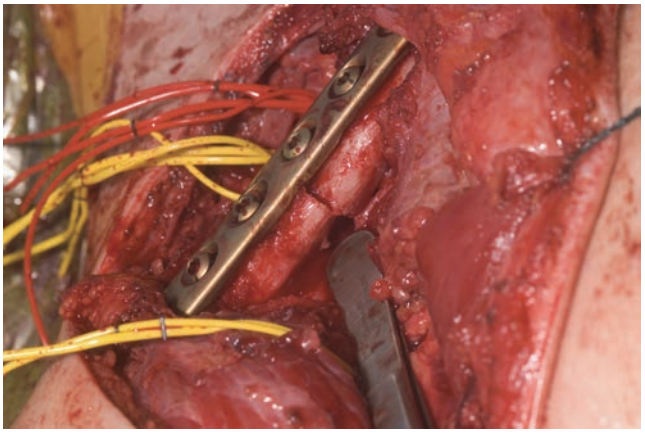

Figure 35-16 Fixation of clavicle in corrected position with dynamic compression plate. (Courtesy of Shriners Hospital for Children, Philadelphia.)

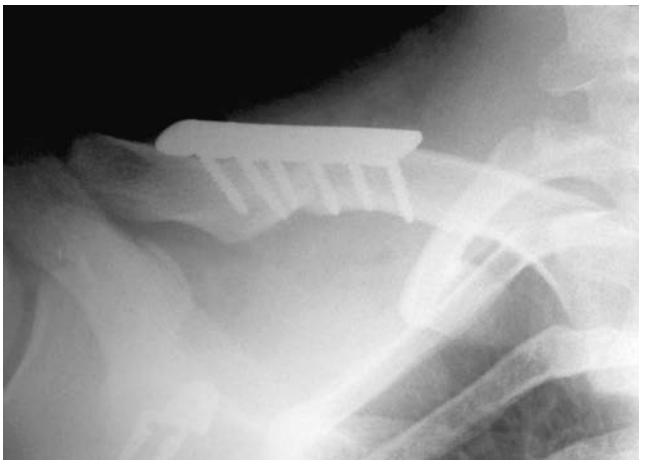

Figure 35-17 Postoperative x-ray reveals marked improvement in clavicle position. (Courtesy of Shriners Hospital for Children, Philadelphia.)

brachial plexus dissection is to differentiate lesions in continuity from those with lesions with loss of continuity. The diagnosis is uncomplicated when the dissection uncovers a transected nerve or avulsed nerve roots. The differentiation, however, can be difficult within dense scar tissue and neuroma formation that intervenes between the proximal and distal limbs of a completely disrupted nerve.[37] In addition, a neurotmesis lesion can occur from severe intraneural damage without overt transaction. This lesion will not recover without excision of the scarred nerve tissue and interposition grafting. Intraoperative determination of SEPs and NAP can facilitate the diagnosis in these equivocal cases, although their usefulness is controversial.

Stimulating a nerve and recording the cerebral cortical response indicates an intact SEP. The presence of a recordable response implies nerve continuity, and the absence of a response indicates nerve discontinuity. This technique can be applied to a spinal nerve to determine the presence of an avulsion injury. The absence of a response from a spinal nerve indicates an avulsion injury and is a contraindication to nerve grafting. The determination of the NAP across the neuroma may be helpful. The sensory NAP is recorded by direct stimulation and recording of the nerve proximal and distal to a suspected lesion. The presence of conduction across the lesion confirms some nerve continuity. Absent nerve conduction indicates fiber discontinuity with complete intraneural scarring.[90,118,122]

Treatment of Avulsion Injuries

Currently, there is no surgical treatment to restore nerve rootlet connection (i.e., an avulsion injury) with the spinal cord.[125] Experimental work with reattachment of the rootlets directly to the spinal cord is ongoing.[23,24] The current treatment for avulsion injuries is nerve grafting using

other viable proximal stumps or nerve transfer from intact adjacent nerves (see Fig. 35-10).

Treatment of Lesions in Continuity

The role of neurolysis for lesions in continuity is controversial. The ultimate effect on neurologic recovery is questionable. Neurolysis of a recovering nerve may induce further damage and reduce subsequent recovery. If neurolysis is performed and fascicular anatomy cannot be identified, resection and grafting is indicated.[118]

Treatment of Lesions without Continuity

After completion of the exposure and preliminary dissection, the lesions with loss of continuity are delineated. The proximal and distal stumps available for nerve reconstruction are defined with exclusion of any rootlet avulsions. The subsequent treatment is to resect the neuroma between the proximal and distal nerve stumps until normal nerve consistency and fascicular anatomy is encountered. The distal stumps are now primed for coaptation with viable inflow for regeneration of axons. The exact connection of proximal and distal stumps requires some decision making, especially in cases with loss of proximal stumps secondary to avulsion. Restoration of continuity can be accomplished by nerve grafting or nerve transfer. Nerve grafting is the placement of intercalary nerve grafts that act as scaffolding for regenerating axons. Nerve transfer is coaptation of an expendable motor nerve into the distal stump to supply axons for ingrowth.

Nerve Grafting

Nerve grafting is the preferred technique for most lesions with loss of continuity. The interposition of a nerve graft links the proximal and distal axons and serves as a conduit to channel the growing axons to the periphery. The donor nerve is usually the one or both sural nerves, harvested at the time of brachial plexus exploration. Other potential nerve graft options are the cervical plexus, ipsilateral medial antebrachial cutaneous, and/or superficial radial nerves. The donor nerve graft is divided into sections that span the defect until a comparable cross-sectional area is obtained for interposition grafting. The nerve grafts are secured between the proximal and distal stumps by epineural sutures and/or biologic adhesives, such as fibrin glue (Fig. 35-18).[47,90,125,126] Biologic adhesives such as fibrin glue can be used to decrease operative time.[57] The nerve grafts are revascularized by vascular ingrowth from the recipient bed.[144,164] Therefore, numerous smaller-diameter grafts are preferable to a single large-caliber graft, to limit necrosis before revascularization begins. Tension is avoided across the proximal and distal coaptation sites because this creates an unfavorable condition for axonal sprouting.[116,118,165] Nerve grafts are best directed toward shoulder and elbow muscles for reinnervation because the distance to the branches is short and axonal regrowth can occur before there is irreversible muscle damage.[125] In addition, the plexus fascicular anatomy shifts rapidly over relatively short distances, which promotes erroneous growth of axons. Therefore, nerve-grafting strategy should attempt to preferentially direct the axons to achieve a defined function. For example, grafts that connect directly to the musculocutaneous, axillary, and suprascapular nerves have less chance of axonal dropout and misdirection compared with grafts placed imprecisely into the cords.

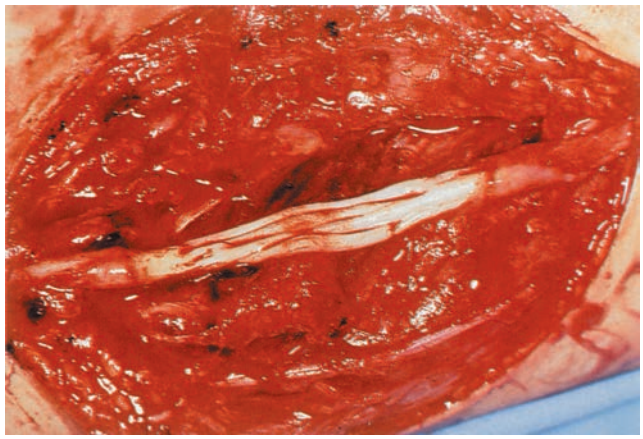

Figure 35-18 A 15-year-old boy, who sustained a gunshot wound to his arm, with transection of ulnar nerve. He was treated with neuroma resection and cable graft with strands of sural nerve.

Nerve Transfer

Nerve transfer is indicated in avulsion injuries and large or extensive defects of the brachial plexus. The indications for nerve transfer increases as the number of avulsed roots multiplies. In addition, long interposition nerve grafts (greater than 10 cm) are limited by available donor nerves and have less chance for recovery.[29]

Nerve transfer involves the connection of an expendable donor motor nerve to provide an axonal source for regeneration of the distal stump. The donor nerves for transfer are mobilized while maintaining their proximal connection and coapted to selected distal stumps, preferably specific peripheral nerves to achieve the desired function. The donor neurons will reinnervate the muscle, and function will require voluntary control of the transferred nerve. Similar to a tendon transfer, activation of the donor nerve to achieve function will require a period of training, and involuntary muscle activation may occur until transformation has taken place. For example, coughing or sneezing will initially activate intercostal nerve transfer, but re-education will occur over time. The technique of nerve transfer can be used in tandem with nerve grafting or may be the only available option in complete avulsion injuries.

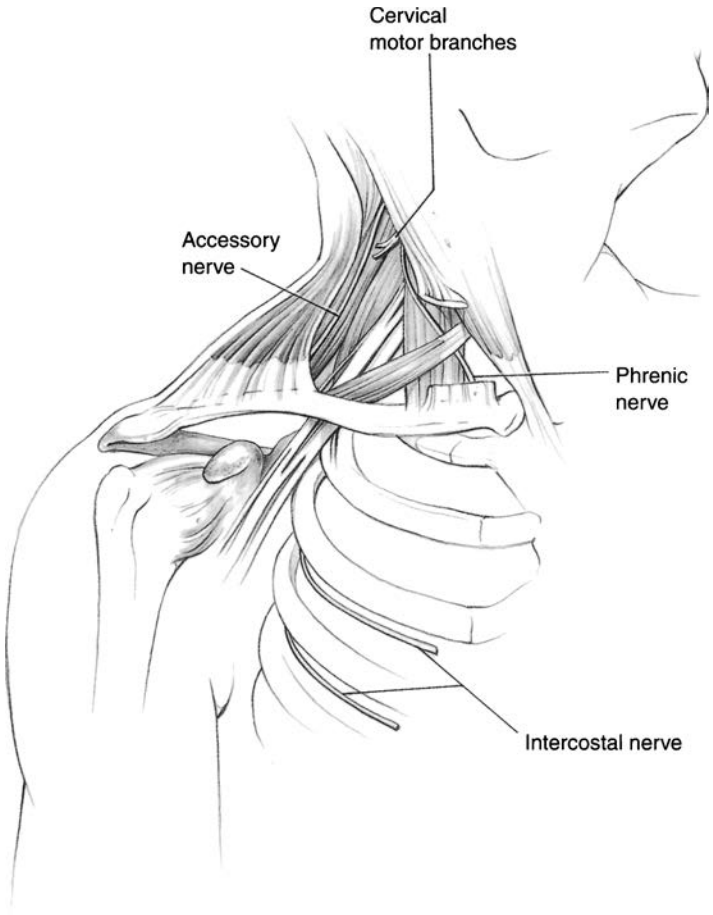

Figure 35-19 Artist's schematic of potential donors for nerve transfer.

Nerve transfers utilize a variety of axonal sources depending upon the level of injury. The number of available nerve transfers is increasing as our knowledge of muscle innervation and its redundancy improves. Options include the spinal accessory nerve, intercostal nerves, a portion of the ulnar nerve, a portion of the median nerve, a portion of the radial nerve, the medial pectoral nerve, the phrenic nerve, and the contralateral C7 nerve root (Fig. 35-19).[11,29,30,31,64,66,100,101,111,125,165,194] Recently, the ipsilateral C7 nerve root has been reported as a potential transfer in cases with isolated avulsion of C5 and C6.[65]

Nerve transfer of the phrenic nerve, the suprascapular nerve, the musculocutaneous nerve, or axillary nerve has been described.[29,30,31,125] Use of the phrenic nerve does reduce pulmonary capacity, which may improve over time.[3,64,125] In infants, the use of phrenic nerve transfer is avoided because the diaphragm is not yet firmly fixed to the vertebral bodies and severe respiratory problems can occur.[30,31]

Transfer of the spinal accessory to the suprascapular nerve for supraspinatus and infraspinatus reinnervation, transfer of the intercostal nerves to the musculocutaneous nerve for biceps function, and transfer of a portion of the ulnar nerve to the musculocutaneous nerve are favored techniques for avulsion injuries of C5, C6, and C7.[30,95,100,101,125,194]

Spinal Accessory Nerve Transfer Technique[11,70] (Fig. 35-20)

After general anesthesia, the patient is positioned on the operating table with a large folded sheet placed in the interscapular region to facilitate access to the trapezius muscle and spinal accessory nerve. The neck is turned to the contralateral side. Muscle relaxant is avoided to allow electrical stimulation of the spinal accessory nerve. A 10-cm transverse incision is made just above the clavicle from the sternocleidomastoid muscle to the acromioclavicular joint. The upper part of trapezius muscle that inserts onto the clavicle and the acromioclavicular joint is divided. The divided portion of the trapezius is retracted in a posterior direction. Dissection is performed along the anterior border of the trapezius muscle looking for the transverse cervical artery and vein. These vessels enter the trapezius muscle at the base of the neck and descend in a vertical direction midway between the vertebral column and the medial border of the scapula. The spinal accessory nerve

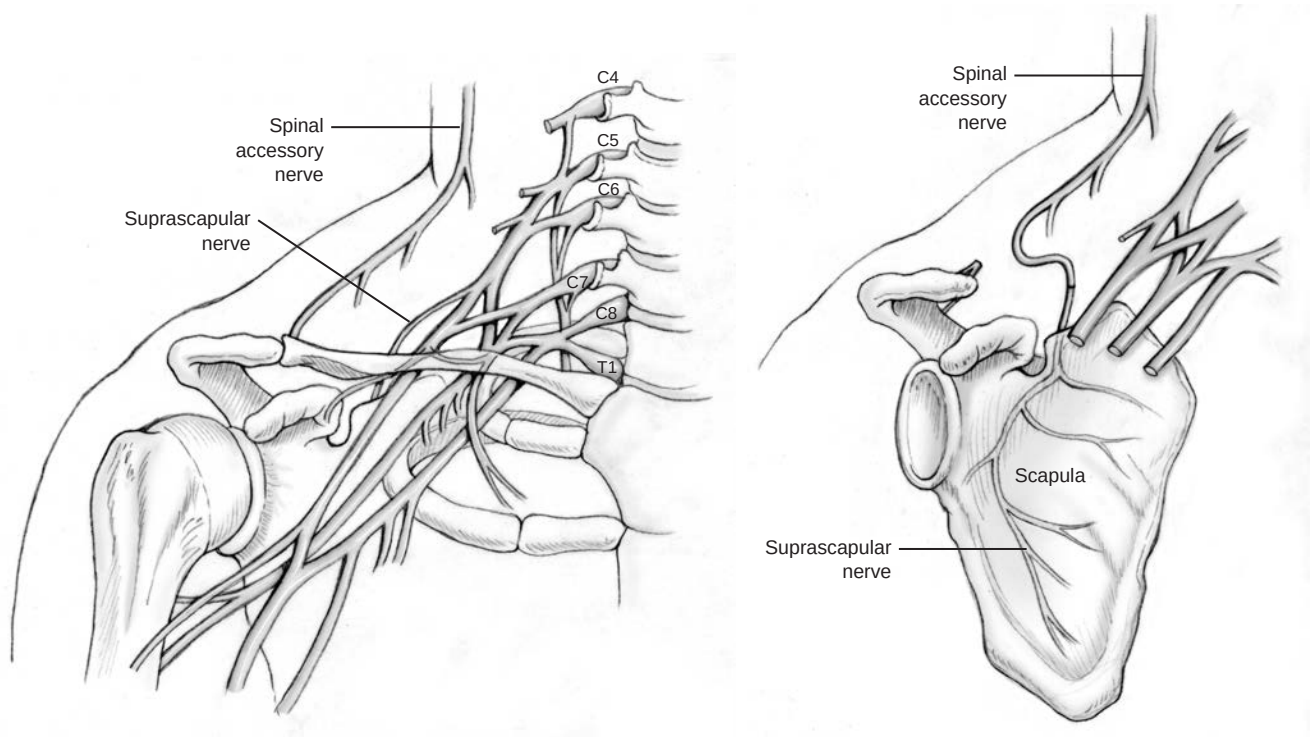

Figure 35-20 Schematic of the spinal accessory to the suprascapular nerve transfer technique.

accompanies the transverse cervical artery and vein. After identification of the vessels, an electrical stimulator is used around the vessels to facilitate identification of the distal part of the spinal accessory nerve. Small branches from the cervical plexus are located in the vicinity; however, electrical stimulation will not result in trapezius contraction.

The spinal accessory nerve is dissected in a distal direction to increase length. A proximal branch to the upper part of the trapezius must be preserved. The terminal division of the nerve is divided and transferred to the supraclavicular fossa for transfer to the suprascapular nerve, which is isolated from the upper trunk.

Intercostal Nerve Transfer Technique[95,119,197]

The intercostal transfer is performed through a transverse thoracic incision in the interspace between the third and fourth ribs, from the midaxillary line to the costochondral junction (Fig. 35-21). The anterior surface of the ribs is exposed by separation of the pectoralis major and minor muscles. The ribs are mobilized by subperiosteal dissection, with protection of the underlying pleura. An umbilical tape is placed around the nerve for traction. The rib is retracted in a cephalad direction and the upper portion of the intercostal muscle spread to identify the motor portion of the intercostal nerve. The nerve is dissected from the costochondral junction to the midaxillary line. This length of dissection will avoid the need for an intervening nerve graft.[184] The third, fourth, and fifth intercostal nerves are harvested in similar fashion (Fig. 35-22). Through a linear incision in the arm, the musculocutaneous nerve is exposed on the undersurface of the biceps brachii muscle. A subcutaneous tunnel is developed between the biceps, axilla, and thoracic incision. The intercostal nerves are then passed to the biceps motor nerve and coapted with epineural sutures and/or fibrin glue.

When transferring the intercostal nerves, two to four nerves are used to provide increased motor fibers to the musculocutaneous nerve, compared with a single-nerve transfer.[28,83,95,119,125] Early reinnervation will be evident during breathing, coughing, and sneezing, which will illicit biceps activation. This involuntary reaction will subside over time as volitional activation becomes predominant.[95,119,122] Intercostal nerve transfer will have a minimal effect on pulmonary function as long as the diaphragm is functional. Diaphragmatic paralysis is a relative contraindication to intercostal nerve transfer.[95,116] In addition, combined transfer of the intercostal and phrenic nerves is not recommended.

Ulnar Nerve Transfer Technique[11,100,101,131,177,194] (Fig. 35-23)

An incision is performed over the medial aspect of the arm, beginning 4 cm distal to the pectoralis major lateral border

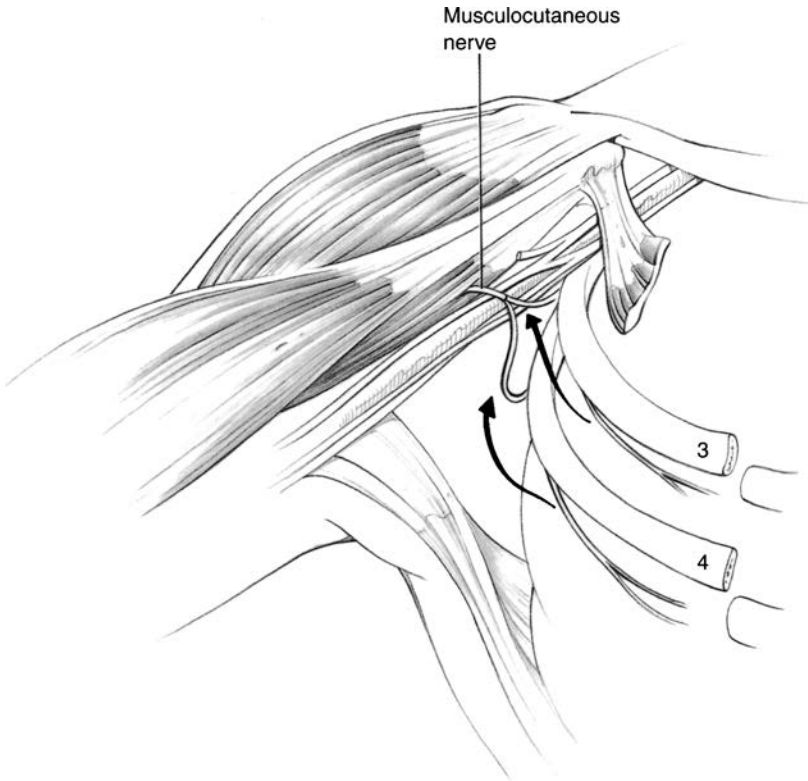

Figure 35-21 Artist's schematic of intercostal nerve transfer to musculocutaneous nerve for elbow flexion.

(Fig. 35-24). The anterior fascia over the biceps muscle is divided and the muscle is dissected from the coracobrachialis muscle. The motor branch of the biceps muscle is identified on the undersurface of the muscle and traced in a proximal direction toward the musculocutaneous nerve. The motor nerve is divided from the musculocutaneous nerve in preparation of transfer.

A second fascia incision is performed posterior to the intermuscular septum and the ulnar nerve is isolated 1 cm proximal to the motor branch of the biceps muscle. Using magnification, a longitudinal epineurotomy is made along the ulnar nerve and the individual group fascicles separated (Fig. 35-25). Usually, three group fascicles are present. Electrical stimulation is used to confirm group fascicles that contain motor fibers to the extrinsic hand muscles. One group fascicle is divided and transferred to the biceps motor branch. The ulnar nerve is coapted to biceps motor branch with epineural sutures and/or fibrin glue. The nerve coaptation is performed with the elbow in extension and the elbow immobilized in flexion to relieve any tension across the nerve repair.

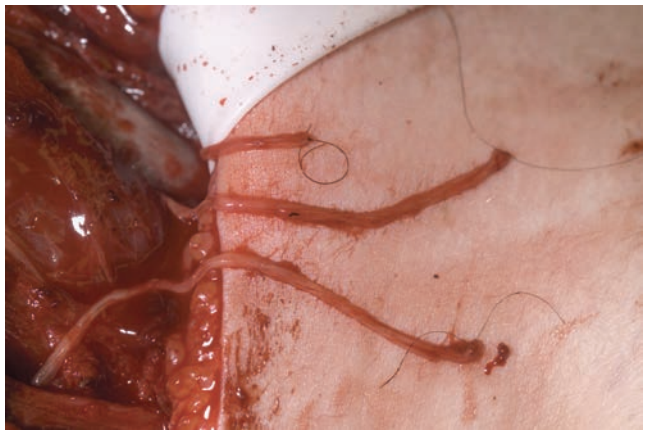

Figure 35-22 Three intercostal nerves harvested for transfer to the musculocutaneous nerve. (Courtesy of Shriners Hospital for Children, Philadelphia.)

Prioritization in Severe Brachial Plexus Injuries

In each microsurgical case, the reconstruction plan is individualized depending on the extent of injury and available reconstructive options. A priority list must be established in difficult brachial plexus injuries. This roster is based on functional importance and prognosis after nerve reconstruction. Better results are achieved from nerve reconstruction in the proximal musculature. Elbow flexion against gravity holds the highest priority in brachial plexus injuries. Restoration of elbow flexion allows the extremity to be better positioned in space and the hand flexed toward the trunk and mouth for use. Shoulder balance and stability is the next priority for reconstruction, as an unstable or contracted shoulder will impede use of the extremity. The triceps is next on the priority list, for nerve grafting or transfer is reliable by connection directly to the radial nerve

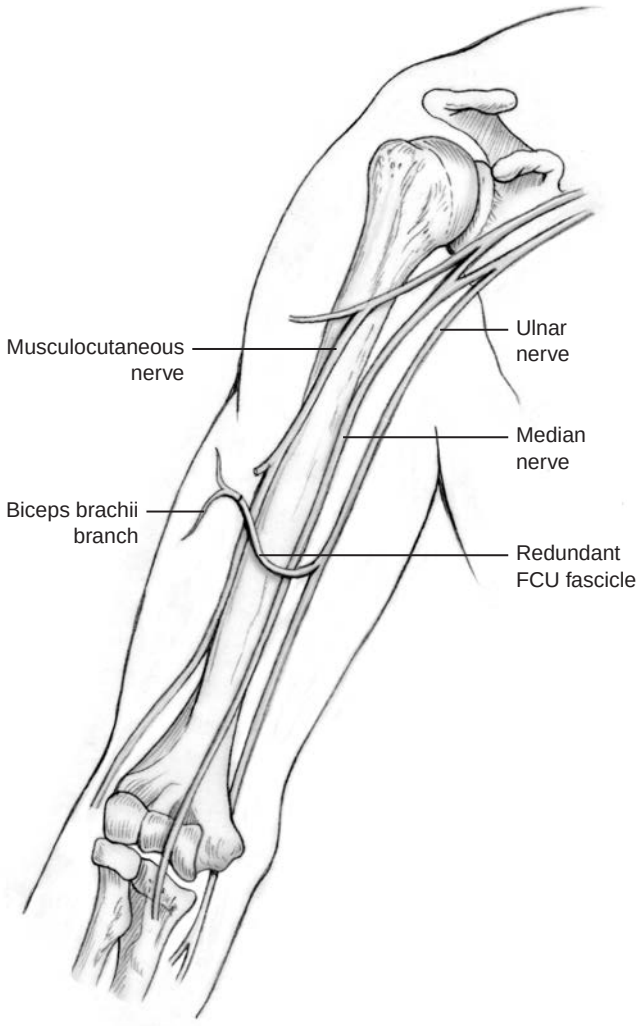

Figure 35-23 Schematic of the transfer of a portion of the ulnar nerve to the musculocutaneous nerve for biceps function.

branches that innervate the triceps. The forearm flexors and digital sensibility (median nerve) are addressed next. The wrist and finger extensors are subsequently considered by linkage to the radial nerve. The ulnar innervated structures are the last priority because of the poor prognosis for recovery.[124–126] The number of axons diminishes as the regenerating axons move toward the periphery, and the hand has the misfortune of being last on the list. Therefore, reconstruction of lower-trunk defects via nerve grafts in adults is usually unsuccessful and is performed only in children that possess the possibility of reinnervation. Nerve transfers, however, are creating options for distal reinnervation as the regeneration distance is diminished.[194]

The priority list provides guidelines for nerve reconstruction in severe cases with limited viable proximal stumps or inadequate donor nerves. The unlimited patterns of nerve lesions in brachial plexus injuries prohibit the development of definitive treatment algorithms for

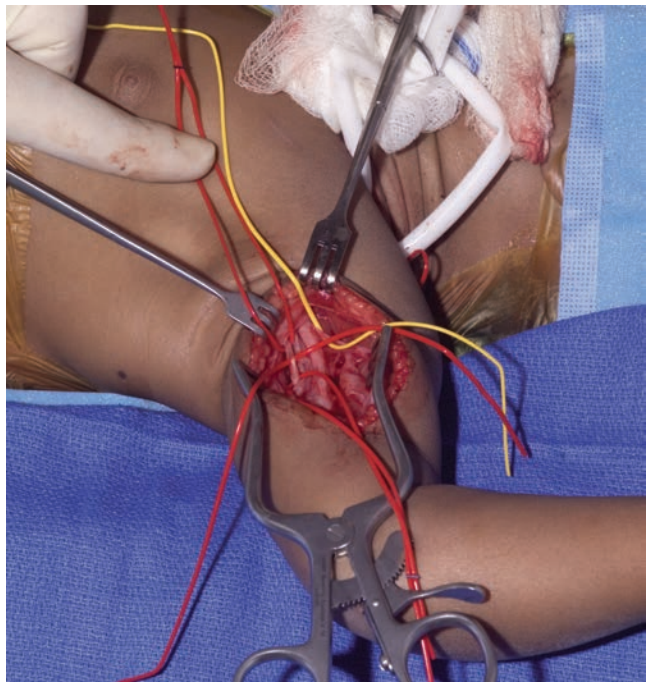

Figure 35-24 Incision over the medial aspect of the arm to identify musculocutaneous and ulnar nerves. (Courtesy of Shriners Hospital for Children, Philadelphia.)

each potential situation. The basic strategy is to precisely define the injury and reconstruct loss of nerve continuity (see Fig. 35-10). Nerve grafts are employed in lesions with viable proximal and distal stumps, with priority given to the elbow (musculocutaneous nerve) and shoulder (suprascapular and axillary nerves). Inadequate proximal

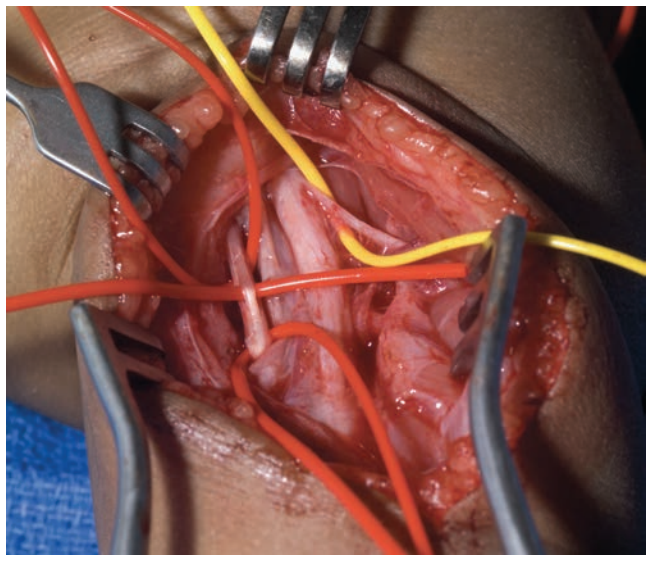

Figure 35-25 Isolation of a group fascicle from the ulnar nerve (*red loop*) for transfer to the musculocutaneous branch to the biceps muscle (*yellow loop*). (Courtesy of Shriners Hospital for Children, Philadelphia.)

inflow requires nerve transfer with expendable donors. In addition, a combination of nerve grafts and nerve transfers can be utilized during brachial plexus reconstruction.

Postoperative Care

The closure is straightforward with repair of divided structures. An osteotomy of the clavicle requires internal fixation. The omohyoid and/or pectoralis minor tendon are sutured. A layered closure is completed and the arm is placed in a Velpeau dressing. Suture removal is at 2 weeks and immobilization continued for 3 to 4 weeks. Gradual mobilization and physical therapy are instituted within the confines of nerve mobilization. Prolonged therapy is necessary to maintain supple joint structure, as nerve regeneration progresses at a sluggish rate of 1 mm/day. Serial clinical and electrodiagnostic evaluations are performed with assessment for signs of reinnervation. Motor recovery is followed by active motion to restore function of the extremity. Sensory recovery may benefit from sensory re-education.

Results after Brachial Plexus Surgery

The results from nerve reconstruction surgery vary with the degree of damage and complexity of the techniques employed. A result is judged to be successful when the outcome improves function, and this usually requires a strength return greater than gravity. This antigravity strength allows independent use of the extremity without assistance of the other extremity.

Neurolysis and Primary Neurorrhaphy

The reported results of neurolysis of lesions in continuity are generally good, with return of muscle strength against gravity in more than 90% of the cases.[38,47,91,92,133] The efficacy of neurolysis, however, is difficult to prove as spontaneous recovery may have occurred without surgery.[111,133]

Primary neurorrhaphy is usually not an option in brachial plexus injury.[47] However, when primary repair is indicated and is performed without excessive tension, most patients regain strength to overcome gravity.[29,90,91]

Nerve Grafting

The results of nerve grafting are better with upper plexus lesions, shorter graft length, and surgery less than 9 months after injury.[29,38,71,165] Return of sufficient proximal muscle strength after nerve graft interposition occurs in approximately 50% to 80% of cases.[4,38,52,90,111,122,123,182] Nerve grafting for lower plexus lesions in adults is uniformly unsuccessful, but may provide some mild return of extrinsic muscle function and protective sensation.[4,18,90,91,116,165] Secondary tendon transfers and arthrodesis can further enhance function after nerve regeneration.

Nerve Transfer

The results of nerve transfer reveal better results when the transfer is performed early and coapted into a peripheral nerve without an intervening graft.[29,30,95,119,122] A variety of donor nerves have been utilized, including the intercostals, partial median, partial ulnar, spinal accessory, and phrenic. Return of elbow flexion against gravity is achieved in 60% to 88% of cases.[29,30,64,78,83,116,119,182] The ulnar nerve to motor branch of the biceps muscle has resulted in consistent elbow flexion against gravity in approximately 75% of cases.[52,101,113,117,123,131,189] Persistent clinical deficits in ulnar nerve function have not been realized.

The results for return of shoulder function are more variable when using a nerve transfer to the suprascapular nerve or axillary nerve to achieve stability and abduction. The results of transfer for suprascapular function are approximately 50% to 80% successful and axillary function about 40% successful.[31,106,116,118,125] The inferior results for restoration of shoulder function compared with elbow flexion are, in part, secondary to mass innervation.[126] Shoulder function is a coupled movement of scapulothoracic and glenohumeral motion to achieve abduction. Simultaneous contraction of the reinnervated shoulder muscles prevents synchronous motion and diminishes effective abduction. In contrast, elbow flexion requires a relatively uncomplicated motion.

A meta-analysis of the English literature to assess the efficacy of individual nerve transfers for restoration of elbow and shoulder function uncovered 1,038 nerve transfers from 27 studies that met the inclusion criteria.[113] Seventy-two percent of direct intercostal-to-musculocutaneous transfers (without interposition nerve grafts) achieved biceps strength greater than or equal to M3 versus 47% using interposition grafts. Direct intercostal transfers to the

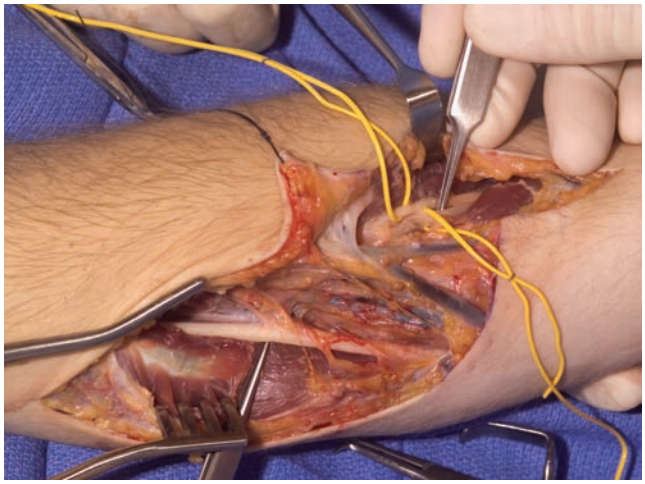

Figure 35-26 A 17-year-old male with posterior cord injury. Isolation of the radial and median nerves within the forearm. (Courtesy of Shriners Hospital for Children, Philadelphia.)

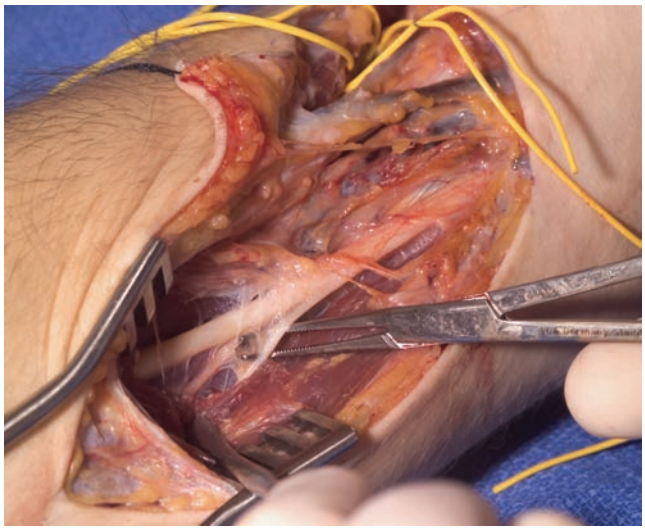

Figure 35-27 Isolation of a redundant FDS branch of the median nerve for transfer to the posterior interosseous nerve (*yellow loop*). (Courtesy of Shriners Hospital for Children, Philadelphia.)

musculocutaneous nerve had a better ability to achieve greater than or equal to M4 elbow strength than transfers from the spinal accessory nerve (41% vs. 29%). The suprascapular nerve fared significantly better than the axillary nerve in obtaining greater than or equal to M3 shoulder abduction (92% vs. 69%).

Nerve transfer for forearm and hand function is less successful, with partial return of function.[83] The use of donor nerves within the forearm, however, may improve the clinical results (Figs. 35-26 and 35-27). Partial median and anterior interosseous nerve transfers to the posterior interosseous and ulnar motor nerve are examples of such transfers.[7,194]

The results of contralateral C7 nerve transfers have been somewhat disappointing. Fortunately, transient weakness and numbness has not persisted into permanent deficits. Sensory and extrinsic motor recovery has been reported in approximately one-third of patients at 3-year follow-up.[66,171]

BRACHIAL PLEXUS BIRTH PALSIES

Pathophysiology

Brachial plexus birth palsies can occur during passage through the birth canal. Traction or stretch across the plexus is the likely mechanism, although the exact timing of stretch is controversial.[45,84,126] The reported incidence has varied from 0.4 to 2.5 palsies per 1,000 live births.[34,63] Brachial plexus injuries have been recreated in stillborns by traction applied to a restrained shoulder.[114] Brachial plexus birth palsies can also occur during caesarean section or breech presentation.[34,97]

Evaluation of Brachial Plexus Birth Injury

The history is extremely important. Prenatal, postnatal, and birth information should be obtained. Risk factors include a difficult or prolonged delivery, large-for-gestational age infants (macrosomia), shoulder dystocia, breech presentation, and forceps or vacuum extraction.[34,53,57,92] Inquiry into the duration of labor, method of delivery, use of forceps or vacuum extraction, fractures of the clavicle or humerus, Horner's syndrome, postnatal position of extremity, and initial extent of involvement is required.

The physical examination of the newborn with brachial plexus birth palsy is difficult because of lack of cooperation. Observation of the affected extremity often provides valuable information regarding the pattern of injury (Fig. 35-28)(see Table 35-3). Upper root (C5, C6) or trunk injuries (i.e., Erb's palsy) present with a typical posture of shoulder adduction and internal rotation, elbow extension, forearm pronation, wrist flexion, and finger flexion. An extended Erb's palsy includes C7 and additional paralysis of elbow and finger extension. Total or global brachial plexus palsy results in a completely flaccid extremity. Isolated lower root (C8, T1) or trunk injuries (i.e., Klumpke's

Figure 35-28 A 6-month-old child with left brachial birth palsy. Posture is indicative of upper trunk palsy with early return of elbow flexion. (Courtesy of Shriners Hospital for Children, Philadelphia.)

palsy) are rare in birth injuries. When present, there is normal posture of the shoulder and elbow, with paralysis of the hand's intrinsic muscles.[34,72] The presence of a Horner's syndrome (ptosis, meiosis, enophthalmos, and ipsilateral facial anhydrosis) is an important observation that implies avulsion injury of the lower trunk, with disruption of the communicating branch supplying sympathetic fibers to the cervicothoracic ganglion. These infants may have an associated phrenic nerve palsy with an elevated hemidiaphragm, which increases the likelihood of an avulsion injury and negates the possibility of spontaneous recovery.

Palpation of the clavicle and shoulder girdle may reveal crepitation or abundant callus formation. An acute fracture will inhibit voluntary movement and mimic brachial plexus injuries (i.e., pseudoparalysis). In addition, the passive shoulder range of motion should be assessed to exclude posterior dislocation. A newborn with a brachial plexus injury should exhibit full passive motion of the shoulder. Limited external rotation implies a concomitant shoulder dislocation.[48] Ultrasound or MRI may be required to confirm this diagnosis because the newborn humeral head is not ossified.[94,121,186,187]

The assessment of motor function in the newborn or infant is a challenging task that requires patience and diligence. The initial goal is to determine the absence or presence of function, as actual grading of muscle strength is impossible. A variety of motor scores have been developed to assess movement (Table 35-8).[33,34,41] In the infant, a collection of toys and rattles is necessary to incite movement of the injured extremity. Tactile stimulation and changing the position of the child to assess weak but functioning muscle can alter the effect of gravity. Motor function for shoulder movement, elbow motion, wrist action, and digital movement is delineated. The newborn can also be aroused by the use of neonatal reflexes, such as the Moro, asymmetric tonic neck, and grasp reflex. The Moro or startle reflex should produce extensor tone in the legs and arms. The grasp reflex should illicit finger flexion via stimulation of the palm. Asymmetry of these reflexes indicates a neurologic deficit. Sensory function is also difficult to assess, as the only reaction obtained may be to painful stimuli, which will erase any chance of further cooperation.

Imaging Studies

Similar imaging studies are used in children and adults. The particular studies ordered vary with the type of injury, physical examination, imaging strategy, and expertise of the radiologist.

Plain x-ray films of the cervical spine, clavicle, shoulder, and chest are routine. Comparable findings suggest particular levels of plexus injury (Table 35-7).

Methods to determine the presence or absence of a pseudomeningocele are less reliable in children (see Fig. 35-9). Myelography, CT myelography, and MRI have been used for assessment. MRI imaging protocols are being developed using adult head coils, smaller slice thickness, and variable-imaging sequences to increase accuracy.[134,170]

Electrodiagnostic Studies

Electrodiagnostic testing is less reliable in children. The presence of motor activity within a muscle has not been an accurate prediction of clinical recovery. Electrodiagnostic testing often underestimates the injury and overestimates the chances of spontaneous recovery.[185]

TABLE 35-8
HOSPITAL FOR SICK CHILDREN ACTIVE MOVEMENT SCALE (AMS)

Shoulder abduction	_____
Shoulder adduction	_____
Shoulder flexion	_____
Shoulder external rotation	_____
Shoulder internal rotation	_____
Elbow flexion	_____
Elbow extension	_____
Forearm pronation	_____
Forearm supination	_____
Wrist flexion	_____
Wrist extension	_____
Finger extension	_____
Thumb flexion	_____
Thumb extension	_____

Gravity eliminated	Score*
No contraction	0
Contraction, no motion	1
<50% motion	2
>50% motion	3
Full motion	4
Against gravity	
<50% motion	5
>50% motion	6

*A score of 4 must be achieved before a higher score can be assigned. Movement grades are within available range of motion. Adapted from Clarke HM, Curtis CG. An approach to obstetrical brachial plexus injuries. *Hand Clin* 1995;11:563–580.

Nonoperative Treatment

Brachial plexus birth palsies are also treated by a period of observation. The diagnostic evaluation is similar to adult injuries, but complicated by lack of cooperation and small size of the infant (Fig. 35-29). Most brachial plexus birth palsies involve the upper trunk and resolve over time.[62,63,72] However, estimates of complete resolution have been downgraded from 90% to approximately 60% to 70%.[75,142] Permanent sequelae can be a debilitating problem that disturbs synchronous motion and hinders activities of daily living. Impairment about the shoulder is particularly prevalent, especially in external rotation and abduction.

The goals of the observational period are similar in adults and children. The objective is to determine the level and extent of injury. Serial examinations every month during infancy are necessary to forecast outcome and indications for surgical intervention. The number of nerve roots involved is a predictor of spontaneous recovery. Therefore, spontaneous recovery is greatest in Erb's palsy and least in global palsies.

Upper-trunk palsies with or without middle-trunk involvement are treated differently than global palsies. Elbow flexion (biceps and brachialis muscles) is the best indicator of upper trunk recovery. Consequently, the evaluation of elbow flexion is a key indicator of nerve regeneration across the injured segment. Those infants that recover antigravity elbow flexion within the first 2 months of life usually have a full and complete recovery over the first 1 to 2 years of life.[15,57,62,63,97,192] Recovery of elbow flexion between 3 and 6 months of injury infers an axonotmesis injury with subsequent nerve regeneration. The prognosis for recovery is guarded and secondary tendon transfers are often required. Infants who do not recover antigravity biceps strength by 5 to 6 months of life are candidates for microsurgical reconstruction, as successful surgery will result in a better outcome than natural history alone.[13,57,192]

Global birth palsies have a poor prognosis for spontaneous recovery. There is usually a combination of ruptures and avulsions. The outlook is poor without attempts at nerve reconstruction. Surgery is recommended at 3 months of age to maximize the available time for regeneration and reinnervation. Although root avulsions are common, there is almost always at least one root remaining that can be used for reconstruction.[57,68,72] In addition, alternative sources of axons may be available to reconstruct the plexus.

Surgical Management

Indications

Surgery for upper- and/or middle-trunk birth palsies without spontaneous recovery of elbow flexion is performed between 6 and 8 months of age (see Fig. 35-29). Surgery for global birth palsies without spontaneous recovery is performed at 3 months of age.

Surgical Approach/Strategy

The surgical exploration and strategy for brachial plexus birth palsies is similar to adult injuries. An exposure of the supraclavicular and/or infraclavicular plexus is required. Ruptures usually involve the upper trunk and are treated with neuroma resection and nerve grafting.[72,97,192] In the typical upper-trunk rupture, sural nerve grafts are performed from the C5 and C6 roots to the following structures: (a) upper trunk anterior division/lateral cord/musculocutaneous nerve, (b) suprascapular nerve, and (c) upper-trunk posterior division/posterior cord/axillary–radial nerves (Fig. 35-30).

Root avulsions usually involve the lower trunk and are treated by nerve grafting using adjacent roots and/or nerve transfers (e.g., ulnar nerve fascicle to the motor branch of biceps muscle). In infants, phrenic nerve transfer is typically avoided for fear of respiratory compromise.[28] In addition, a combination of nerve grafting and nerve transfers may be used to reconstruct the plexus. In children, nerve reconstruction for lower-trunk injuries is attempted because youth favors nerve regeneration and recovery is possible.[68,72164] Also, salvage procedures that include arthrodesis are avoided in the skeletally immature child.

Results

Similar to adults, there is a limited role for neurolysis in the treatment of brachial plexus birth palsies.[22,34,97,192,202] The recovery of muscle strength results with nerve grafting is superior to neurolysis. Clarke and colleagues[22,34] reported better long-term results after resection and grafting of both conducting and nonconducting neuromas compared to neurolysis, despite an initial deterioration of function with resection.

The results of surgery for brachial plexus birth palsies are more difficult to extract from the literature because complex evaluation is hampered in the child and recovery can be prolonged.[34,57,72,167] In addition, few patients have long-term microsurgery follow-up without secondary surgery. Upper-plexus (C5 and C6) injuries treated with neuroma resection and grafting produce encouraging results, with good recovery of shoulder function in 60% to 80% and reliable return of biceps in approximately 80% to 100%.[15,57,72,97,178] Unfortunately, global palsies have a more pessimistic outcome and more variable recovery.[57,68,72,202] Additional functional improvement can be obtained by secondary surgeries, such as tendon transfer and osteotomy.[27,56,88,93]

Shoulder Problems after Brachial Plexus Birth Palsies

Residual shoulder problems are universal after incomplete recovery of brachial plexus palsy. Incomplete recovery of the upper trunk lesions leads to muscle imbalance with

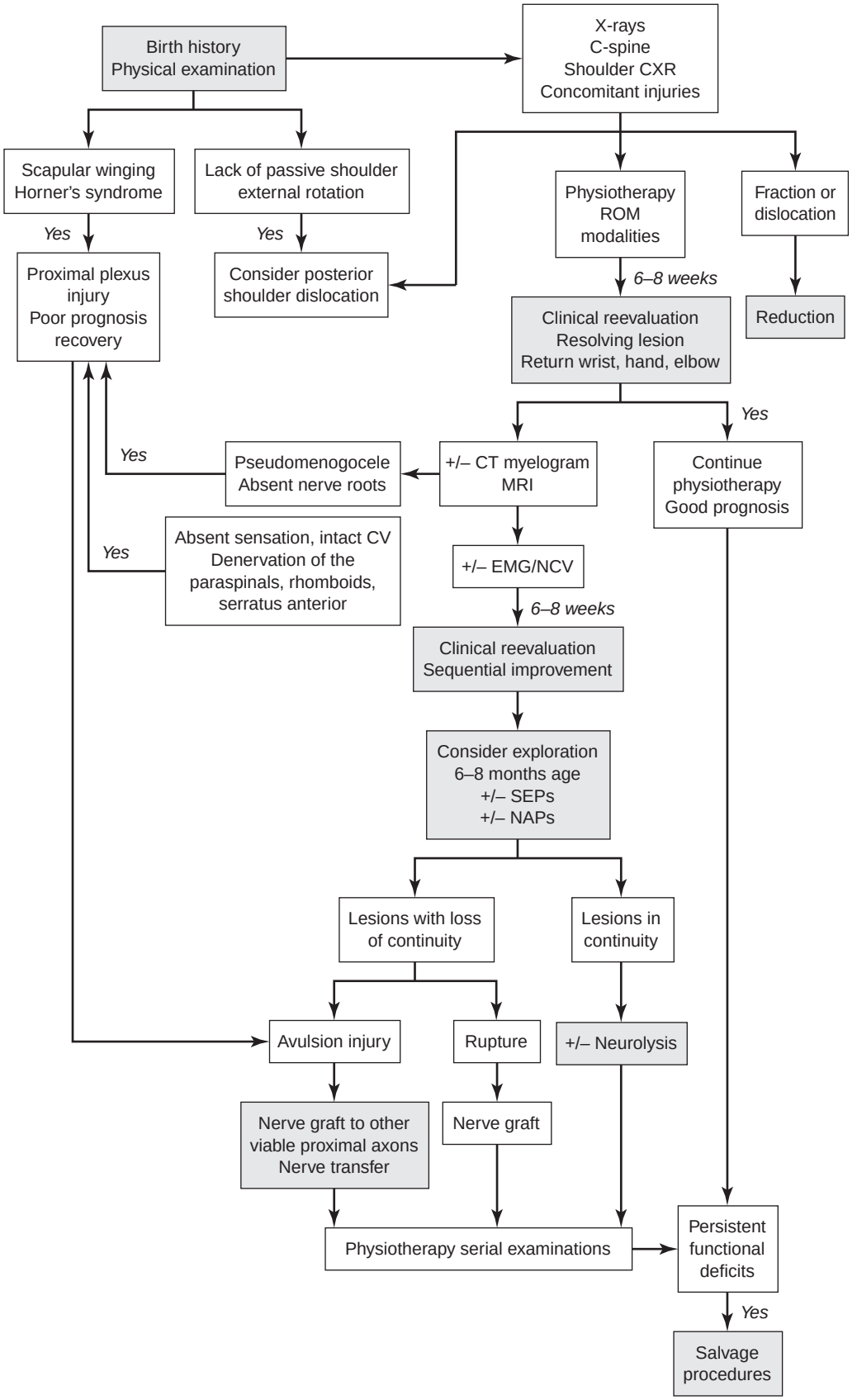

Figure 35-29 Treatment algorithm for brachial plexus birth injuries.

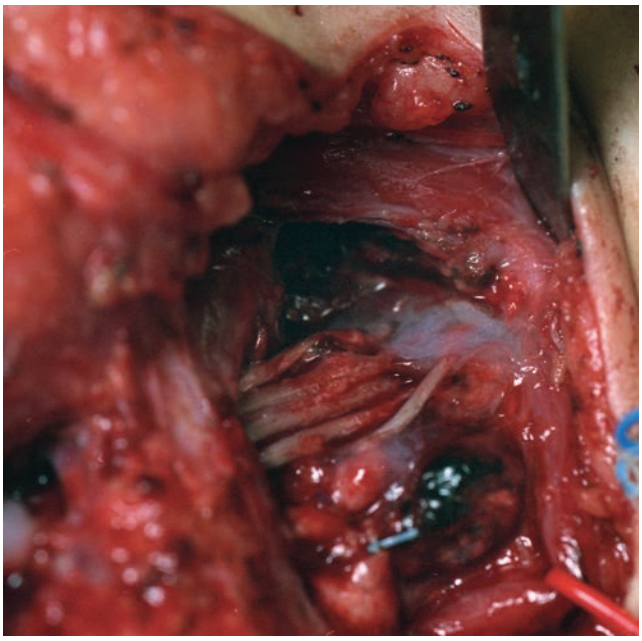

Figure 35-30 A 6-month-old child with left brachial birth palsy. Supraclavicular exposure revealed ruptures of C5 and C6. Sural nerve grafts to upper-trunk anterior division, upper-trunk posterior division, and suprascapular nerve secured with fibrin glue. Child is supine and head is to the right. (Courtesy of Shriners Hospital for Children, Philadelphia.)

strong internal rotators and weak external rotators, which results in an internal rotation contracture that is detrimental to glenohumeral joint development.[76,94,137,168,186,191] The constant position of internal rotation leads to early glenohumeral joint deformity by 6 months of age and advanced deformity by 2 years, which is characterized by increased glenoid retroversion and posterior humeral head subluxation.[10,47,77,94,121,191,193] To achieve hand-to-mouth function, compensatory shoulder or scapulothoracic abduction is required (Fig. 35-31). This abduction allows the hand to be delivered to the mouth despite persistent shoulder internal rotation, but appears abnormal and is inefficient for feeding. In severe deformity, the shoulder may dislocate in a posterior direction (Fig. 35-32), or when the child cannot obtain 80 degrees of abduction and has an internal rotation contracture greater than 45 degrees, hand-to-mouth function is impossible.[195]

Classification and Imaging

The glenohumeral deformity is progressive without management (Table 35-9).[191] The deformity evolves sequentially by grade from normal (I) to increased glenoid retroversion (II); to posterior glenohumeral subluxation with posterior glenoid dysplasia (III); to development of a false glenoid (IV); to flattening of the humeral head and glenoid (V). In addition, on occasion a true infantile glenohumeral joint dislocation (VI) occurs with immediate loss of passive motion.

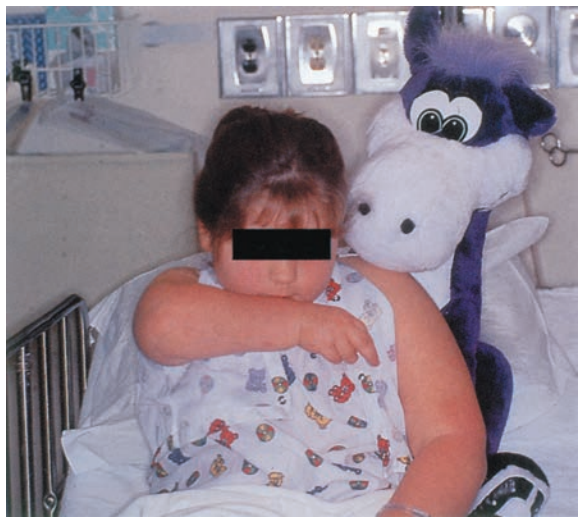

Figure 35-31 An 8-year-old child with impaired hand-to-mouth function from residual brachial plexus birth palsy and lack of external rotation.

MRI is our preferred modality to evaluate the pediatric glenohumeral joint.[94] MRI visualizes the articular cartilage without ionizing radiation and allows a true assessment of glenoscapular angle and humeral head position. Plain x-rays fail to visualize the cartilaginous structures and do not show the early changes of the unossified glenoid. This deficiency results in a misinterpretation of humeral head position and glenoid version.[67,137]

CT scanning can be used in the older child after substantial ossification of the humeral head and glenoid. Recently, ultrasound has been described as an alternative imaging modality to assess shoulder congruity without the

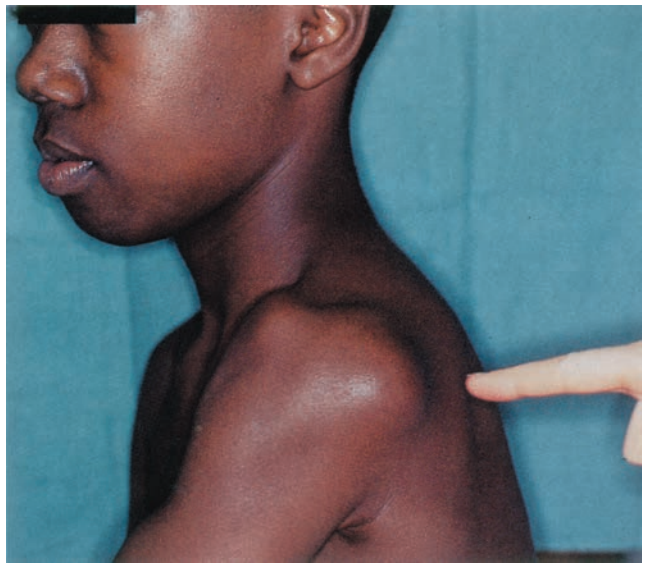

Figure 35-32 A 10-year-old boy with posterior shoulder dislocation from brachial plexus birth palsy and unbalanced muscle pull.

TABLE 35-9
CLASSIFICATION OF GLENOHUMERAL JOINT DEFORMITY

Type or Grade	Glenohumeral Joint
I	Normal glenoid (<5° retroversion compared to contralateral normal)
II	Minimal deformity (<5° retroversion compared to contralateral normal)
III	Moderate deformity (posterior humeral head subluxation)
IV	Severe deformity (posterior humeral head subluxation with false glenoid)
V	Severe flattening of humeral head ± complete dislocation
VI	Infantile glenohumeral dislocation
VII	Proximal humeral growth arrest

Adapted from Waters PM, Smith GR, Jaramillo D. Glenohumeral deformity secondary to brachial plexus birth palsy. *J Bone Joint Surg Am* 1998;80:668–677.

need for anesthesia.[121,158] The diagnostic accuracy of ultrasound appears lower than MRI or CT and the specific glenoid morphology is not visualized.[158]

In an attempt to standardize the cross-sectional slice chosen for measurements, certain criteria for image selection have been established.[94] The axial image selected should be inferior to the coracoid apophysis and spinoglenoid notch. The slice should visualize the subscapularis tendon and often include the tip of the coracoid. Using this image, the glenoscapular angle (the degrees of version) and the percent of humeral head anterior to the middle of the glenoid fossa (PHHA) are measured (Figs. 35-33 and 35-34).[54,147,191]

A line is drawn along the labral surfaces of the glenoid connecting the anterior and posterior margins. A bisecting line is constructed along the axis of the scapula that connects the medial margin of the scapula and the middle of the glenoid fossa. The posterior medial quadrant angle is calculated. Ninety degrees are subtracted from the angle measured to calculate the glenoid version. A negative value indicates glenoid retroversion and a positive value designates glenoid anteversion. In cases with a pseudoglenoid (humeral head articulating with a posterior articular concavity that is retroverted in relationship to the original glenoid), the posterior concavity was measured as the angle of version (Fig. 35-35).[94,137] The PHHA is calculated by measuring the humeral head distances perpendicular to the line along the axis of the scapula. The distance the humeral head projects anterior to the axis line is divided by the diameter. A lower value for PHHA indicated less humeral head anterior to the axis line and more posterior subluxation of the humeral head.

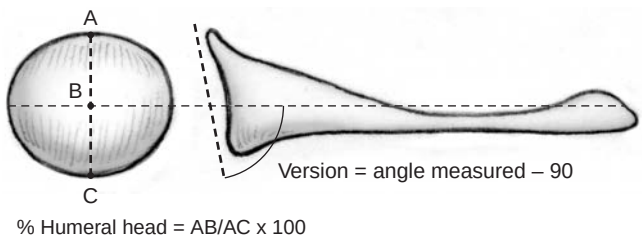

Figure 35-33 Schematic of glenoscapular angle (the degrees of version) and the percent of humeral head anterior to the middle of the glenoid fossa measurements.

Treatment

The goal of treatment is to maintain a reduced glenohumeral joint. Humeral head subluxation and glenoid

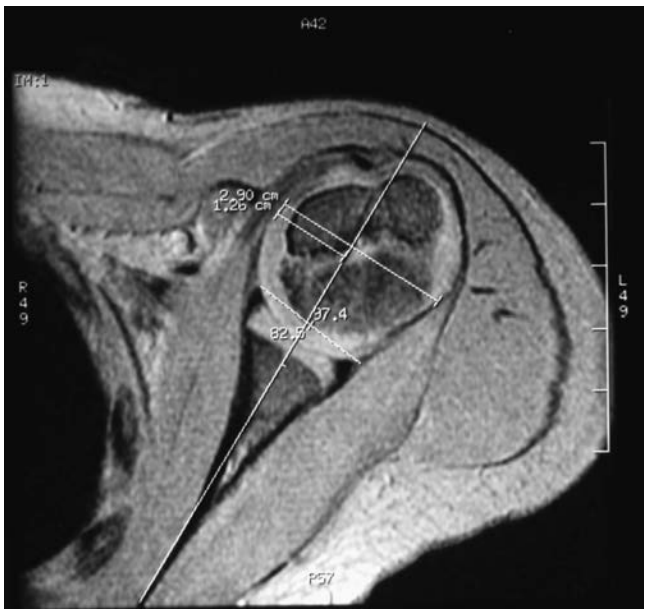

Figure 35-34 Magnetic resonance imaging of normal shoulder. The cartilage is readily apparent around the humeral head and the glenoid cavity is readily visible. The lines for calculating the glenoscapular angle (–7.5 degrees) and percent of humeral head anterior to the middle of the glenoid fossa (43%) are drawn. (Courtesy of Shriners Hospital for Children, Philadelphia.)

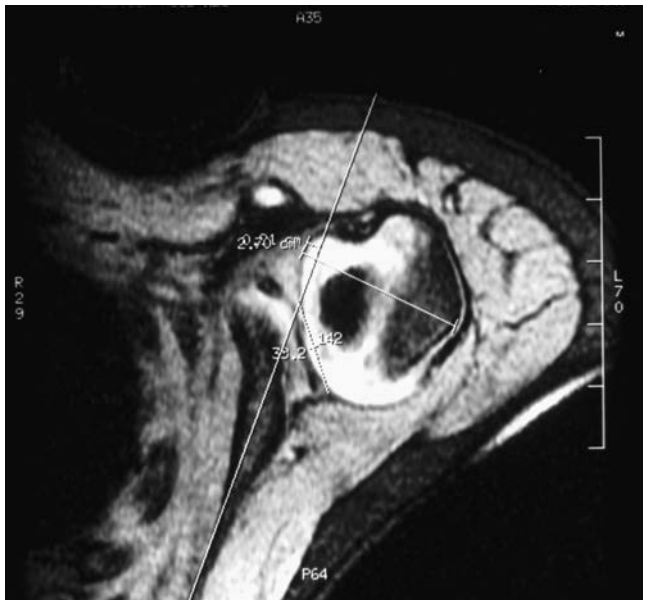

Figure 35-35 Shoulder magnetic resonance imaging of 4-year-old with –10 degrees of passive external rotation. The humeral head articulates with a posterior articular concavity that is markedly retroverted and measures 51 degrees. The humeral head barely crosses the midportion of the glenoid (percent of humeral head anterior to the middle = .21 cm/2.7 cm or 7.7%). (Courtesy of Shriners Hospital for Children, Philadelphia.)

deformity negate normal shoulder development. Early management is passive range of motion to maintain a supple glenohumeral joint. Specific exercises should be prescribed for glenohumeral external rotation combined with scapular stabilization (Fig. 35-36).

Failure to preserve passive glenohumeral external rotation motion beyond the neutral plane of the scapula is an indicator of considerable underlying joint deformity and warrants imaging (e.g., MRI).[94] If the imaging studies reveal maintenance of glenohumeral alignment without substantial humeral head subluxation (grades I and II), therapy may be continued in the young child (less than 3 years of age) or tendon transfers can be performed to rebalance the joint and enhance motion. Tendon transfers appear to halt progressive glenohumeral joint deformity. Considerable glenoid retroversion and humeral head subluxation (grades III and IV), however, warrant joint realignment, as current evidence suggests that concentric joint alignment is necessary to allow joint remodeling over time. Numerous methods can achieve joint reduction depending upon the age of the child and degree of deformity. Hui and Torode[80] performed open reduction and soft tissue release in 23 patients with an average age of 2 years and 5 months. Preoperative and postoperative computerized axial tomograms revealed improvement in the glenoid version of approximately 9% per year. Pearl[138] has advocated arthroscopic release of the thickened anterior capsule combined with subscapularis tenotomy to allow external rotation and glenohumeral joint reduction. This arthroscopic approach is less invasive and our experience has been promising with regard to its ability to achieve glenohumeral joint reduction.[139] Tendon transfers can be performed at the time of joint reduction or delayed until glenohumeral joint motion can be reassessed. In the deferred instance, failure to demonstrate active external rotation or limited abduction warrants secondary transfers.

Figure 35-36 Glenohumeral external rotation exercises must be combined with scapular stabilization. (Courtesy of Shriners Hospital for Children, Philadelphia.)

Certainly, there is a degree of deformity and age that prohibits joint reduction and remodeling. However, the exact age or degree of humeral head subluxation is unknown. We use treatment parameters similar to developmental dysplasia of the hip. In other words, the rare infantile dislocation requires immediate reduction, usually via an open procedure. In progressive humeral head subluxation and glenoid retroversion attributed to muscular imbalance, reduction is recommended for children less than 4 years of age with moderate to severe deformity (grades III and IV). In children greater than 8 years of age with severe deformity (grade V), reduction is usually not recommended, as humeral osteotomy is preferred to reposition the limb (Figs. 35-37 and 35-38).[48,60,88] In children between the ages of 4 and 8 years (i.e., "grey zone"), the treatment depends upon the severity of the deformity and the options are discussed with the parents.

External Rotation Tendon Transfer Technique[50,76,103,140,145,195]

The technique of latissimus dorsi and teres major tendon transfer is performed with the patient in the lateral decubitus position. The extremity and posterior thorax are prepared for surgery. A posterior axillary incision is performed from the tendon of the latissimus dorsi muscle to the posterior acromion (Fig. 35-39). Skin flaps are elevated and the latissimus dorsi, teres major, triceps, and posterior deltoid muscles are isolated. The posterior humeral circumflex artery and axillary nerve are identified between the

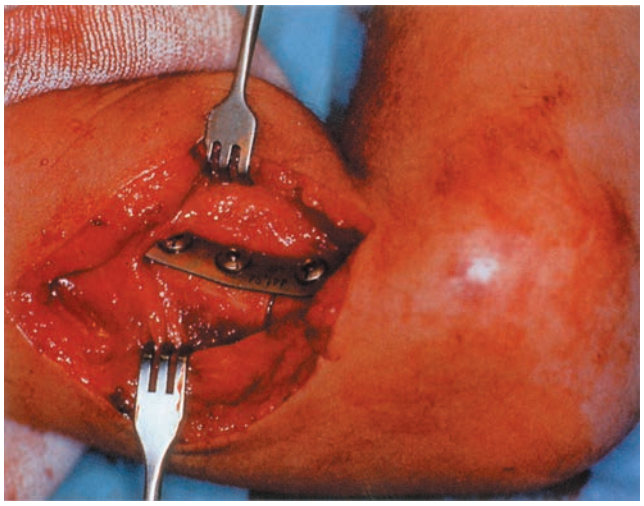

Figure 35-37 Distal humeral osteotomy in a 10-year-old child with a brachial plexus birth palsy, lack of external rotation, and glenohumeral dysplasia.

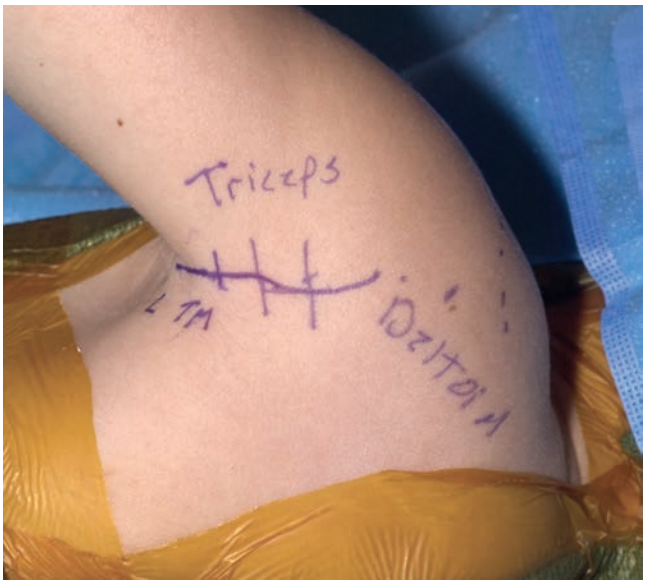

Figure 35-39 Posterior axillary incision from the tendon of the latissimus dorsi muscle to the posterior acromion. (Courtesy of Shriners Hospital for Children, Philadelphia.)

triceps and teres major muscles. The latissimus dorsi and teres major tendons are traced to their humeral insertion and released with a strip of periosteum (Fig. 35-40). The muscles are freed in a proximal direction until the thoracodorsal neurovascular pedicle is encountered. The interval between the posterior deltoid and triceps is isolated (Fig. 35-41). The posterior deltoid is retracted in a cephalad direction and the undersurface of the acromion is exposed. The subacromial bursa is removed and the humerus is placed into external rotation to expose the supraspinatus and infraspinatus insertions. Sutures are placed at this area into the tendon/periosteum or secured through drill holes (Fig. 35-42). The latissimus and teres major tendons are transferred superficial to the long head of the triceps and fixed firmly using the previously placed sutures. The arm is maintained in external rotation and the wound is closed.

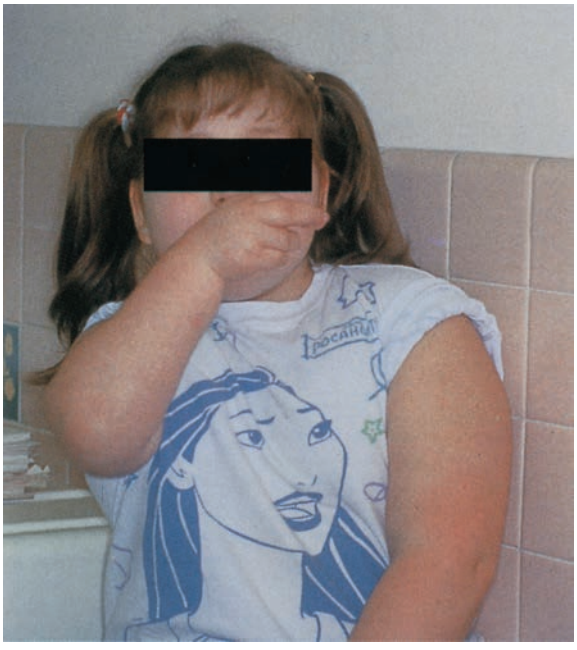

Figure 35-38 Improved hand-to-mouth function after humeral rotational osteotomy.

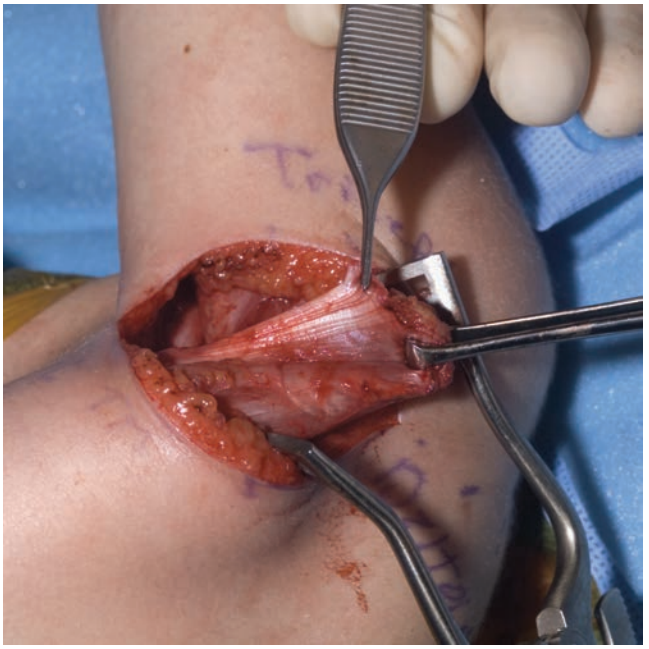

Figure 35-40 Latissimus dorsi and teres major tendons are traced to their humeral insertion and released with a strip of periosteum. (Courtesy of Shriners Hospital for Children, Philadelphia.)

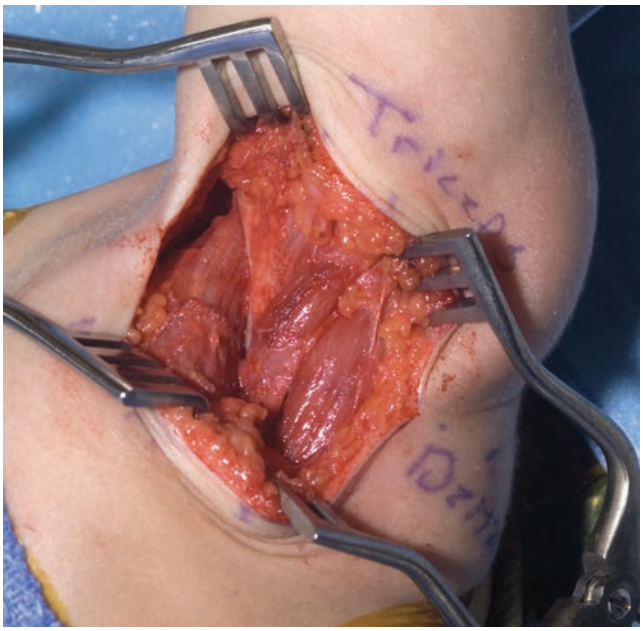

Figure 35-41 The interval between the posterior deltoid and triceps is isolated. (Courtesy of Shriners Hospital for Children, Philadelphia.)

A shoulder spica cast is applied with the shoulder in 90 to 120 degrees of abduction and full external rotation (Fig. 35-43). The spica cast is removed at 5 weeks and active therapy is initiated. Protective splinting is continued for 3 months after surgery.

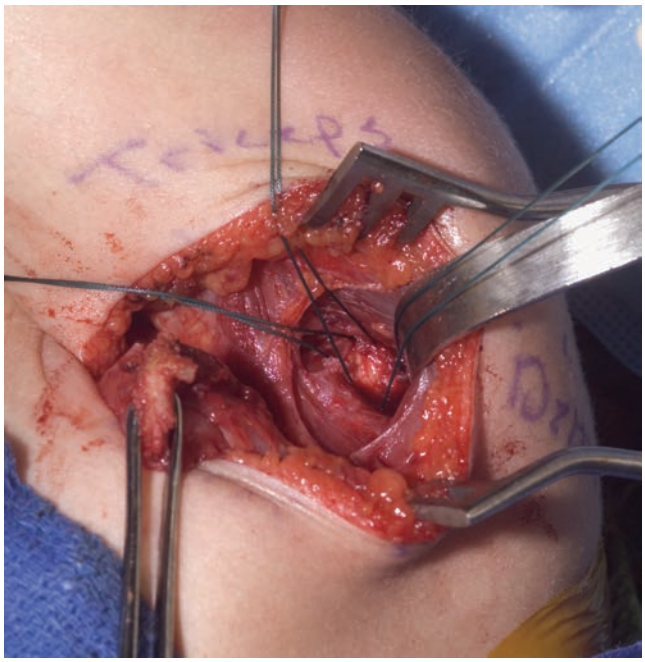

Figure 35-42 Sutures are placed into the interval between the infraspinatus and supraspinatus insertions. (Courtesy of Shriners Hospital for Children, Philadelphia.)

Figure 35-43 Shoulder spica cast applied in the operating room with the shoulder in 90 to 120 degrees of abduction and full external rotation. (Courtesy of Shriners Hospital for Children, Philadelphia.)

Arthroscopy Surgical Technique[138,139]

Arthroscopy is performed in a lateral decubitus position using a 2.7-mm arthroscope. A posterior portal is established through the posterior soft spot after localization with a spinal needle and joint insufflation with saline. An anterior portal is made under direct visualization using an 18-gauge spinal needle inserted inferior to the biceps tendon. The anterior capsule, anterior ligaments, and subscapularis tendon are identified. An electrocautery is introduced through the anterior portal. The thickened superior glenohumeral ligament, the middle glenohumeral ligament, and the tendon of the subscapularis are released. If possible, the inferior muscular portion of the subscapularis humeral attachment is preserved. The electrocautery is removed and exchanged for an arthroscopic punch. The inferior glenohumeral ligament is released to the midportion of the axillary pouch with protection of the axillary nerve. The arthroscopic equipment is removed from the joint and the glenohumeral joint is manipulated into external rotation. Marked improvement of external rotation is noted, often with an audible clunk associated with glenohumeral joint reduction. Failure to achieve joint reduction or passive external rotation less than 45 degrees with the arm in adduction requires additional arthroscopic release of the axillary pouch and the tight subscapularis. Concomitant tendon transfers to augment external rotation are often performed in children greater than 3 years of age. The child is placed in a shoulder spica cast with the glenohumeral joint positioned in 45 to 60 degrees of external rotation. The amount of abduction varies according to whether tendon transfers were performed.

Results

Tendon Transfer

Tendon transfers reliably improve abduction and external rotation (Table 35-10). Significant improvement in clinical

TABLE 35-10
PUBLISHED SERIES ON TENDON TRANSFERS IN PATIENTS WITH BRACHIAL PLEXUS INJURES

Authors	Number of Patients	Procedure	Preoperative Abduction	Preoperative External Rotation	Postoperative Abduction	Postoperative External Rotation
Covey et al.[40]	19	Latissimus and teres major transfer	49° active	3° active	74° active	32° active
Hoffer et al.[76]	11	Pectoralis major release and latissimus/teres transfer	74° active	0° passive	138° active	64° active
Hoffer and Phipps[77]	35	Pectoralis major release & latissimus/teres transfer	74° active	5° passive	120° active	31° active
Chuang et al.[27,*]	29	Teres major transfer, lengthening of pectoralis major muscle (sternal part), and transfer pectoralis major (of the costal portion)	74° active	24° passive	151° active	72° active
Waters and Peljovich[193]	32	Pectoralis major lengthening and latissimus/teres transfer			–8.1° passive	17.1° passive

*Seven patients were excluded from external rotation measurements secondary to concomitant procedures.

scores for overhead motion, external rotation, and hand-to-neck maneuvers are reported, along with measured improvement in active abduction and external rotation motion after surgery. The beneficial effects of motion, however, do not correlate with similar improvements in joint configuration. This lack of association is most likely related to the inability of the tendon transfer to reposition the humeral head within the glenoid.

Joint Reduction

Hui and Torode[80] performed open reduction and tendon lengthening on 23 patients with brachial plexus birth palsy and humeral head subluxation or dislocation. CT scanning was performed before and after surgery. The mean age of surgery was 2 years and 5 months. Mean follow-up was 3 years and 7 months. Concentric reduction was achieved under direct visualization and remodeling occurred during the follow-up period. Bony glenoid retroversion measured by CT scanning decreased by a mean of 31% after open reduction and improved at a rate of 9% per year. The difference between the normal and affected sides also decreased (Fig. 35-44).

Pearl[138] described an arthroscopic technique for contracture release in children with brachial plexus birth palsy. Forty-one children underwent arthroscopic release of the

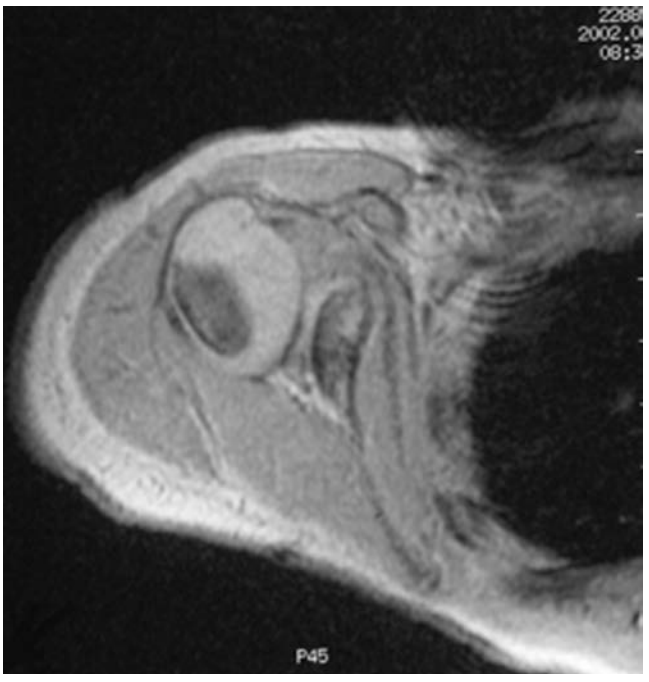

Figure 35-44 A 2-year-old child with progressive loss of external rotation. Magnetic resonance imaging shows posterior humeral head subluxation and glenoid retroversion. (Courtesy of Shriners Hospital for Children, Philadelphia.)

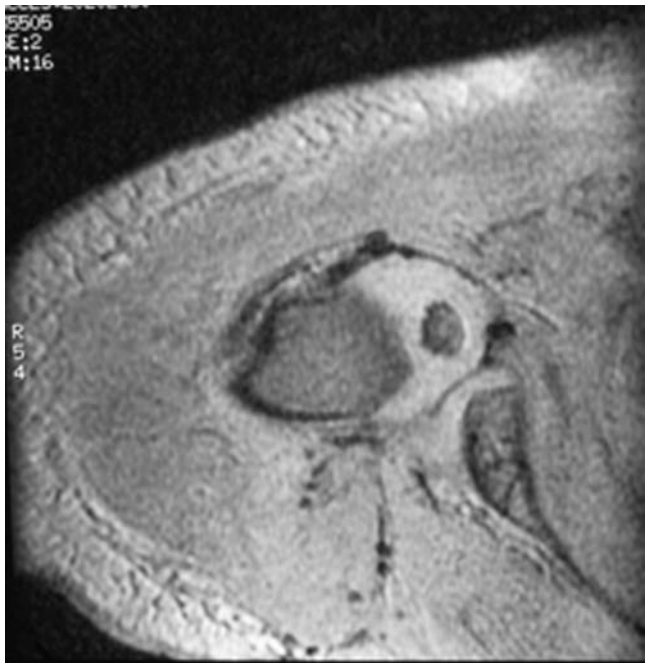

Figure 35-45 Magnetic resonance imaging after arthroscopic capsulectomy and glenohumeral reduction. Humeral head located within the socket and significant improvement in subluxation and version. (Courtesy of Shriners Hospital for Children, Philadelphia.)

anterior capsule and subscapularis tendon. The mean age of the children was 3.5 years. Eighteen children were treated with arthroscopic release alone, while 23 children also underwent latissimus dorsi transfer. The arthroscopic contracture release effectively restored passive external rotation in 40 of the 41 children. The single patient that did not achieve external rotation was 12 years of age with advanced glenoid deformity. The status of the glenohumeral joint, however, was not evaluated after surgery.

In our series (SHK and GRW), 20 children with an average age of 46 months (range, 19 to 100) underwent preoperative MRI, arthroscopic surgery, and postoperative imaging in their spica cast.[139] For the uninvolved shoulder, the mean PHHA was 45.2% ± 4.8% and the glenoid version was −7 ± 3%. The involved shoulder preoperative mean PHHA was 16.9% ± 16.1% and the mean glenoid version was −39° ± 17°. The postoperative mean PHHA corrected to 41.4% ± 13.3% and the mean glenoid version improved to −12° ± 12°. There was a significant improvement in the mean PHHA ($p < 0.001$) and mean glenoid version ($p < 0.001$) that approached the values of the uninvolved shoulder. We found that arthroscopic capsular release and subscapularis tenotomy was successful in reducing the glenohumeral joint subluxation in all patients (Fig. 35-45).

THORACIC OUTLET SYNDROME

The diagnosis of thoracic outlet syndrome is controversial and more common when surgeons are convinced this is a valid entity.[6,21] The cause is presumed to be a compressive neuropathy of the brachial plexus because neurologic symptoms predominate in virtually all (94% to 97%) cases, with preferential involvement of the lower plexus. Venous and arterial thoracic outlet syndromes are much less common, appearing in 2% to 3% and 1% to 2%, respectively.[6,130] The potential sites of compression (see Table 35-1) are described in the preceding anatomy section. These anatomic sites are normally not problematic unless additional factors are present that initiate the compressive neuropathy. For example, shoulder muscle atrophy and loss of strength can depress the entire limb and cause the inferior trunk to be compressed against the first rib. Further narrowing of the costoclavicular space can occur from downward pressure on the shoulder girdle from heavy backpacks or pendulous breasts.[74,98] A preceding minor or major trauma may result in fibrosis and scarring within the thoracic outlet, which can directly compress the plexus and cause symptoms.[6,51,162] In predisposed persons, repetitive motion may play a factor, although this remains speculative.[107,159]

Regardless of the pathologic site, intermittent compression causes temporary alterations in nerve physiology that reverses following removal of the compression. Persistent nerve compression yields intraneural edema and alteration of the blood supply, which results in diminished axonal transport to the compressed nerve. The initial nerve response is segmental demyelination (i.e., neurapraxia), but prolonged ischemia will cause wallerian degeneration (i.e., axonotmesis).[6,129,143] The degree of pathology is proportional to the intensity and duration of the entrapment pressure.

Arterial signs and symptoms can be induced by compression along the thoracic outlet. Pressure can be from the tip of a cervical rib, a fibrous band, or costoclavicular narrowing. Subclavian or axillary artery stenosis, with or without poststenotic dilation, is possible. This situation can advance to mural thrombus formation, emboli, and distal ischemia.[6]

Evaluation

The clinical presentation of thoracic outlet syndrome differs drastically from an overt brachial plexus injury. The symptoms tend to be vague and the signs more subtle.[6,98,129,130] Females are more commonly affected (4:1). A thorough examination with provocative maneuvers is required for accurate diagnosis. Virtually all patients present with neurologic symptoms affecting the lower plexus (C8 and T1). The distribution of pain or paresthesias is often the medial side of the arm and ulnar digits. Loss of dexterity during fine manipulation is a common complaint. Vascular thoracic outlet syndrome is much less common (less than 5%) and can affect venous outflow or arterial inflow.[130] Venous thoracic outlet syndrome is more

common than arterial involvement, and edema or vascular congestion may signal vascular outlet obstruction. Arterial compression may produce pain, cold intolerance (similar to Raynaud's phenomenon), and claudication. Rarely, pallor of the limb can result from arterial insufficiency.[39] The history should include inquiry about the onset of symptoms, antecedent trauma, and current activities that produce symptoms. The use of heavy backpacks and frequent maneuvers above the shoulder level are specific activities that can elicit symptoms.[74,159]

The physical examination begins with an assessment of the posture of the individual. Poor posture can narrow the thoracic outlet and provoke symptoms. A careful cervical spine and peripheral nerve evaluation is critical in search of additional sites of compression that can cause a double crush neuropathy. Coexisting entrapment at the carpal or cubital tunnel can be present in thoracic outlet syndrome. Proximal thoracic outlet compression causes the nerve to be more vulnerable to an additional distal compressive neuropathy.[6,129,198] The area of sensory abnormalities should be defined and careful manual muscle testing performed for objective signs of motor weakness that can occur in long-standing thoracic outlet syndrome. The ulnar nerve innervated muscles of the hand are most commonly affected by long-standing neurologic thoracic outlet syndrome and should be specifically assessed for weakness. Two-point discrimination in the ulnar and median nerve distribution should be determined. Sensibility may also be altered in the medial brachial and medial antebrachial nerve distribution.

The presence of edema, venous distension, or cyanosis may indicate a venous thoracic outlet syndrome, whereas pallor, splinter hemorrhages, or ischemic changes suggest arterial involvement secondary to vessel constriction or embolization.[6,162] Symmetry of the radial and ulnar pulses should be ascertained, and transient compression should be evaluated by the provocative maneuvers described in the following paragraph.

The supraclavicular region should be palpated for areas of tenderness, and percussion should be performed to elicit a Tinel's sign, which indicates axonal irritation. Provocative maneuvers are instrumental in the diagnosis of thoracic outlet syndrome, but must be interpreted carefully (Fig. 35-46). A positive test must not only produce the expected outcome, but also reproduce the patient's symptoms to be noteworthy. These tests attempt to narrow the anatomic constraints of the thoracic outlet and compress the neural or vascular elements. The Adson test is performed with the patient seated, placing the arm at the side, and palpating the radial pulse.[1] The patient rotates and extends the head and neck toward the affected side while taking a deep breath. This narrows the scalene triangle, and a pulse diminution or obliteration with corresponding symptoms implies thoracic outlet syndrome. The Wright hyperabduction test is performed by placing the arm in 90 degrees of abduction and external rotation.[201] The loss of a radial pulse and symptom reproduction are considered a positive finding. The overhead exercise stress test and 3-minute stress test are similar and performed by instructing the patient to abduct and externally rotate the shoulder to place the arms above shoulder level. Active opening and closing of the hands is then executed and a positive response will reproduce symptoms such as paresthesias or fatigue.[98,152,154] The diagnostic dilemma with these provocative maneuvers is the lack of specificity for isolation of the entrapment site and the significant rate of false-positive results that occur in both asymptomatic volunteers and in other common entrapment neuropathies of the upper extremity.[130,148,201] For example, shoulder abduction and external rotation places traction across the lower plexus at the level of the first rib, narrows the costoclavicular space, and pulls the plexus around the coracoid process. This maneuver also increases the pressure within the cubital tunnel and can produce symptoms of ulnar neuropathy at the elbow.

Imaging Studies

Imaging studies to evaluate a patient with suspected thoracic outlet syndrome are plain radiographic films, angiography, and MRI. Plain x-ray films of the cervical spine are used to identify cervical ribs, elongated transverse processes, or degenerative disc disease. The incidence of cervical ribs is approximately 0.5% to 1% and may be present in asymptomatic individuals.[6,141,169] A chest radiograph is routine to evaluate the lung apices for tumor.[12] Angiography and other noninvasive vascular studies are used selectively in suspected cases of vascular thoracic outlet syndrome for visualizing a mural thrombus, stenosis, and aneurysmal dilation. However, false-negative and false-positive results can occur.[6,98,188] MRI can be used to visualize the plexus anatomy and identify areas of compression and anomalous bands.[135] This study is both scanner- and radiologist-dependent and may be more applicable as imaging techniques and interpretation skills improve.

Electrodiagnostic Studies

Electrodiagnostic studies can be valuable in the diagnosis of thoracic outlet syndrome. Severe compression will produce denervation of the affected muscles, usually the intrinsic muscles of the hand.[58] Fibrillations and positive sharp waves will be evident upon needle insertion. However, most patients with neurologic thoracic outlet syndrome do not present with atrophy and EMG findings. A reduction of the CV across the pathologic thoracic outlet would seem to be a reliable indicator of this disorder. Unfortunately, this technique is difficult to perform, interpret, and reproduce.[98] Dynamic recording of nerve conduction across

Figure 35-46 Artist's schematic of provocative maneuvers for thoracic outlet syndrome.

the brachial plexus during provocative maneuvers is also logical, but is difficult to perform and to obtain consistent results.

The use of SEPs may play more of a diagnostic role in the assessment of conduction across the plexus than the standard nerve conduction tests.[21,104] Peripheral stimulation at the wrist or forearm with recording at Erb's point, cervical spine, and sensory cortex may demonstrate changes in the pattern or timing of particular waveforms in thoracic outlet syndrome. The interpretation of the SEP signal across the plexus, however, remains difficult and dependent on the proficiency of the neurophysiologist.

Treatment

The mainstay of treatment for thoracic outlet syndrome is conservative management directed at alleviating any compression of the thoracic outlet and avoidance of aggravating factors.[98,129] The patient often has poor posture associated with the weakness of the parascapular muscles, causing the scapula to sag. This position narrows the thoracic outlet and drags the lower trunk over the first rib. Postural exercises and training are instituted and aimed at the correction of poor body mechanics. Activity modification and avoidance of aggravating factors is a key to success. An exercise program, directed at selective muscle strengthening of the shoulder girdle with maneuvers designed to stretch the scalene muscles and relax the first rib, is part of the treatment regimen. Supervision is required to selectively exercise and strengthen the parascapular muscles (trapezium, levator scapulae, and rhomboids) without further compression of the thoracic outlet.[6,143] The goal is to expand the thoracic outlet to allow the brachial plexus to pass unimpeded. A variety of additional therapeutic modalities (ultrasound, biofeedback, and electrical stimulation) have been proposed with ambivalent success.[6]

Women with macromastia may obtain relief from better breast support. Reduction mammaplasty has been recommended in severe cases.[84,98] Ergonomic modification, aimed at the decrease of overhead activity and elimination of any downward force on the shoulder girdle, can also be beneficial. Conservative treatment is effective in improving symptoms in most patients, and only 10% to 30% of patients will be surgical candidates.[130,161,166,180,181]

Surgery

Surgery is indicated for intractable pain, a considerable neurologic deficit, vascular compromise, or when conservative measures have failed to alleviate symptoms.[130,161,166,180,181] The surgery is not without inherent risks and mild discomfort from mild compression is better left alone. The operative procedure is directed at decompression of the thoracic outlet, with release of any anatomic constrictions. There is no consensus on the most efficacious approach or procedure to correct thoracic outlet syndrome.[130] The fundamental components to the described procedures include cervical rib resection, first rib resection, scalenotomy, scalenectomy, excision of anomalous fascial bands, claviculectomy, pectoralis minor release, or a combination of these. The procedure can be accomplished by a variety of approaches, including a supraclavicular, transclavicular, subclavicular, transaxillary, posterior, or a combined approach.

The resection of the first rib, with or without anterior scalenectomy, has become the preferred procedure for thoracic outlet syndrome in most cases (Figs. 35-47 to 35-50).[98,130,154,155] Isolated scalenectomy is preferred for the less common upper plexus thoracic outlet syndrome. The supraclavicular approach favors scalenectomy and upper plexus exploration, but sacrifices complete first rib exposure.[181] Approximately 80% to 90% of the anterior scalene muscle and 40% to 50% of the middle scalene muscle are excised during scalenectomy.[5] The phrenic and long thoracic nerves must be identified and protected.

The first thoracic rib serves as a fulcrum for T1, an attachment site for the scalene muscles, and borders the costoclavicular space. Therefore, removal of the first rib relieves multiple potential sites of compression. The transaxillary route is more cosmetic and affords the best access to the first rib, but precludes scalenectomy and exploration of the upper plexus.[180] Cervical ribs can usually be removed through the axillary approach, and the pectoralis minor tendon can be divided if there is suspected subcoracoid compression. A combined supraclavicular and

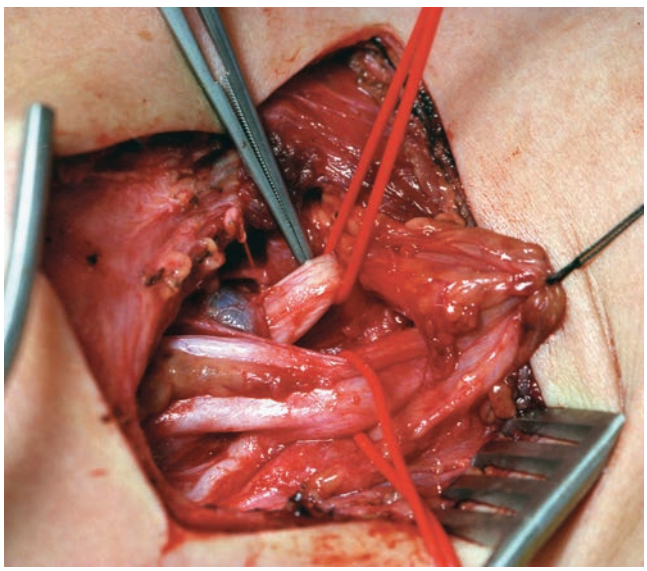

Figure 35-47 A 16-year-old female with recalcitrant thoracic outlet syndrome that failed to improve with therapy. Supraclavicular approach with isolation of the brachial plexus. Patient is supine and head is to the right. Bottom vessel loop around upper and middle trunk and top vessel loop around lower trunk. (Courtesy of Shriners Hospital for Children, Philadelphia.)

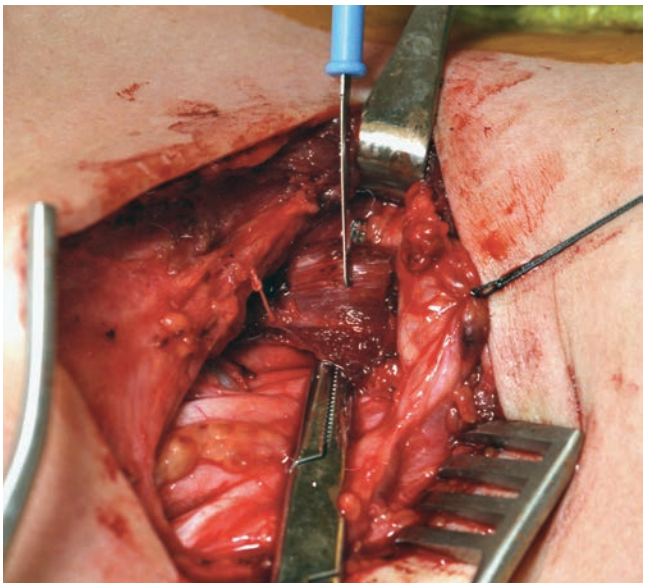

Figure 35-48 Anterior scalenectomy with protection of phrenic nerve and brachial plexus. (Courtesy of Shriners Hospital for Children, Philadelphia.)

transaxillary method can be used in complicated or recurrent cases.[146]

Intermittent venous or arterial thoracic outlet syndrome can also be treated by decompression. Persistent longstanding venous or arterial thrombosis may require thrombectomy or bypass grafting in recalcitrant cases.

Results

The results of thoracic outlet surgery are highly variable, with success rates dependent on the procedure employed and the definition of success. Isolated anterior scalenotomy has a 50% failure rate, with a significant recurrence rate.[59] The results of first-rib resection are successful (improvement of symptoms) in 37% to 92% of individuals, with lower recurrence rates.[51,99,152] Scalenectomy alone or combined with first-rib resection has reported success rates of approximately 68% to 86%.[6,146,161,162, 180,181] Improvement following thoracic outlet surgery may require 2 years or longer.[6] In contrast, the initial symptomatic relief may be nullified by scar formation, which culminates in recurrent compression. Comparison studies of first-rib resection versus scalenectomy do not reveal any appreciable difference.[162]

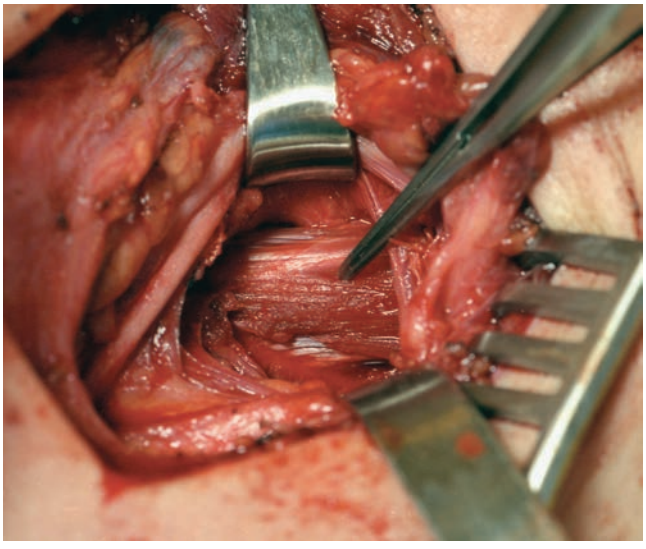

Figure 35-49 Isolation of middle scalene behind brachial plexus. (Courtesy of Shriners Hospital for Children, Philadelphia.)

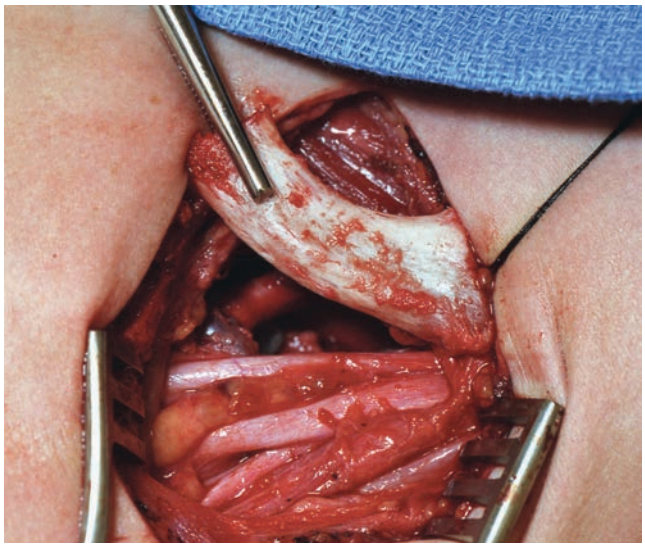

Figure 35-50 First rib resection via supraclavicular approach. (Courtesy of Shriners Hospital for Children, Philadelphia.)

Salvage Procedures

Salvage procedures are performed for residual brachial plexus injuries. Persistent deficits may result from irreparable lesions and partial recovery after nerve reconstruction. Early salvage procedures may be recommended in irreparable injuries or elderly individuals with limited capacity for nerve regeneration.[126] The goals of secondary procedures are to improve function of the shoulder, elbow, wrist, and fingers. The specific operation performed will vary with the brachial plexus lesion and residual deficit.

The evaluation of the patient must consider the original injury and treatment performed to date. A review of previous records is recommended. The extent of the initial paralysis and subsequent neurologic recovery is an important factor. The patient's physical and emotional health is a valuable component of the evaluation. The initial injury tends to alter the patient's life forever and the manner of acceptance is a consideration during formulation of

the reconstructive plan. The injury often overwhelms the patient's physical and social life for a time period. Fear of a similar episode of dependence after reconstructive surgery is genuine, and the reconstructive plan should be developed with minimal time away from work and activity. This strategy may require multiple procedures at a single setting.

A general assessment of shoulder, elbow, wrist, and hand function is performed. The balance of each joint complex and absence of a particular motion is noted. Any joint contractures may require therapy or operative release prior to tendon transfer. An inventory of the available muscles for transfer is recorded by completion of a brachial plexus injury sheet that records manual muscle testing (see Table 35-5). A potential donor muscle must have sufficient strength against gravity because transfer will decrease strength by one grade.[8,174] Muscles that have undergone reinnervation often lack normal strength and excursion, which limits usage as a donor for transfer. Ideally, reinnervated muscles would not be used for transfer, although often there are no other available options.[126]

In general, a similar priority is based on elbow flexion to improve arm positioning and encourage hand-to-mouth activity. Shoulder function is addressed concurrently or following elbow flexion. Wrist extension, finger extension, and finger flexion are managed after elbow and shoulder function. In lower plexus lesions, wrist and digit grasp and release assume priority.

Elbow Flexion

The restoration of elbow flexion requires a mobile joint and an available muscle for transfer. The goal of surgery is to regain a functional arc of elbow motion from 30 to 130 degrees. The potential donor muscles are the flexor–pronator group (Steindler flexorplasty), triceps, pectoralis major muscle, latissimus dorsi transfer, and free-muscle transfer.[17,25,26,32,108,172,173,174] The selection of the donor muscle varies with the strength of those donors available and consideration of additional tendon transfers that may use similar muscles. For example, if a latissimus dorsi transfer is planned for deficient shoulder external rotation, then an alternative elbow flexorplasty should be planned. The exact choice of transfer is variable, and each transfer has certain advantages and disadvantages.[9,93,174]

The Steindler flexorplasty transfers the flexor–pronator mass 2 in. proximal to the elbow and reattaches this muscle group to the anterior humerus (Fig. 35-51). Transfer of the flexor–pronator muscle group with a portion of the medial epicondyle and use of a compression screw provides better fixation (Fig. 35-52).[8] The procedure is relatively uncomplicated, but also produces weak elbow flexion.[29,86] Accordingly, this transfer has been used to augment existing weak elbow flexion.[29] In addition, elbow flexion may be accompanied by concomitant fist and forearm pronation, which compromises independent hand function. Technical modifications to reduce the pronation and flexion contracture have been proposed.[110]

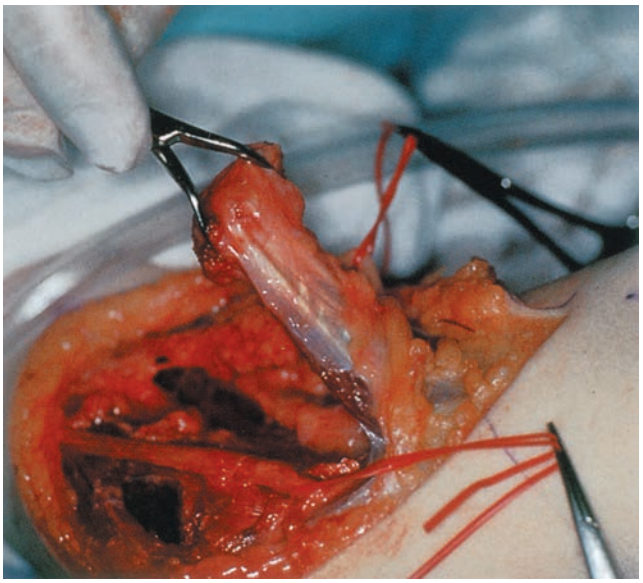

Figure 35-51 A 14-year-old boy with history of C5 to C6 transverse myelitis with absent elbow flexion. He was treated with proximal transfer of the medial epicondyle and flexor–pronator mass to the anterior humerus. Vessel loops around median and ulnar nerves.

The triceps transfer sacrifices elbow extension for flexion.[25] This procedure relies on gravity for elbow extension and is contraindicated in those patients who require powerful extension for transfer or crutch ambulation. This transfer is reserved for those individuals without other options for transfer or when co-contracture of the biceps and triceps is present.[108]

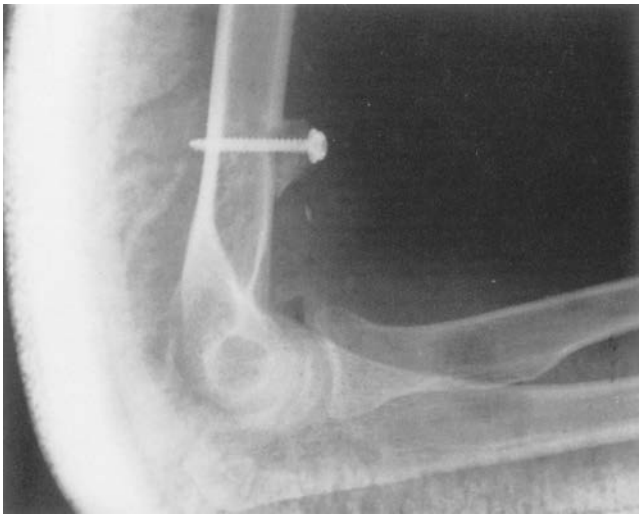

Figure 35-52 Postoperative radiograph of medial epicondyle and attached flexor–pronator mass secured to the anterior humerus with a compression screw.

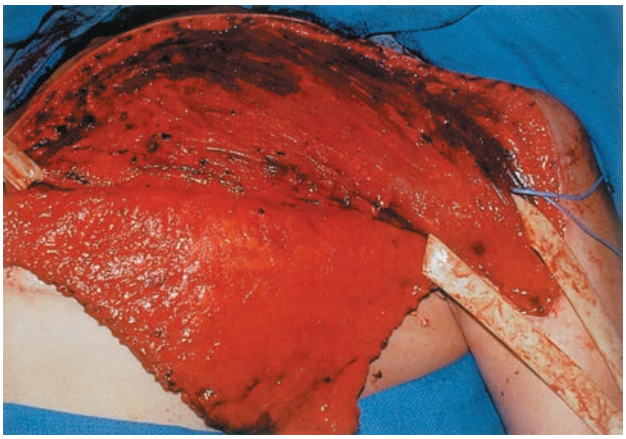

Figure 35-53 A 31-year-old man, status postbrachial neuritis (Parsonage-Turner syndrome), with persistent musculocutaneous nerve palsy. He was treated with bipolar pectoralis major muscle transfer for elbow flexion.

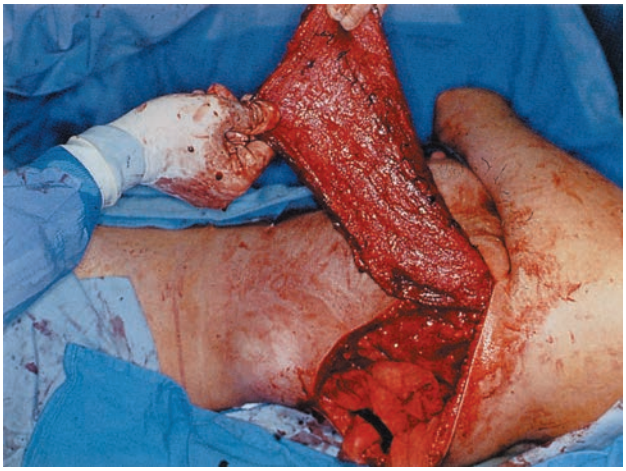

Figure 35-54 A 30-year-old woman with absent elbow flexion after brachial plexus injury secondary to having her arm struck by a moving train. Latissimus dorsi muscle harvested from distal to proximal for elbow flexorplasty. Note stay sutures placed to record resting length.

The pectoralis major muscle may be transferred by a variety of techniques, including elongation of the humeral insertion by tendon graft, transfer of the sternocostal origin to the biceps (monopolar technique), or transfer of the sternocostal origin to the biceps combined with transfer of the humeral insertion to the acromion (bipolar technique).[8,17,26,36,60,61] The bipolar technique provides a more powerful elbow flexion, as the mechanical advantage is improved by transfer of the origin and insertion. Bipolar transfer allows the entire mass of pectoralis major to be used for elbow flexion (Fig. 35-53).[26] However, this technique is technically demanding and cosmetically disfiguring.[108] Nonetheless, the bipolar technique is my preferred technique when the pectoralis major muscle is selected for flexorplasty, as function overrides cosmesis following brachial plexus injuries.

The latissimus dorsi transfer for elbow flexion can also be accomplished by a monopolar or bipolar technique. The bipolar method transfers the origin to the biceps tendon and the humeral insertion to the coracoid process or acromion.[93,204] The unipolar variant does not disturb the humeral attachment. This flexorplasty has certain distinct advantages that make bipolar latissimus flexorplasty my preferred procedure for elbow flexion.[93] The latissimus muscle can be sacrificed with minimal morbidity, provides excellent strength and excursion, and reinforces the anterior support to the shoulder, which may obviate the need for arthrodesis.[20,26] The drawbacks are related to the magnitude of the procedure and the difficult preoperative assessment of muscle grade, because bulk can overestimate strength.

Bipolar Latissimus Dorsi Transfer Technique[93,204]

The bipolar latissimus dorsi transfer is performed with the patient in the sloppy lateral position. The arm and torso are prepared to the midline for adequate exposure. The ipsilateral thigh is also prepared for possible fascia lata graft. A linear incision is performed in the posterior axillary line extending proximal from the thoracodorsal origin of the latissimus dorsi muscle and distal along the muscle belly toward the iliac crest. A large skin flap is elevated over the posterior aspect of the latissimus to the midline. The lateral border of the muscle is identified and elevated from the underlying serratus anterior muscle. Stay sutures are placed on the surface of the muscle to record the resting length. The neurovascular pedicle is identified by palpation at the junction between the proximal one-third and distal two-thirds on the undersurface of the latissimus. The neurovascular pedicle is carefully isolated. The muscle with thoracodorsal fascia is harvested from distal to proximal (Fig. 35-54). Meticulous hemostasis is attained throughout this dissection using electrocautery. The entire latissimus dorsi muscle is harvested on the thoracodorsal neurovascular pedicle. The vascular pedicle is mobilized to the subscapular artery, with ligation of the branch to the serratus muscle. The insertion of the latissimus tendon is tagged and divided. An anterior deltopectoral approach to the shoulder is performed and the latissimus dorsi muscle and pedicle are passed into the anterior wound (Fig. 35-55). Care must be taken not to kink the vessels. A large subcutaneous passage is tunneled from the deltopectoral interval to the antecubital fossa, where the biceps tendon is exposed. The origin is attached to the biceps tendon by a weave technique. The addition of fascia lata interwoven into the latissimus dorsi for augmentation is necessary in certain instances. The elbow wound is closed before setting final tension at the anterior shoulder level. Final tension of the latissimus dorsi flexorplasty is determined by recreating resting length, using the previously placed stay sutures as a guideline and creating a 30-degree-tenodesis effect at the elbow. The humeral insertion is attached to either the

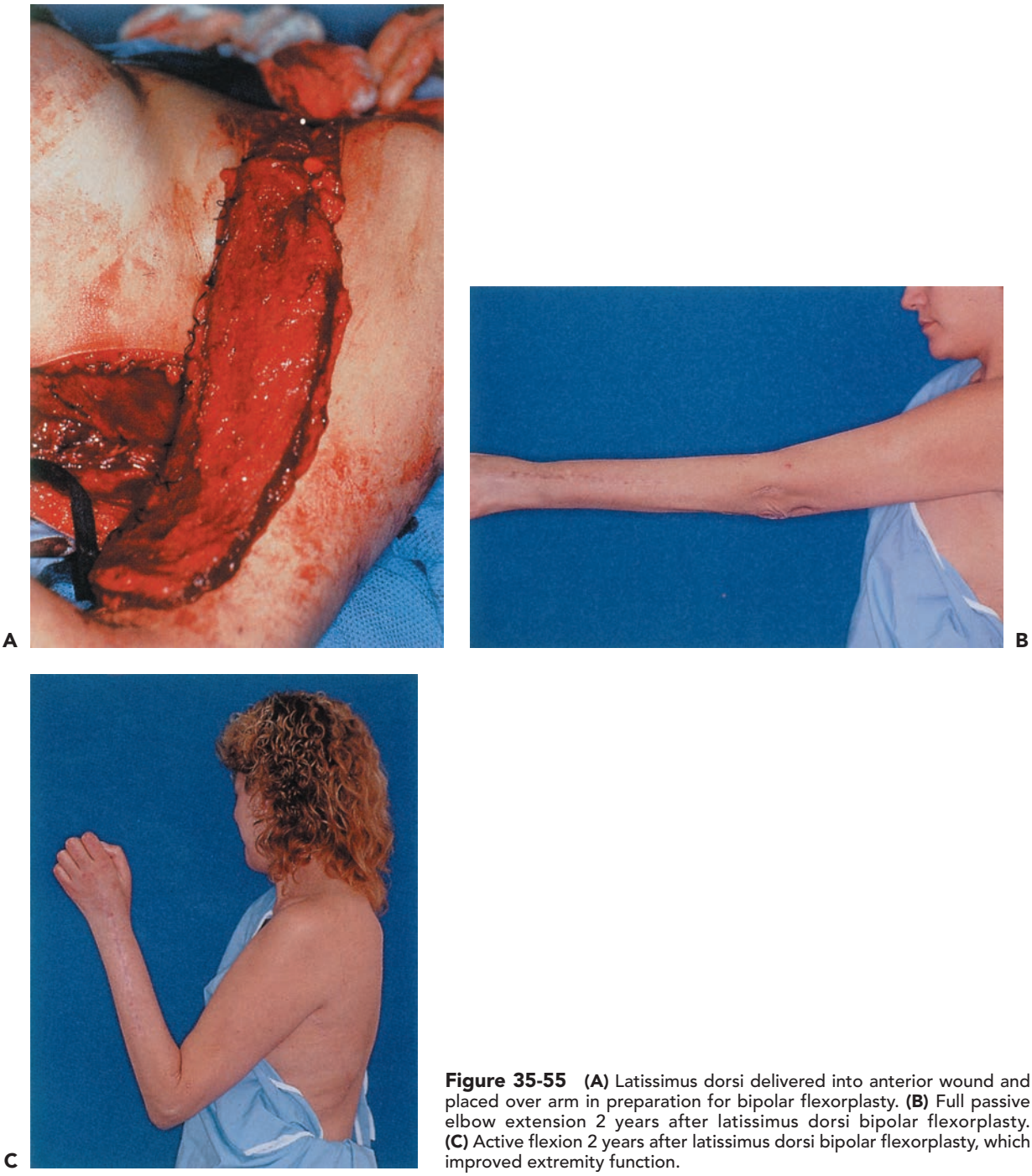

Figure 35-55 **(A)** Latissimus dorsi delivered into anterior wound and placed over arm in preparation for bipolar flexorplasty. **(B)** Full passive elbow extension 2 years after latissimus dorsi bipolar flexorplasty. **(C)** Active flexion 2 years after latissimus dorsi bipolar flexorplasty, which improved extremity function.

coracoid or acromion, depending on which provides the correct tension. Suction drains are placed into the back wound and the arm is immobilized to the chest for 6 weeks before active flexion. Protective extension block splinting is continued until 3 months after surgery.

The results after elbow flexorplasty are generally good, with a return of flexion against gravity (Figs. 35-56 and 35-57).[8,25,26,108,204] These transfers often lack lifting power, but there is considerable improvement in function compared with a paralyzed elbow.[8,29,93] A flexion contracture is common, but this posture also provides a mechanical advantage to initiate flexion. The presence or persistence of poor shoulder control will inhibit use of the transfer and curtail the result.[86,108,204]

Free-muscle transfer with nerve coaptation is a valiant technique to restore flexion.[32,95] This procedure involves a free microvascular muscle transfer of an expendable muscle with coaptation of a viable donor nerve to the motor nerve of the transferred muscle. This technique is indicated when other less complicated procedures are unavailable.

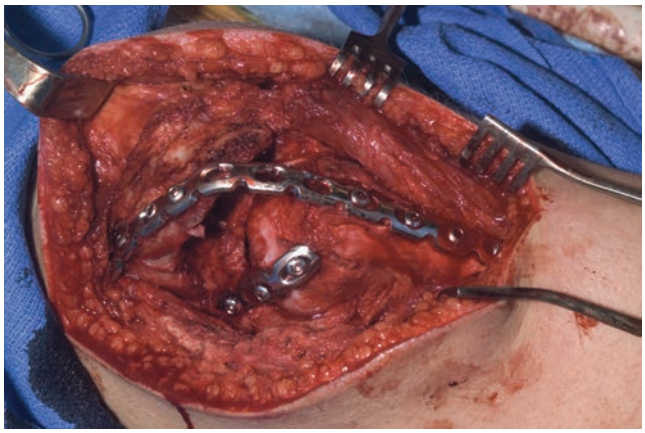

Figure 35-56 A 14-year-old male with flail shoulder after traumatic brachial plexus injury. Treated with glenohumeral arthrodesis using dual plate fixation. (Courtesy of Shriners Hospital for Children, Philadelphia.)

The gracilis is the most common muscle used for neurovascular transfer, with the intercostal nerves or spinal accessory nerve coapted to the obturator motor nerve for reinnervation.[29,32,95] The vascular supply to the gracilis is reestablished by microsurgical anastomosis of the pedicle artery and vein to the brachial artery (end to side) and a local vein (e.g., cephalic vein). The muscle is attached in a similar fashion to a latissimus dorsi flexorplasty with proximal fixation to the coracoid or clavicle and distal weaving into the biceps tendon. The latissimus dorsi muscle has also been used as a free muscle transfer for elbow and finger motion.[45]

The recent results after functioning free-muscle transplantation are impressive, with elbow strength greater than gravity achieved in approximately 75% to 80%, which are similar results to the pedicled latissimus dorsi transfer.[32,95]

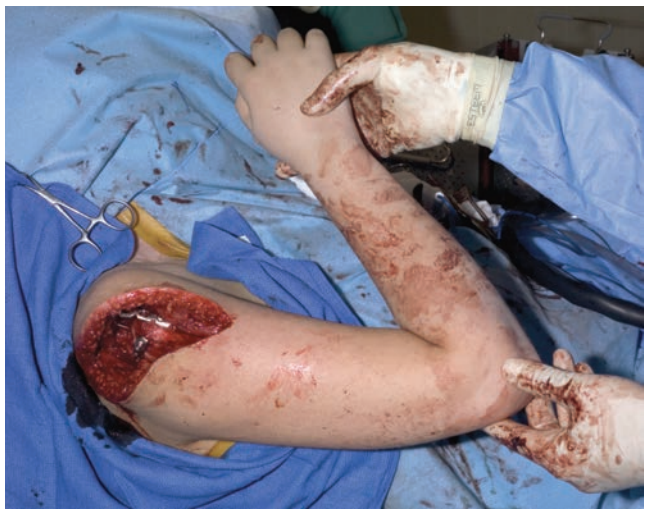

Figure 35-57 Shoulder is positioned in 30 degrees of abduction, 30 degrees of internal rotation, and 30 degrees of forward flexion to allow hand–to-mouth activity. (Courtesy of Shriners Hospital for Children, Philadelphia.)

Shoulder Function

Shoulder function is impaired in upper brachial plexus lesions with paralysis of the deltoid and rotator cuff muscles.[50,56] The shoulder may be unstable by muscle absence or unbalanced by asymmetrical muscle pull. The paralysis of the infraspinatus and teres minor muscles prevents external rotation. The subscapularis, pectoralis major, and/or latissimus dorsi muscles position the arm in marked internal rotation. This position is disabling for hand-to-mouth function.[108,145] This problem is particularly relevant in patients with residual brachial plexus birth palsies and has been covered in the previous section.[60,140]

The treatment of residual shoulder deformities is based on tendon transfer to rebalance the shoulder, humeral osteotomy to improve position, or arthrodesis to provide a stable shoulder. The most common tendon transfer is relocation of the latissimus dorsi and teres major tendons to the infraspinatus insertion to restore external rotation.[48,76,140,145,195] This procedure is common in children with residual brachial plexus birth palsy with loss of external rotation (see previous section).

Another tendon transfer about the shoulder for residual brachial plexus palsy involves restoration of glenohumeral abduction. Donor muscles used include the trapezius, levator scapulae, and latissimus dorsi.[50,56,82] The trapezius is transferred with a portion of the acromion to the decorticated posterolateral humerus. The levator scapula is elongated with a fascial graft to reach the tendon of the supraspinatus. The latissimus is transferred on a pedicle similar to the bipolar technique for flexorplasty.[82] Experience with these transfers is small and expected abduction is limited to only 30 to 60 degrees.[157] In persistent cases of shoulder instability or subluxation, arthrodesis is preferred in adult plexopathies.

Shoulder arthrodesis relies on the scapulothoracic muscles for motion and predictably corrects painful glenohumeral subluxation. Absent scapular control is a contraindication for shoulder fusion. The optimal position for fusion is controversial, but should allow hand-to-mouth function with active elbow flexion. The preferred position is 30 degrees of abduction, 30 degrees of internal rotation, and 30 degrees of forward flexion (see Figs. 35-56 and 35-57).[149,150] Numerous operative techniques have been described to achieve union. Rigid internal fixation is preferred, with an AO reconstruction plate across the scapular spine and onto the lateral humerus (Fig. 35-58). AO large cancellous screws are secured from the lateral humerus into the glenoid, and bone graft is added (Fig. 35-59). Postoperative immobilization varies from an abduction pillow to a shoulder spica, depending on the quality of the bone and rigidity of the fixation. Shoulder arthrodesis reliably corrects painful subluxation and provides stability to

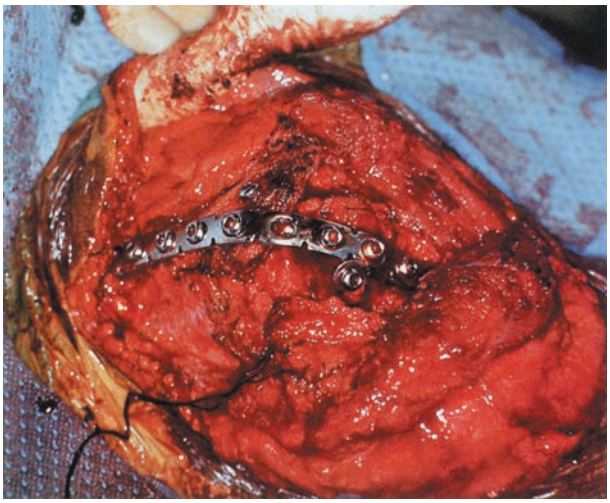

Figure 35-58 Shoulder arthrodesis for an unstable, arthritic, and painful glenohumeral joint after a shoulder dislocation with persistent axillary nerve palsy.

the extremity.[149,150,157] This proximal stability will allow better use of the extremity via scapulothoracic motion and allow further restoration of distal function using additional tendon transfers.

Forearm, Wrist, and Hand

The forearm, wrist, and hand impairment is variable in residual brachial plexus injuries.[14] The goals are to rebalance the extremity by tendon transfer or arthrodesis. Transfer is preferred, but the limited availability of donors is a frequent problem. Wrist arthrodesis can provide a stable platform and will liberate wrist tendons as donors for transfer. The principles are to achieve grasp and release for function. The potential transfers are innumerable, but all must comply with the basic requirements and tenets of tendon transfers. The most common transfer involves restoration of wrist and digit extension with an expendable wrist or finger flexor tendon.

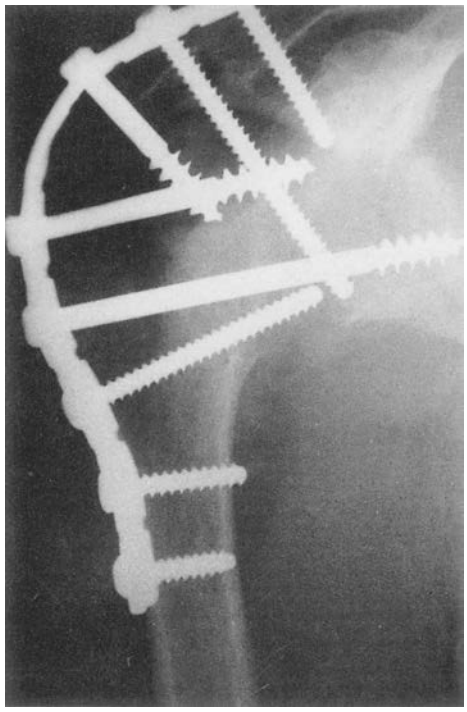

Figure 35-59 Postoperative x-ray film of shoulder arthrodesis performed with reconstruction plate.

COMPLICATIONS OF BRACHIAL PLEXUS SURGERY

Brachial plexus surgery is a considerable undertaking with potential for both minor and major complications. These problems can be mild and transient, or severe and life-threatening. The proximity of major vascular, nervous, lymphatic, and pulmonary structures, coupled with surrounding scar, increases the risk of injury (Table 35-11). Vascular injury can occur during the surgical approach or during separation of the brachial plexus from the surrounding vasculature. The subclavian artery and vein are most susceptible to injury, especially after a previous repair or interposition grafting. Vascular injury can cause profuse bleeding that requires expedient measures to avert a catastrophic complication.

Lymphatic injuries can occur during surgery on the left side of the plexus, and chylous drainage or a chylothoracic sinus may result after unrecognized injury.[6] Pulmonary injury can occur during plexus dissection or harvesting of the intercostal nerves for transfer. During first-rib resection, pleura violation and pneumothorax are not uncommon and require chest tube placement. In addition, direct injury

TABLE 35-11
COMPLICATIONS OF BRACHIAL PLEXUS INJURIES

System	Injury	Treatment
Vascular	Laceration Thrombosis	Repair Thrombectomy
Pulmonary	Pneumothoracic pleural effusion Phrenic nerve	Tube thoracostomy Avoidance
Lymphatic	Thoracic duct	Ligation
Nervous	Laceration Traction Dura puncture RSD	Repair Intermittent relaxation Repair

to the phrenic nerve during anterior scalenectomy will alter hemidiaphragm mobility during inspiration and can cause dyspnea.[6] Supraclavicular dissection can enter a pseudomeningocele and cause cerebrospinal fluid drainage. Signs of cerebrospinal drainage are a diminution in pulse rate and treatment requires immediate repair.[126]

Nerve injuries can result from inadvertent laceration or traction during surgery. Motor and sensory nerves are susceptible. The intercostal brachial cutaneous nerve is vulnerable during axillary dissection for first-rib resection or latissimus dorsi transfer. This nerve emanates from the third interspace and supplies sensation to the medial aspect of the arm. A neuroma or neuritis can develop that can be painful and disabling. Treatment requires localized nerve blocks or excision of the intercostal brachial cutaneous nerve. The lower plexus is particularly susceptible during first-rib resection for thoracic outlet syndrome secondary to traction from arm elevation or direct transection.[42] Intermittent arm relaxation will prevent stretching and ischemia of the lower plexus. The prognosis for recovery after a substantial lower-trunk injury is poor and residual deficits are likely. An injury at this level can also involve the sympathetic fibers and result in a Horner's syndrome. The long thoracic nerve is also in danger during first-rib resection and middle scalenectomy, as the nerve travels along the middle scalene.[199]

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36 Suprascapular and Axillary Nerve Injuries

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SUPRASCAPULAR NERVE INJURIES

Anatomy

The suprascapular nerve is a mixed motor and sensory nerve arising from the superior trunk of the brachial plexus. Although cadaveric dissections have found contributions from the fourth cervical nerve in up to 22% of specimen, it receives its fibers predominantly from the fifth and sixth cervical nerves.[1,52] The nerve innervates and provides the motor function to the supraspinatus and infraspinatus muscles. It provides sensory input from the coracoacromial and coracohumeral ligaments, the subacromial bursa and the acromioclavicular and glenohumeral joints. Rarely, there may be also be a cutaneous branch of the nerve that innervates the proximal lateral one-third of the arm.[1,42]

After leaving the superior trunk of the brachial plexus, the nerve courses across the posterior cervical triangle parallel to the omohyoid muscle, deep to the trapezius muscle, and adjacent to the posterior surface of the clavicle. Proximally, the nerve is located lateral to the brachial plexus. As it heads toward the superior edge of the scapula and the suprascapular notch, however, the nerve migrates posterior to the plexus. At this location, the nerve runs through the suprascapular notch, most often staying deep to the superior transverse scapular ligament. The suprascapular artery and vein, which travel with the nerve, course superior to the ligament. After passing through the notch, the nerve runs inferiorly along the posterior neck of the scapula before turning medially around the spinoglenoid notch. It then terminates in several branches innervating the infraspinatus muscle (Fig. 36-1).[22,71,78] There has been some controversy regarding the presence of a true spinoglenoid or

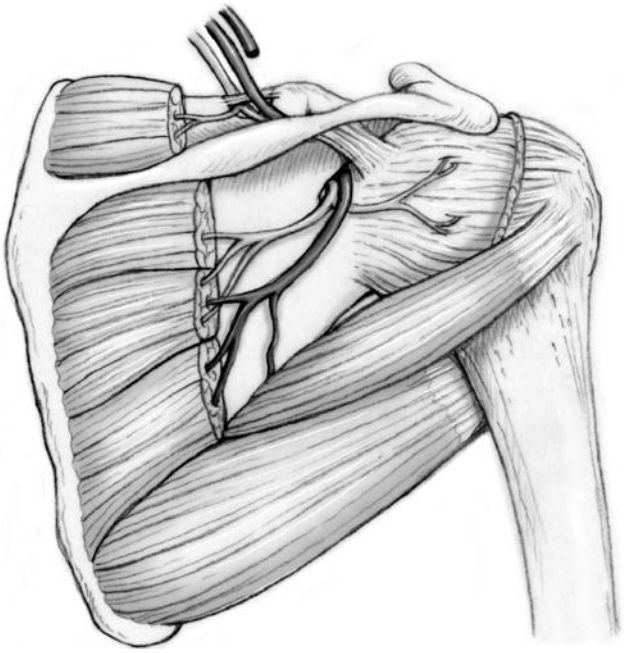


Figure 36-1 Anatomy of the suprascapular nerve. Note that the nerve runs deep to the superior transverse scapular ligament at the suprascapular notch while the vessels run superior to the ligament. After running along the posterior aspect of the scapula neck, the nerve courses through the spinoglenoid notch and heads medially along the inferior border of the scapula spine.

an inferior transverse scapular ligament. A recent anatomic study, for example, demonstrated that a true ligament existed in only 20% of the specimen and that 60% of the specimen contained just a thin fibrous band at that site.[21]

Classification and Etiology

Injuries to the suprascapular nerve are primarily classified according to the location and cause of the lesion. In addition, they may also be characterized by the degree of intraneural damage. The nerve can be injured at any point along its path, but common locations include (1) the supraclavicular or infraclavicular region prior to entering the suprascapular notch, (2) the suprascapular notch, (3) the posterior scapular neck, and (4) the spinoglenoid notch. Suprascapular nerve injuries can be caused by traction, extrinsic compression, and direct trauma. The injuries may be iatrogenic or associated with a generalized brachial plexus disorder.[78]

Traction injuries are believed to occur most commonly at the suprascapular and the spinoglenoid notches, as the nerve has little excursion at these sites. Furthermore, variations in the osseous and ligamentous anatomy may limit the available space for the nerve. For example, a cadaveric study demonstrated that cross-body adduction and internal rotation of the shoulder tightened the spinoglenoid ligament and posterior capsule, which, in turn, stretched the suprascapular nerve underneath the ligament.[26] First described by Kopell and Thompson in 1963, these types of traction injuries may occur following repetitive overhead activities.[49] Anatomic variations of the suprascapular notch have been investigated extensively by Rengachary and colleagues, who created a classification system of these variations (Fig. 36-2).[74] The implication of this classification was that the likelihood of a traction injury is clearly related to the available space for the nerve as well as the sharpness of the bony edges. These characteristics may be more accentuated when the shoulder is hyperabducted, as this would increase the contact between the nerve and the edge of the notch.[75] Rengachary et al. described this mechanism as "the sling effect."[75] Subsequently, clinical case reports of suprascapular nerve injuries have supported this proposed mechanism of injury.[16,81]

Extrinsic compression can also injure the suprascapular nerve. As discussed previously, compression of the nerve may occur at the suprascapular and the spinoglenoid notches by different anatomic structures. In addition, extreme shoulder abduction and external rotation that occur with overhead sports can cause compression of the nerve between the rotator cuff tendons and the scapular spine.[80] The most common cause of extrinsic compression on the suprascapular nerve is a ganglion cyst. These cysts originate from the glenohumeral joint and are often associated with tears in the superior or posterior labrum. The size of the ganglion cyst can vary, which, in turn, can affect the clinical presentation. Thus, for example, larger cysts can cause a more proximal lesion on the nerve and change the presenting symptoms. In rare instances, other masses such as synovial sarcomas, Ewing's sarcoma, chondrosarcoma, renal cell carcinoma, and schwannoma have all been reported to cause suprascapular nerve compression.[32,82]

Trauma is another cause of suprascapular nerve injury. Penetrating injuries, for example, can directly lacerate the nerve.[93] More commonly, however, suprascapular nerve injuries have been associated with blunt and more generalized shoulder girdle trauma such as glenohumeral dislocations and fractures. Nearby fractures in the scapula, particularly those involving the glenoid neck or the suprascapular notch, have caused suprascapular nerve injuries including traction and transection.[10,84]

Vascular insult of the suprascapular nerve has been proposed as a cause of injury. The proposed mechanism involves an intimal injury to the suprascapular artery following repetitive overhead activities. The vascular injury, in turn, is thought to produce microemboli that eventually compromise the blood supply to the vasa nervorum and cause an ischemic injury to the nerve.[76] Unfortunately, however, this hypothesis has not been substantiated clinically.[22]

Rare instances of iatrogenic suprascapular nerve injury have also been reported in the literature. For example, Mallon et al. reported two cases of suprascapular nerve injury

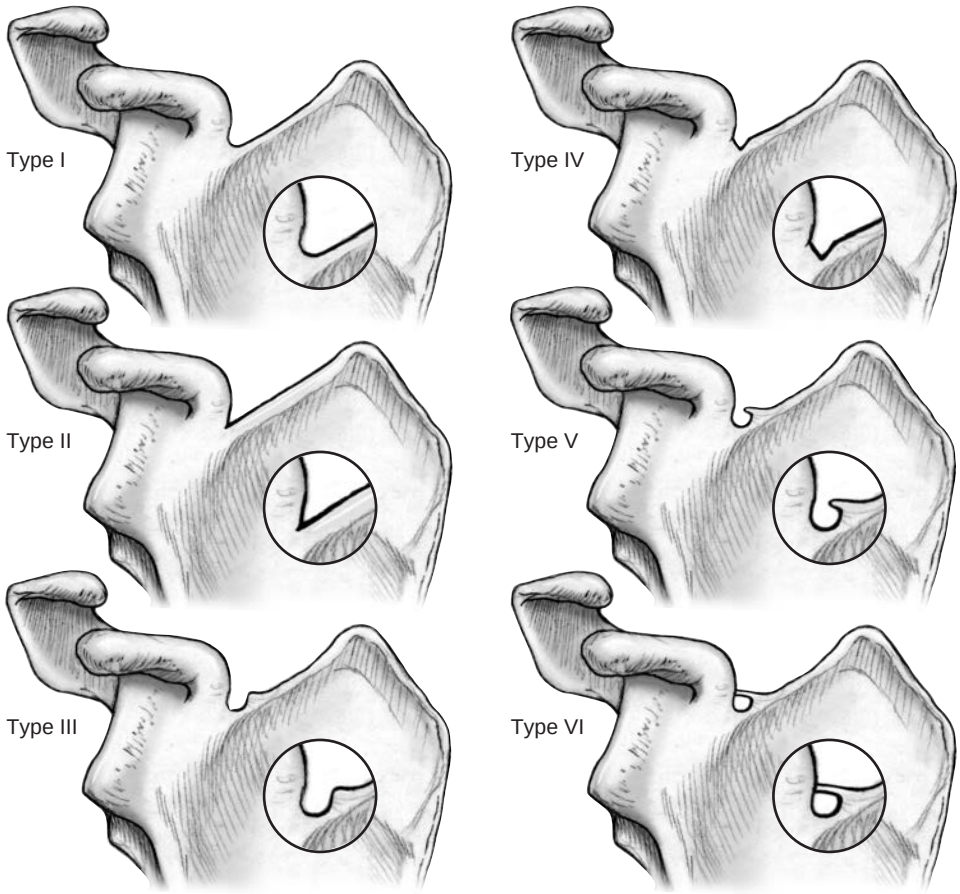


Figure 36-2 Classification of suprascapular notch morphology as described by Rengachary et al.[74] With varying bony and soft tissue anatomy, the potential for nerve compression injury may be increased at this site.

following distal clavicle resection. In both cases, excessive resection of the clavicle was believed to be related to the injury. Hence, the authors recommended that the distal clavicle not be resected beyond 1 cm.[56] In addition to distal clavicle resection, rotator cuff repair surgery may also cause suprascapular nerve injuries. According to a cadaveric study, excessive lateral advancement of the supraspinatus and the infraspinatus tendons may alter the orientation of the nerve's terminal branches by 180 degrees. As such, these authors recommended limiting lateral advancement of the rotator cuff tendons to 3 cm and suggested that further advancement (e.g., repair of massive rotator cuff tendons) may lead to suprascapular nerve injuries.[91] Another rotator cuff repair procedure called the disinsertion-advancement technique allows mobilization of the tendons while altering the orientation of the nerve by only 90 degrees.[24] Unfortunately, however, the first motor branch to the supraspinatus muscle remained trapped under the suprascapular ligament and limited the lateral advancement of the tendons.[91] Although the anatomic data are compelling, clinical studies of massive rotator cuff repairs with significant lateral advancement have not substantiated these concerns.[35,77,96] Other procedures associated with iatrogenic suprascapular nerve injury involve exposure of the posterior glenoid neck. Cadaveric studies have established a "safe zone" for avoiding the nerve. The average distance from the supraglenoid tubercle to the nerve is between 23 and 30 mm. At the base of the spine, as the nerve courses around the spinoglenoid notch, this distance shortens to 14 to 18 mm. Therefore, surgical procedures should remain lateral to this area to avoid an injury to the nerve.[8,83]

Clinical Symptoms

Despite the numerous causes of suprascapular nerve injury, most patients present with similar symptoms. Often, the patients complain of poorly localized but constant "achy" pain over the posterior and lateral aspects of the shoulder. Although the onset of pain may be associated with a specific trauma or change in activity level, most patients report an insidious onset. Activities that involve repetitive overhead motion may exacerbate the symptoms. Pain and weakness tend to be more severe in patients with a proximal nerve lesion. In contrast, patients with a distal lesion often complain of weakness only and may report minimal or no pain. This finding is consistent with the anatomic distribution of the nerve fibers described previously. In rare instances, the patients may be completely asymptomatic. As such, the nerve lesions may represent an incidental finding of unknown clinical significance.[22,30,71,78]

Clinical Evaluation

In addition to a deliberate shoulder examination, a thorough upper extremity and cervical spine examination must be performed on all patients. During the early stages following a suprascapular nerve injury, the examination may only yield nonspecific findings. Chronic nerve injuries, however, may reveal asymmetry of the shoulder girdle musculature, denoting atrophy of the supraspinatus and/or infraspinatus muscles. Due to the overlying trapezius muscle, atrophy of the supraspinatus muscle may be difficult to appreciate in some patients. Atrophy of the infraspinatus muscle, however, should be readily apparent (Fig. 36-3). Palpation along the course of the nerve can reveal focal tenderness at the site of compression. Examination may also reveal mild to moderate loss of strength in shoulder abduction and external rotation. Weakness of abduction will occur only with lesions involving the supraspinatus muscle as it acts in concert with the deltoid muscle to elevate and abduct the humerus.[43] Involvement of the infraspinatus muscle does not always result in external rotation weakness as some patients can compensate with the teres minor and posterior deltoid muscles.[30,38]

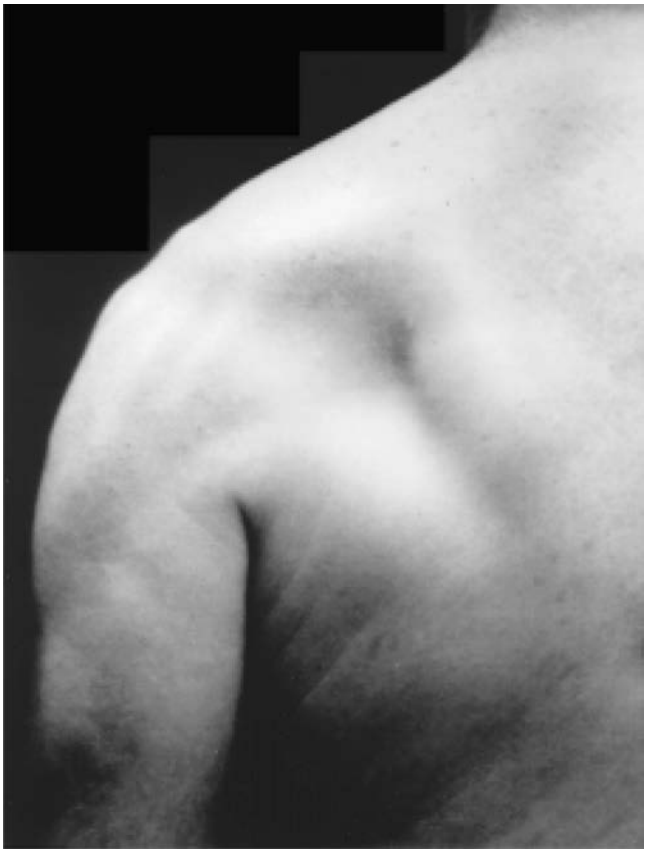


Figure 36-3 Clinical picture of infraspinatus muscle atrophy due to suprascapular nerve compression at the spinoglenoid notch. Note the loss of normal posterior shoulder contour due to the muscle atrophy. (Reprinted with permission from Cummins CA, Messer TM, Nuber G. Suprascapular nerve entrapment. *J Bone Joint Surg Am* 2000;82-A:415–424.)

Unlike other peripheral neuropathies, there are no specific provocative maneuvers to accentuate the symptoms of a suprascapular nerve lesion. Cross-arm adduction of the shoulder may place the nerve under increased tension as it passes under the suprascapular ligament. Hence, if a patient notes increased pain or discomfort with this maneuver, a suprascapular nerve injury may be suspected.[15] It is important to differentiate the exact location of pain, however, as acromioclavicular joint pathology may also cause pain with this maneuver.[28] For some patients whose symptoms are suspicious for a suprascapular nerve lesion, a diagnostic injection can be considered. Injection of the suprascapular notch, for example, may provide excellent alleviation of pain. However, by itself, this test is not specific for a nerve lesion as patients with other types of shoulder pathology may also find pain relief from a suprascapular nerve block.[71,72,78]

Electrodiagnostic Studies

A clinical suspicion for a suprascapular nerve injury should be confirmed with electrodiagnostic studies. The mean normal latencies between Erb's point and points in the supraspinatus and infraspinatus muscles have been determined to be between 2.7 and 3.3 milliseconds, respectively. Increased latency on nerve conduction velocity studies may signify a nerve lesion such as entrapment.[48,50] Denervation injuries typically reveal increased spontaneous activity, polyphasic signals, muscle fibrillations, positive sharp waves, and amplitude reduction on electromyography (EMG).[16,48,71] It must be noted, however, that these tests are both invasive and operator dependent. In addition, positive results will not always accurately locate the lesion or define the pathology.[22,71,78] In one study of patients with preexisting denervation injuries, preoperative EMG changes were predictive of patient outcome after nerve exploration and/or repair. Surprisingly, fewest improvements were observed in patients with minor preoperative EMG changes. The authors hypothesized that more severe denervation was usually associated with an identifiable pathology that can be addressed at the time of surgery and that less severe denervation may not improve with nerve decompression alone.[3]

Imaging Studies

Several imaging modalities can be utilized to identify the potential sites of nerve injury. Standard radiographs of the cervical spine and the shoulder are typically the first imaging studies to be evaluated. When clinically indicated, dedicated clavicle views may be useful. A Stryker notch view, an anteroposterior view of the scapula with the beam directed caudally 15 to 30 degrees, allows visualization of

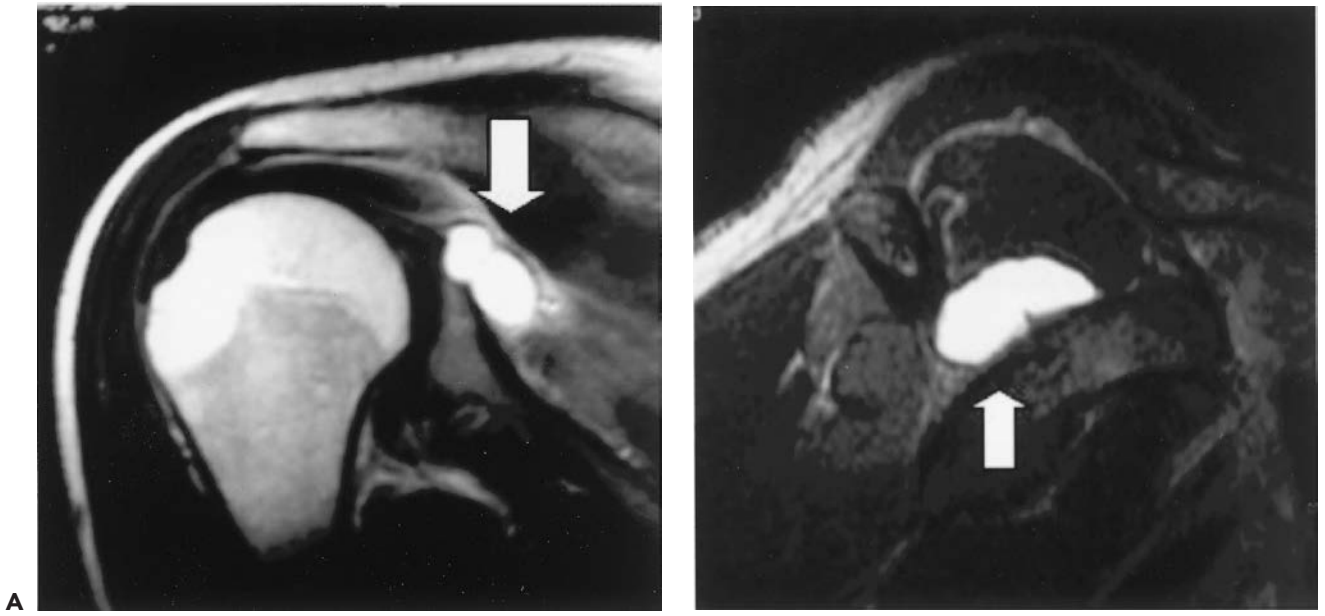


Figure 36-4 Magnetic resonance imaging of a ganglion cyst. Coronal **(A)** and sagittal **(B)** views of T-2 weighted images clearly demonstrate a well-circumscribed homogeneous high-signal intensity mass, consistent with a fluid-filled cyst.

the suprascapular notch. Radiographs are particularly useful for patients with a suspected fracture. In the acute setting, fracture lines may be observed near the anatomically sensitive regions. In more chronic cases, callous or fibrous tissue may compress or entrap the nerve. For the majority of patients, however, radiographs are likely to be unremarkable.[22,70,78] A computed tomography (CT) scan offers better delineation of the osseous architecture. Unfortunately, similar to radiographs, CT scans are often normal and provide little additional diagnostic value.[41]

For most patients with a nerve injury, magnetic resonance imaging (MRI) often provides the most valuable information. MRI allows clear identification of various intraarticular and extraarticular soft tissue pathologies.[41] In addition, it is also particularly effective in identifying soft tissue masses that can injure the suprascapular nerve.[32] Ganglion cysts, for example, are clearly visible on the MRI (Fig. 36-4). On the T-2 weighted images, these cysts are seen as high-signal, well-defined masses originating from the posterosuperior aspect of the glenohumeral joint.[22,33,41,45] If unclear, gadolinium can enhance the visualization of the cyst by increasing the signal intensity of its outer rim.[22] Denervation changes within the muscle mass, such as fatty infiltration and decreased mass, are also well defined on the MRI.[32,45,51] Denervated muscles typically show high-signal changes on the T-2 weighted images and, according to one study, the degree of denervation correlated with the degree of signal intensity.[45] On some MRIs, the actual nerve can be visualized. T-2-weighted oblique sagittal views, for example, can demonstrate the suprascapular nerve as it courses through the suprascapular fossa.[78]

If an MRI is not readily available, ultrasonography may be utilized to identify ganglion cysts and other masses around the shoulder. A ganglion cyst will appear as a hypoechoic, well-defined, homogeneous mass.[39] Ultrasound may also detect muscle atrophy consistent with denervation.[51] However, while ultrasonography offers a cost-effective alternative, it is operator dependent and may not be an accurate imaging modality in the hands of inexperienced operators.

Nonoperative Treatment

A trial of nonoperative therapy is typically the initial treatment of choice for patients with a suprascapular nerve injury. In the majority of patients who do not have a well-defined lesion, most of the symptoms will resolve spontaneously.[57,69] Unfortunately, however, a significant amount of time, perhaps as long as 12 months, is often required for a complete resolution of symptoms.[78] Therefore, patient counseling is a key component to nonoperative treatment. In addition, patients should be instructed to avoid repetitive overhead activities that can cause trauma and irritation to the nerve. Physical rehabilitation should focus on regaining and improving the flexibility, strength, and endurance of the shoulder girdle musculature. The scapular stabilizers are of particular importance in reestablishing proper glenohumeral and scapulothoracic kinematics.

The overall success rate of nonoperative management, however, is not well established in the literature and can vary widely among individual reports. For example, one study reported that all patients in the small series were

successfully treated with a home exercise program, while another series reported that all patients in the study failed nonoperative therapy and required surgical intervention.[27,71] In a retrospective review of 15 patients, Martin et al. reported that five excellent and seven good results were obtained after nonoperative management. The remaining three patients (20%) continued to suffer from persistent symptoms and required surgical management.[57]

Despite these conflicting reports, the likelihood of successful nonoperative management appears to depend primarily on the cause and location of the nerve lesion.[22,78] In general, patients with a distal nerve injury without a discrete lesion respond favorably to nonoperative management. For example, one study reported that most patients with an isolated infraspinatus muscle atrophy were successfully treated nonoperatively as they obtained good pain relief, increased muscle mass, and improved strength at 6 to 12 months.[22] Another study by Ferretti et al. reported similar results as they reviewed their experience with 38 professional volleyball players who were suffering from isolated infraspinatus atrophy.[29] Thirty-five patients (92%) responded well to nonoperative treatment that consisted of physical rehabilitation focusing on external rotation strengthening exercises. The authors noted that isolated infraspinatus muscle atrophy did not affect the competitive performances of these athletes and that surgical intervention should be reserved for those patients suffering from persistent pain.[29]

Nonoperative management of patients with a well-defined lesion, particularly ganglion cysts, appears to be associated with unfavorable outcomes.[2,22,28,57] Cummins et al. performed a review of the literature and identified 21 cases of suprascapular nerve injuries secondary to ganglion cysts that were managed nonoperatively. Five of these patients had complete resolution of their symptoms and

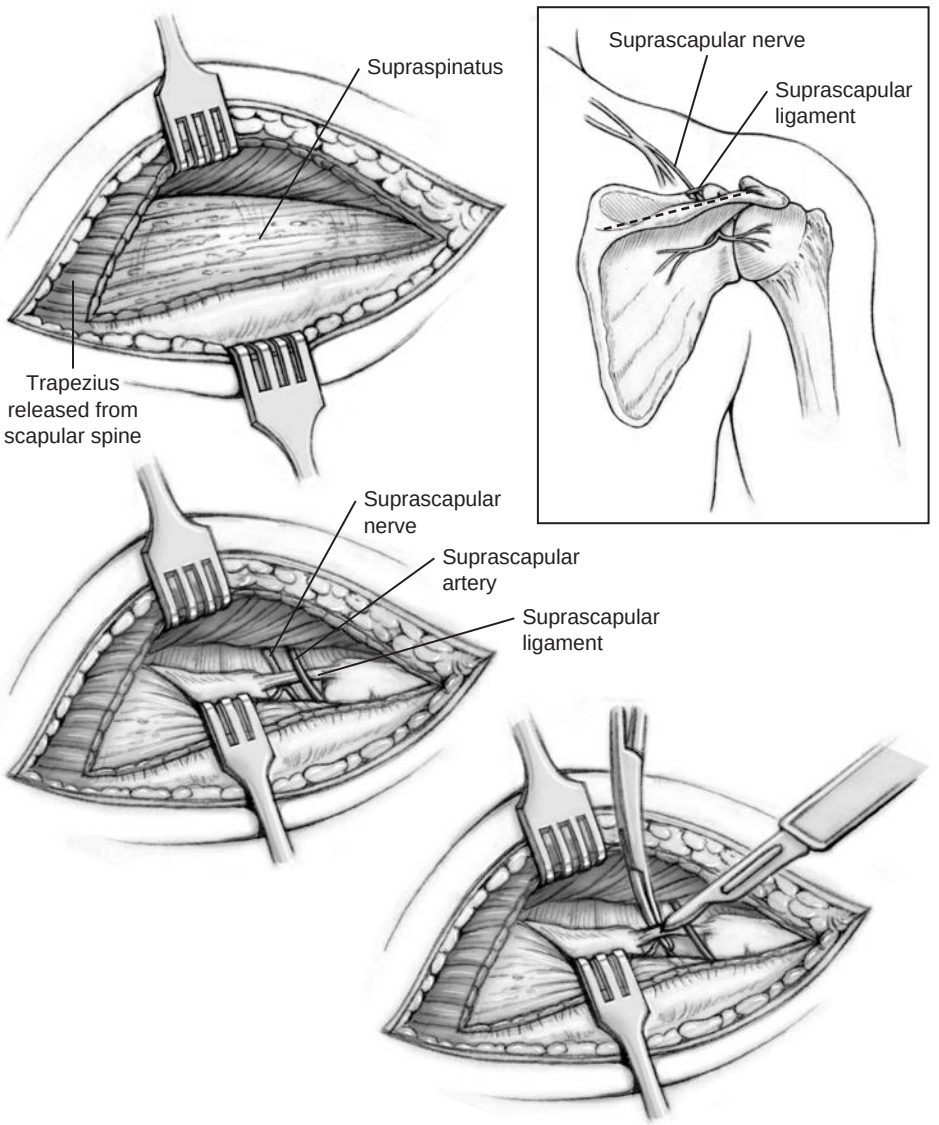


Figure 36-5 Surgical approaches for suprascapular nerve decompression. The nerve is commonly explored using either a posterior **(A)** or a superior **(B)** approach (refer to text for details).

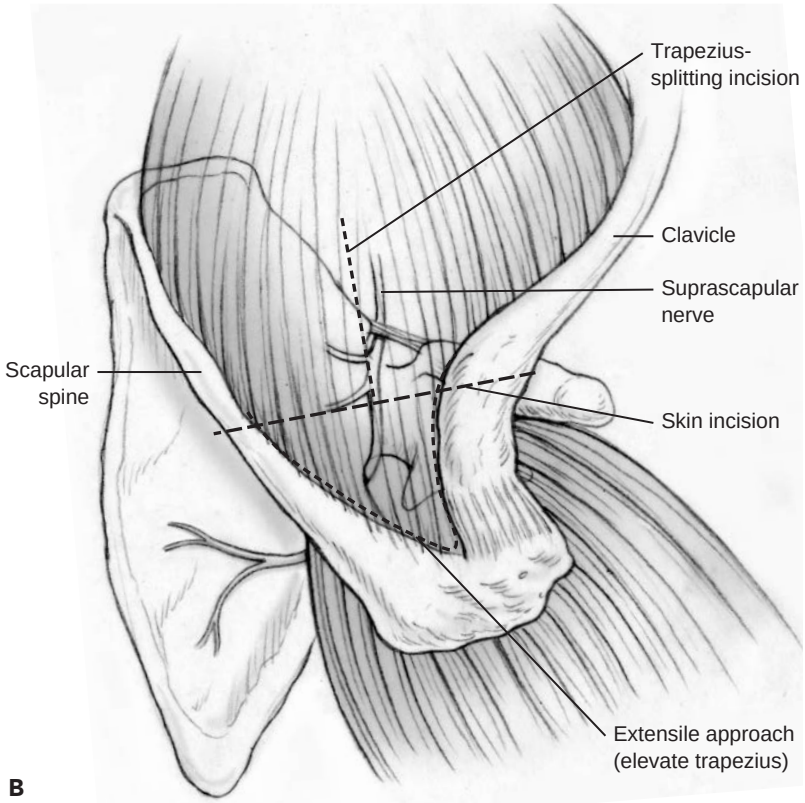
another experienced partial pain relief. The remaining 15 patients (71%) had "unsuccessful" results, with nine patients requiring surgical decompression.[22] Thus, although these patients with a discrete lesion can be initially managed with nonoperative therapy, they should be counseled that they are less likely to have a successful outcome and that they may require surgical intervention.

For some of these patients with a documented ganglion cyst, ultrasound or computed tomography guided aspiration may be useful.[32,39,86] In their review of the literature, Cummins et al. identified eight such cases, and found that these patients all reported satisfactory pain relief at short-term follow-up.[22] The theoretical concern regarding guided aspiration is that it does not allow an opportunity to address the intraarticular pathology involving the glenoid labrum. Hence, over time, the cysts may recur. According to one study, for example, the rate of recurrent cyst formation after aspiration was as high as 48%.[40] Therefore, while promising, further experience with guided aspiration is necessary to document its long-term clinical efficacy.

Operative Treatment: Open

Exploration and decompression of the suprascapular nerve have been the surgical treatments of choice. The surgical approach is usually dictated by the location and cause of the lesion (Fig. 36-5). While an anterior approach to the suprascapular notch has been described, it has not been routinely utilized due to the difficult dissection, increased risk of neurovascular injuries, and poor visualization of the nerve.[62,71,78] Therefore, the majority of the surgeons tend to use either the posterior or the superior approaches to the nerve.

Initially described by Post and Mayer, the advantages of the posterior suprascapular nerve decompression include excellent exposure, limited risk to the neurovascular structures, and avoidance of major muscle damage.[22,70,71] For this approach, the patient is placed in a semiprone position with the arm draped free. The skin incision is made just superior, but parallel, to the scapular spine. The underlying trapezius muscle is then sharply elevated and retracted to expose the supraspinatus muscle. Once the supraspinatus muscle is retracted inferiorly, the superior transverse scapular ligament can be visualized. If the ligament is the source of the nerve lesion, it can be excised in its entirety. Otherwise, it is incised to expose the nerve and any compressing structures. Care should be taken to ensure that the vascular structures that lie just superior to the ligament are not injured. It has also been suggested by some authors that isolated ligament resection may be insufficient to decompress the nerve and that the nerve can still be tethered by its course over the notch.[73] Therefore, in addition to ligament release, all resected edges of bone as well as the notch should be contoured to minimize any potential damage to the nerve.[78]



B

Figure 36-5 *(continued)*

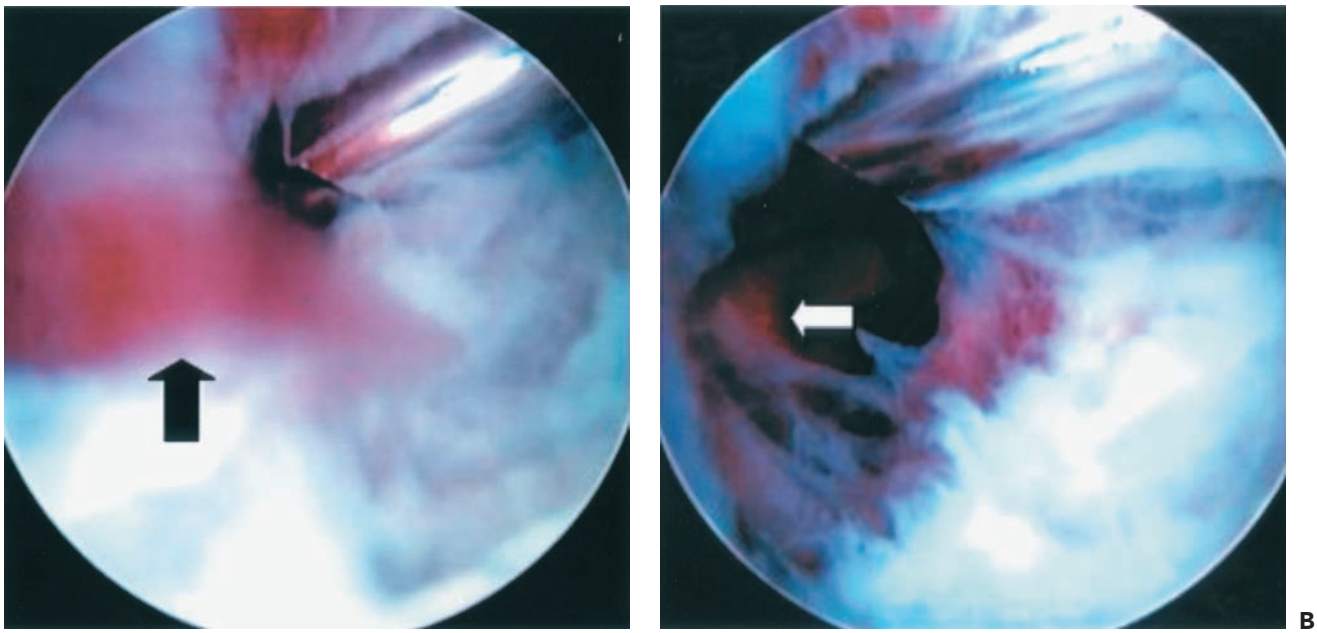


Figure 36-6 Arthroscopic decompression of a ganglion cyst. After incising the superior glenohumeral joint capsule and decompressing the cyst **(A),** gelatinous cyst material (*arrow*) is commonly encountered. Upon completion of the decompression **(B),** the suprascapular nerve may also be visible (*arrow*).

For patients with an isolated ganglion cyst, an alternative posterior approach can be used. While the patient is positioned in a similar manner, the incision is made along Langer's lines 3 cm medial to the posterolateral corner of the acromion. The deltoid muscle is then split in line with its fibers. Once identified, the infraspinatus muscle can then be retracted inferiorly to reveal the ganglion cyst and the terminal branches of the nerve.

Another commonly utilized technique for suprascapular nerve decompression is the superior trapezius splitting approach. The patient is positioned in a beach-chair or lateral position. The skin incision begins 2 cm medial to the acromioclavicular joint and follows Langer's line to the distal third of the scapular spine. The trapezius muscle is then split in line with its fibers to expose the scapula. For a more extensile exposure, the trapezius muscle can be sharply elevated off of the bone and then later repaired using bone tunnels.[61] By identifying and then retracting the supraspinatus muscle inferiorly, the superior transverse scapular ligament is visualized. The ligament is transected and the underlying pathology can be addressed.

Operative Treatment: Arthroscopic

Arthroscopic management of a suprascapular neuropathy has recently gained significant attention due to the high association between this pathology and labral tears.[40,86] Commonly associated labral pathology includes fraying as well as frank tears. Arthroscopic techniques are routinely utilized to address these pathologies. Once the labral pathology is addressed, the ganglion cyst can then be isolated and decompressed by either open or arthroscopic techniques[28,44,78] (Fig. 36-6). Various arthroscopic techniques for cyst decompression have been described. Iannotti and Ramsey, for example, recommended performing a posterosuperior capsulotomy to approach the cyst. If clearly identified, the cyst can then be excised in its entirety. If not, the cyst can be drained into the joint by manually applying pressure to the posterior aspect of the joint.[44] Romeo et al. described another similar technique to identify and excise the cyst. For their approach, however, accessory posterolateral and anterosuperior portals were often necessary to clearly visualize and decompress the cyst.[78] It should be noted, however, that arthroscopic procedures can be technically demanding. Clear identification and visualization of the cyst is crucial to a successful excision. With the nearby suprascapular nerve, poorly performed decompression may actually increase the risk of further neurologic injury.

Postoperative Considerations

Upon completion of the procedure, patients are immobilized in a simple sling for comfort. They are allowed to remove the sling for hygiene purposes as well as to perform daily stretching exercises. As soon as their pain is tolerable, typically 10 to 14 days after the surgery, the immobilization is removed and active motion exercises are instituted.[70] The only exceptions are patients in whom a superficial muscle had to be detached from the bone for surgical

exposure. In such situations, active motion is typically instituted 4 to 6 weeks after the surgery. Once active exercises are initiated, it is important to restore normal kinematics of the shoulder girdle and the periscapular musculature. The majority of patients experience pain relief soon after the procedure. Muscle atrophy and weakness, however, improve gradually over 3 to 6 months.[36,71]

Results and Complications

Although the pathology and the diagnosis are well established, the small number of surgical cases has limited the amount of available information in the literature. In general, however, open surgical treatment of patients with a suprascapular nerve lesion is associated with a good clinical outcome. Callahan et al., for example, evaluated 23 patients who underwent suprascapular ligament release and open nerve decompression and found that 91% of the patients experienced immediate pain relief. The symptoms recurred in four patients, with three requiring a second operation. Overall, they noted that long-term pain relief and resolution of weakness were obtained in 87% of the patients.[16] Similarly, Vastamaki and Goransson reported on their experience of treating 54 patients with a surgical release of the suprascapular ligament. Immediate pain improvement was observed in 39 (72%) patients, with 24 (44%) patients having no residual pain and 15 (28%) having a notable decrease in pain. Improvement in muscle atrophy, however, was more variable. Only 1 of the 16 patients with a preoperative *supraspinatus* atrophy showed any residual atrophy. In contrast, of the 26 patients with a preoperative *infraspinatus* muscle atrophy, 11 noted residual atrophy. The authors conceded that they did not address any pathology at the spinoglenoid notch that could have contributed to the residual atrophy of the infraspinatus muscle.[90] Finally, Post reviewed his experience with 39 patients who had a surgical release of the suprascapular nerve. Twenty-seven patients (69%) had excellent results and returned to work at an average of 3.3 months after the operation. An additional 11 patients (28%) experienced good results, and only one patient had a fair result. The author noted that residual muscle atrophy correlated inversely with a delay to surgical intervention. Therefore, patients with a longstanding and severe muscle atrophy were unlikely to regain full muscle strength.[71]

Similar findings were noted for patients who underwent an operative excision of the ganglion cyst at the spinoglenoid notch. These patients were also able to experience predictable pain relief with variable return of muscle strength.[22,29,37,80] Fehrman et al., for example, reported complete pain relief in five of the six patients who underwent combined arthroscopy and open excision of the cyst. While these patients subjectively felt that their shoulders were normal, objective examination revealed persistent muscle weakness at short-term follow-up.[28] Similarly, Moore et al. reported on 16 patients who underwent either open or arthroscopic excision of the ganglion cyst at the spinoglenoid notch. All but one patient experienced a good to excellent result. The remaining patient with a poor result was subsequently found to be suffering from a missed labral tear. Upon repair of the tear, this patient then experienced a good overall result as well.[61] Finally, Hawkins et al. reported on their experience with 73 patients who were suffering from symptomatic spinoglenoid ganglion cysts. Patients were treated nonoperatively, by needle aspiration, or by arthroscopic decompression. The authors noted that the arthroscopically treated group had the best outcome in terms of pain relief, resolution of atrophy, and return of strength.[40]

AXILLARY NERVE INJURIES

Anatomy

The axillary nerve is a mixed sensorimotor nerve originating from the fifth and sixth cervical nerve roots. It is a terminal branch of the posterior cord of the brachial plexus. Initially, the nerve runs posterior to the axillary artery along the anterior surface of the subscapularis muscle. Typically, it courses obliquely across the inferior border of the muscle, 3 to 5 mm medial to the musculotendinous junction. The nerve then turns posteriorly, entering the quadrilateral space with the posterior humeral circumflex artery. Anterior to the glenohumeral joint, the boundaries of the quadrilateral space consist of the humerus, the triceps longus, the teres major, and the subscapularis. Posterior to the joint, the superior boundary of the quadrilateral space is replaced by the teres minor (Fig. 36-7).[66,85]

During its passage under the glenohumeral joint, it divides into two discrete trunks. The posterior trunk lies medial to the anterior trunk and runs adjacent to the inferior rim of the glenoid. One of its terminal branches enters and innervates the teres minor muscle. Another terminal branch, the superolateral brachial cutaneous nerve, runs inferiorly deep to the posterior deltoid fibers passing around the medial border of the deltoid. In most patients, this branch can be identified approximately 9 cm inferior to the posterolateral corner of the acromion. The last terminal branch of the posterior trunk innervates the posterior deltoid muscle. In a cadaveric study, this branch had some variations in its origin. Rather than arising directly from the posterior trunk, it occasionally originated from the superolateral brachial cutaneous nerve.[5]

The anterior trunk of the axillary nerve circles posterolaterally around the surgical neck of the humerus, traveling on the deep subfascial surface of the deltoid muscle toward the anterior deltoid. Along its course, the nerve generates several branches to the lateral and anterior deltoid muscle.[66,85] In most patients, the nerve can be located 4 to

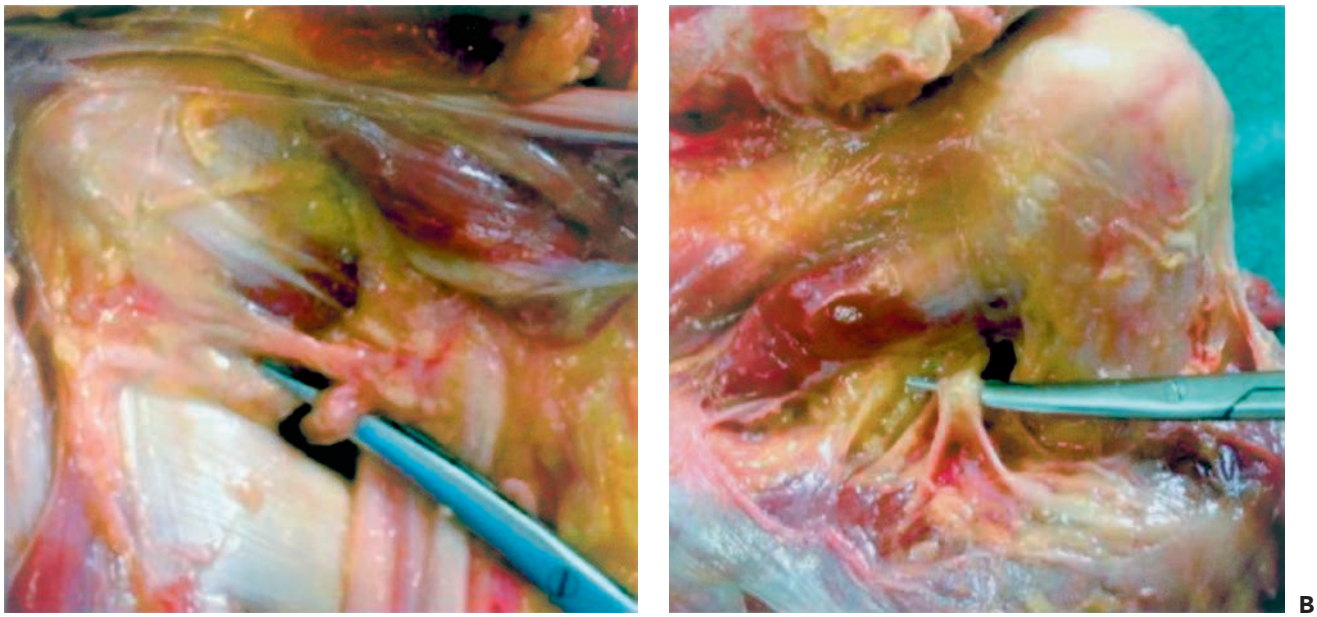


Figure 36-7 Anatomy of the axillary nerve about the scapula. On the anterior aspect of the shoulder **(A),** the nerve runs inferior to the subscapularis muscle before entering the quadrangular space. During its passage within the quadrangular space, the nerve divides into two discrete trunks, which, in turn, separate into additional terminal branches. Therefore, as the nerve exits the quadrangular space in the posterior aspect of the shoulder **(B),** several discrete branches may be seen.

7 cm inferior to the anterolateral corner of the acromion.[13] Based on this anatomy, the axillary nerve is believed to be susceptible to injury at the following locations: (1) its origin from the posterior cord, (2) along the inferior edge of the clavicle, (3) along the inferior aspect of the glenohumeral joint, (4) at the posterior quadrilateral space, and (5) within the subfascial surface of the deltoid.[66]

Classification and Etiology

Similar to the suprascapular nerve, injuries to the axillary nerve may be classified according to the degree of intraneural injury or by location and cause. Most commonly, axillary nerve injuries are discussed in the context of cause. Traumatic mechanisms for injury include direct nerve lacerations, blunt nerve trauma, and traction injuries. Glenohumeral dislocations, for example, have been associated with axillary nerve injuries. Although the true incidence of axillary nerve injuries after glenohumeral dislocations is largely unknown, some authors have reported rates of less than 5% for patients younger than 60 and up to 9% in patients older than 60.[34] These rates were based on clinical findings alone. With electrodiagnostic analysis, however, Toolanen et al. found that 54% of the patients with traumatic anterior glenohumeral dislocations showed EMG changes consistent with an axillary nerve injury.[87] The duration of dislocation appears to be an independent risk factor for axillary nerve injuries, as one report demonstrated that the likelihood of axillary nerve injuries increased significantly if the shoulder was dislocated for more than 12 hours.[65] According to Milton, axillary nerve injury after glenohumeral dislocations is a result of a combined traction and compression.[60] Another common cause of traumatic nerve injury is proximal humeral fractures. Unfortunately, due to pain and associated guarding, axillary nerve injuries may be difficult to identify in patients with acute fractures and is likely to be underreported in this setting.[9]

Blunt trauma and traction injuries have also been observed to result in axillary nerve injuries during contact sports.[6,65,67] Direct blows to the anterolateral deltoid can result in a compression injury to the anterior branch of the axillary nerve as it courses along the humerus in the subfascial deltoid. There may also be a component of traction injury with this mechanism, likely in the region of the quadrilateral space, as many of these patients have weakness of the posterior deltoid as well as sensory deficits along the distribution of the superolateral brachial cutaneous nerve. Additionally, some athletes suffer from axillary nerve injuries following shoulder depression and contralateral neck flexion, which would clearly indicate a traction mechanism.[6,65,67]

Compression of the axillary nerve and posterior circumflex humeral artery within the quadrilateral space has been described as an entity called the quadrilateral space syndrome (QSS).[14] It has been postulated that this syndrome is caused by fibrous bands and hypertrophic muscles that surround the quadrilateral space. As a result, static as well as dynamic compression of the nerve can result in the

clinical presentation of pain and weakness.[14,31,66] Other, and less common, causes of compressive axillary nerve injuries include tumors and aneurysms.[85]

Finally, the axillary nerve can be damaged as a result of an iatrogenic injury during surgical procedures. Open shoulder stabilization procedures, for example, involve exploration of the inferior glenohumeral joint and may place the nerve at risk for injury as it courses along the inferior glenoid rim. Potential mechanisms of injury include tension, suture compression, or direct laceration.[66] In a series of 40 patients who underwent inferior capsular shift procedures, Neer and Foster reported three cases of axillary neurapraxias despite careful protection of the nerve during the surgery.[64] To decrease the likelihood of an iatrogenic axillary nerve injury, some authors have recommended placing the shoulder in external rotation such that capsular incision can be placed more laterally. They also recommended that a blunt retractor be placed superior to the nerve to protect it from sharp instruments.[66] Recently, use of the subscapularis splitting technique has become popular as a method of limiting axillary nerve injury during anterior shoulder stabilization procedures.[47,58] One study examining the use of the subscapularis splitting approach found that only 1 of 128 patients had a temporary axillary neuropathy, and that none had any permanent deficit.[58]

Other commonly performed procedures that have been associated with axillary nerve injuries include open rotator cuff repairs and various arthroscopic procedures. The axillary nerve is vulnerable to injury during open rotator cuff repair surgery if the deltoid splitting approach is used and if this split is carried too inferiorly. As described previously, most surgeons limit the deltoid splitting to within 5 cm of the acromion as the axillary nerve is typically located inferior to this interval.[66] If the deltoid split is carried out more anteriorly, this safe interval of splitting may need to be shortened, as one cadaveric study found that terminal branches course more superiorly in the anterior deltoid.[11] During arthroscopic procedures, the axillary nerve can be injured during the creation and the establishment of a posterior portal. During normal placement of the posterior portal, the trocar may be only 5 to 25 mm from the nerve.[11] As such, the portal must be placed carefully using normal anatomic landmarks. Fortunately, despite this close proximity to the nerve, the incidence of axillary nerve injury after arthroscopy is believed to be quite rare.[2]

Clinical Symptoms

In traumatic situations, patients tend to focus on the primary injury. Therefore, subjective weakness and sensory deficits may be ignored or disregarded in the acute setting. When pain from the initial trauma has subsided, complaints regarding persistent weakness and numbness are voiced. Even in this stage, however, the clinical presentation of axillary nerve injuries can be quite variable. Patients may complain of weakness, numbness, or a combination of both. Although an isolated sensory defect is more common, an isolated motor deficit with normal sensation is also possible. Additionally, these deficits may be complete or partial.[9]

Similar to above, clinical presentation of nontraumatic axillary nerve injuries can also be quite variable. Patients may present with varying degrees of pain, numbness, and weakness that can be insidious or acute in onset. In QSS, for example, patients will often complain of chronic, dull, and aching pain in the posterolateral aspect of the shoulder and arm. This may be associated with subjective deltoid weakness, which can then progress to clinical weakness.[14,66,85] Parsonage-Turner syndrome, in contrast, will present as an episode of severe posterior shoulder pain without prior trauma. The pain is usually short-lived and resolves spontaneously. Upon its resolution, the patient will then develop profound weakness.[89] Although the symptoms may be limited to the axillary nerve, in most cases, Parsonage-Turner syndrome will involve multiple branches of the brachial plexus.

Clinical Evaluation

To document or eliminate the possibility of cervical radiculopathy or brachial plexopathy, patients with a suspected axillary nerve injury should undergo a thorough cervical spine and upper-extremity examination. As a part of the initial inspection, any particular asymmetry in the shoulder contour should be noted. Although acute axillary nerve injuries may not demonstrate changes, chronic denervation can be associated with significant deltoid atrophy (Fig. 36-8). Particular attention should be given to identifying specific denervation patterns as they may suggest the location of the injury. Thus, for example, specific atrophy of the lateral and anterior deltoid muscle with normal posterior deltoid would suggest a lesion distal to the quadrangular or quadrilateral space.

Sensory examination should concentrate on the distribution of the superior lateral cutaneous nerve. Sensation to light touch as well as pinprick should be documented. It should be noted that axillary nerve injuries with motor deficit can exist with intact sensation.[9] Although most patients with an axillary nerve injury do not demonstrate any focal tenderness, patients with chronic QSS may exhibit some tenderness on the posterior aspect of the shoulder joint.[85]

All motion of the shoulder joint should then be examined. Both passive and active motion must be documented. Although axillary nerve injuries should not cause any loss of passive motion, some patients with a chronic lesion may demonstrate mild stiffness in the shoulder. Strength testing should be performed for shoulder flexion, extension, rotation, and abduction. The deltoid muscle plays a significant role in shoulder abduction, but only a minor role in flexion and extension and a minimal role in

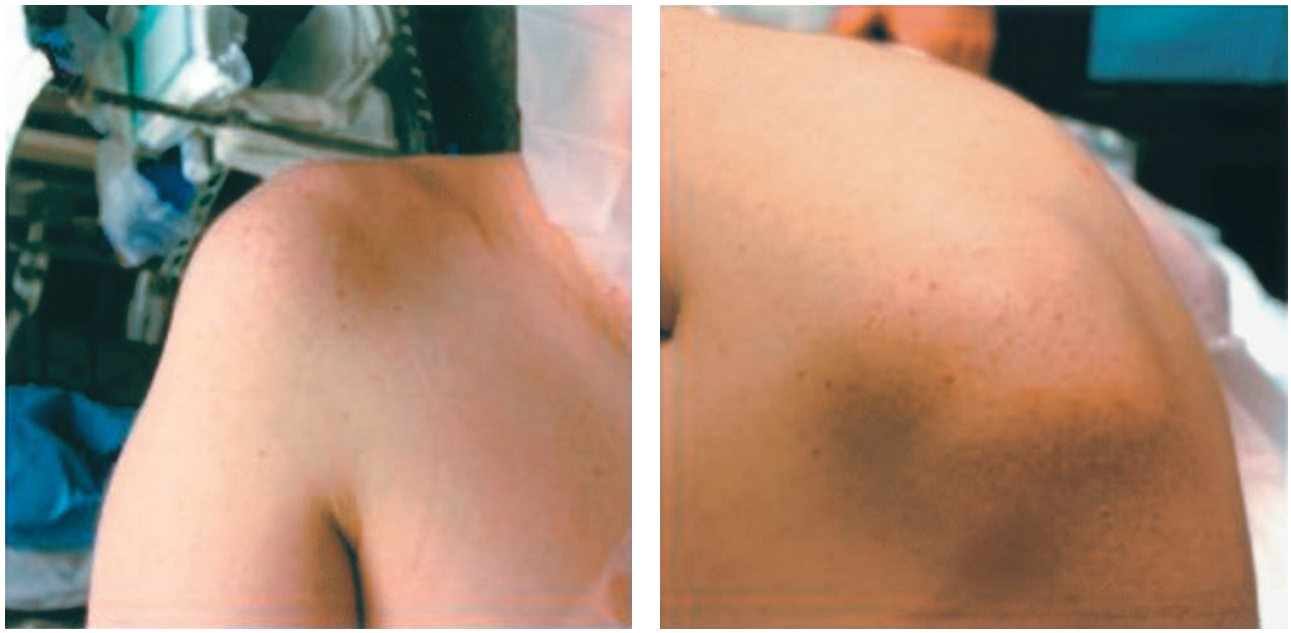


Figure 36-8 Clinical picture of deltoid muscle atrophy due to an axillary nerve lesion at the quadrangular space. Note that the overall contour is lost from both anterior **(A)** and posterior **(B)** aspect of the shoulder.

shoulder rotation. Therefore, shoulder abduction strength and endurance is most often affected in patients with axillary nerve injuries. Selective axillary nerve block studies have demonstrated that the deltoid muscle is responsible for approximately 50% of the generated torque about the shoulder.[19] Nevertheless, studies have also shown that most patients can compensate for the loss of deltoid function. By adapting the use of other shoulder musculature, including the rotator cuff, pectoralis major, and coracobrachialis muscles, full active motion may be possible.[4,12,25,66,85] Thus, full active motion of the shoulder does not eliminate the possibility of a nerve injury.

Electrodiagnostic Studies

A clinical suspicion for an axillary nerve injury should be confirmed with electrodiagnostic studies. Increased latency on the nerve conduction velocity examination can confirm a nerve injury. In addition, EMG changes such as increased spontaneous activity, polyphasic signals, muscle fibrillations, and amplitude reduction may demonstrate muscle denervation.

Once obtained, these studies can provide valuable insight into the location of the lesion as well as any associated brachial plexus injuries. They can also establish a reference point to assess recovery. For acute cases with no clinical evidence of recovery, these tests should be performed within 2 to 4 weeks from the injury.[85] For chronic cases, they should be obtained as a part of the initial evaluation. The studies should then be repeated after a period of 3 to 4 months to identify any signs of recovery.

Imaging Studies

All patients should obtain standard radiographs of the shoulder and cervical spine to rule out any abnormal osseous anatomy.[85,95] If a bony abnormality is suspected, CT scans should be considered, as they can further characterize the bony anatomy. In the majority of patients with an axillary nerve lesion, however, the bony architecture will be unremarkable.

Analysis of the soft tissues can be greatly aided by MRI and, therefore, this study is generally recommended for patients with a suspected axillary nerve injury. Soft tissue masses that are compressing the axillary nerve can be visualized with an MRI. In addition, for chronic nerve injuries, involved muscles will demonstrate abnormal signal changes consistent with atrophy and fatty infiltration.[88] In patients with QSS, for example, the teres minor and the posterior deltoid muscles should demonstrate such changes.[55] The diagnosis of QSS can then be confirmed with a subclavicular arteriogram, which will show limited flow through the posterior circumflex humeral artery with shoulder abduction and external rotation.[14,31]

Nonoperative Treatment

Based on the available information in the literature, the natural history of axillary nerve lesion appears to be quite variable. For example, injuries associated with glenohumeral dislocations have rates of recovery that vary from 17% to 100%.[34,54,87,92] Nerve injuries from blunt trauma to the shoulder tend to exhibit less favorable recovery rates.[66]

Penetrating injuries and traumatic avulsion injuries possess even less favorable outcomes. Nevertheless, some patients with persistent axillary injuries can compensate sufficiently to obtain full active motion and the ability to perform activities of daily living. Therefore, nonoperative management of axillary nerve injuries should be the initial treatment of choice in most patients.

After the acute phase of injury, with dissipating pain, range-of-motion exercises can commence. Restoration of motion and avoidance of contractures are essential to obtaining a good outcome. For patients with other shoulder pathology such as rotator cuff tear or a fracture, regaining full motion can be a difficult task.[66] Once the overall level of pain and motion has been stabilized, strengthening exercises can begin. These exercises are utilized to augment the power provided by the remaining musculature of the shoulder girdle. As such, the regimen should concentrate on the rotator cuff muscles as well as the scapula stabilizers. Although these general principles are clear, no single rehabilitation protocol has gained universal acceptance in the nonoperative treatment of patients with axillary nerve injuries. Therefore, specific rehabilitation protocol and physical therapy must be individualized to each patient and injury pattern.

Operative Treatment: General Principles

Operative management of axillary nerve injuries is typically reserved for patients who remain symptomatic and demonstrate no evidence of recovery based on both the clinical examination and the repeated electrodiagnostic studies. Although a general consensus does not exist, surgical intervention can also be considered after an acute penetrating injury. The type of surgery is dictated by multiple factors including patient expectations, likelihood of recovery, nature of the injury, location of the lesion, and chronicity of the symptoms. As such, a number of different procedures such as decompression, neurolysis, grafting, neurotization, and muscle transfers have been used with varying degrees of success. Therefore, as stated previously, the choice for a specific procedure must be individualized to each patient and injury pattern.

Operative Treatment: Decompression

Axillary nerve decompression is utilized in situations where a discrete compressing structure can be identified. Thus, this is typically the procedure of choice for patients with QSS. Although other surgical approaches are available, the quadrilateral space is best exposed using the posterior approach (Fig. 36-9). The skin incision starts just medial to the posterior axillary crease and parallels the inferior border of the scapular spine. Once past the axillary crease, the incision is then curved distally along the posterior aspect of the humerus. Full-thickness skin flaps are then raised to expose the deltoid fascia. The posterior deltoid muscle is then released from its origin on the scapula spine. During closure, this muscle must be adequately repaired back onto its origin, using bone tunnels if necessary. After reflecting the posterior deltoid muscle, the underlying rotator cuff muscles are exposed. The inferior border of the teres minor is carefully dissected and the entire muscle is then retracted superiorly. An alternative approach is to release a portion of the teres minor from the greater tuberosity and then reflecting it medially. Although most surgeons would repair the released tendon back onto its insertion on the greater tuberosity during closure, Cahill and Palmer noted no postoperative weakness even when the tendon was not repaired.[14] Once the neurovascular structures are identified, they are isolated and safely retracted out of the surgical field. All borders of the quadrilateral space, including its anterior compartment, should then be explored to identify any compressing lesions. After excising these lesions, the posterior humeral circumflex artery can be palpated while the arm is abducted and externally rotated. Adequate decompression is thought to be achieved only if the pulse remains in this position. Using this similar exposure, surgeons have also performed neurolysis of the axillary nerve in patients whose nerve was encased and compressed by fibrotic scar tissue.[68]

Operative Treatment: Nerve Grafting or Neurotization

In cases of acute lacerations, neurorrhaphy may be performed. By several weeks after the injury, however, retraction and scarring of the nerve endings may preclude a primary end-to-end repair. In addition, in patients with a blunt trauma that requires excision of a nerve segment or in patients where a neuroma had to be excised, a significant gap may be present between the nerve endings. In these situations, rather than trying to mobilize the remaining nerve, which has minimal excursion, nerve grafting may be the best available surgical option. As expected, exposure for this procedure is dependent on the site of the injury. In patients whose nerve deficit is likely to reside within the quadrilateral space, a combined anterior and posterior approach to the nerve may be necessary. For this exposure, the patient is placed in a lateral decubitus position. The posterior approach is performed in a manner described previously. For the anterior approach to the quadrilateral space, a skin incision is made along the anterior border of the clavicle and then curved distally along the deltopectoral interval. The deltopectoral interval is then developed to expose the subscapularis muscle. The neurovascular bundle can be readily identified in the anterior quadrilateral space just inferior to the glenohumeral joint on the inferior border of the subscapularis muscle. If needed, the pectoralis minor

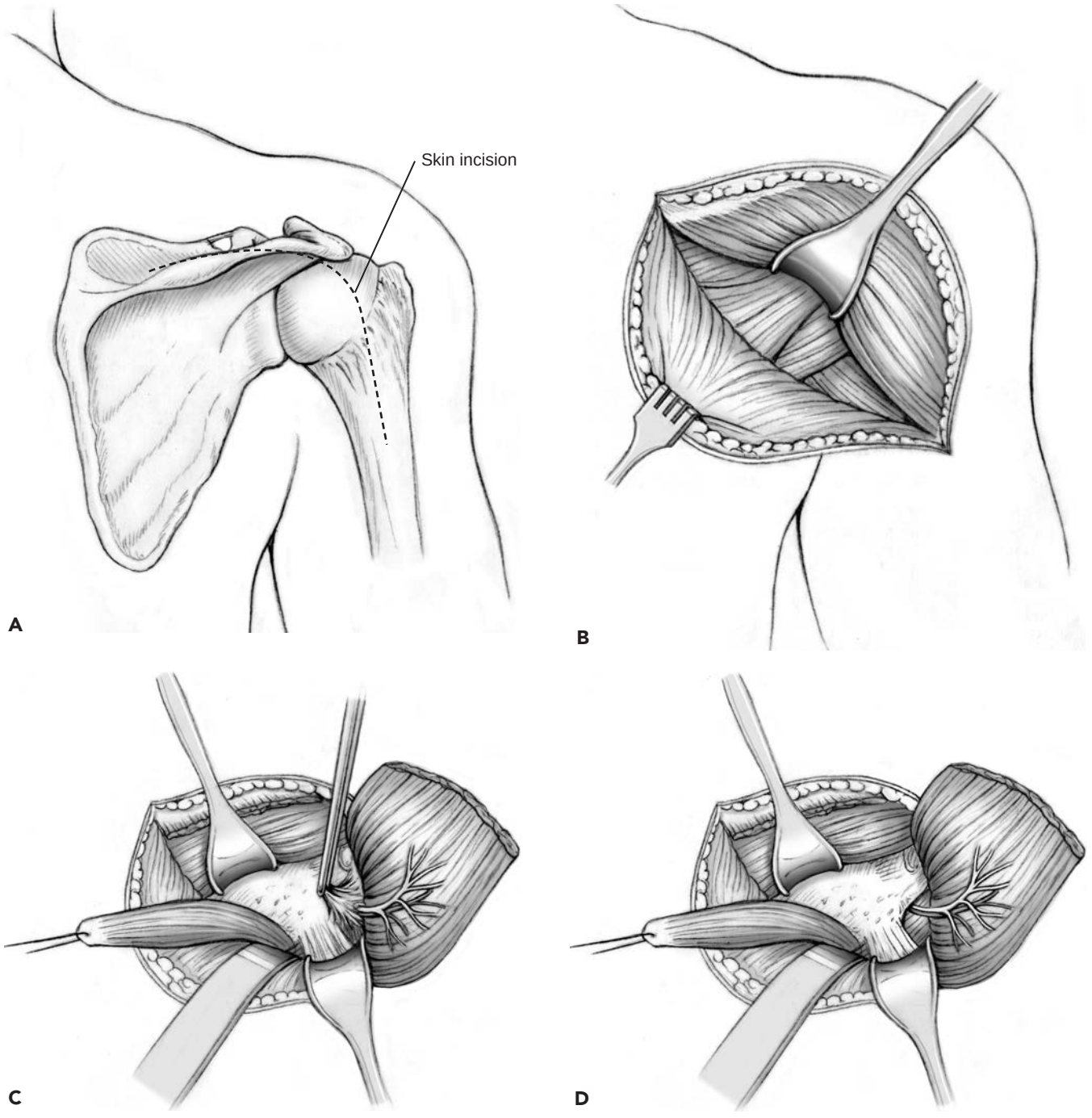


Figure 36-9 Surgical approaches for axillary nerve decompression at the quadrangular space (refer to text for details).

muscle can be detached from the coracoid process and reflected medially to expose the origin of the axillary nerve from the posterior cord.[68] The released pectoralis minor should be anatomically repaired during closure. After identifying this origin, the nerve can then be traced to its location on the entrance of the quadrilateral space. Once the lesion is identified and the gap is estimated, a nerve graft of appropriate length is obtained. Although multiple sources of autologous nerve grafts are available, most surgeons favor the use of sural nerve grafts. The graft is then sutured on to the nerve endings. Care must be taken to ensure that minimal tension is placed on the repair throughout all shoulder motion.

With a similar surgical exposure, some surgeons have performed neurotization of the axillary nerve with mixed results.[17,23] These procedures have been performed

with a number of different graft sources that include the thoracodorsal, spinal accessory, and intercostal nerves. Recently, an anatomic dissection study and case series have presented a new technique of transferring a branch of the radial nerve that normally innervates the long head of the triceps into the anterior branch of the axillary nerve.[53,94] The cadaveric study demonstrated that the nerve to the long head of the triceps muscle could be reliably identified and transferred with minimal morbidity. Histologic analysis also showed that the diameter and the number of axons of the donor nerve were roughly equivalent to the recipient nerve.[94] Although long-term follow-up data from the clinical case series are still lacking, early results of this technique have demonstrated promising outcomes.[53] Thus, neurotization may provide an acceptable alternative to nerve grafting in some patients with isolated axillary nerve injuries.

Operative Treatment: Muscle Transfers

Patients with chronic axillary nerve injuries may demonstrate significant muscular changes such as atrophy and fatty degeneration. In these patients, procedures that repair or restore the nerve function will be of limited benefit. In these select cases, a muscle transfer procedure can be considered. The type of muscle transfer is dependent on the nature of the functional deficit. If, for example, only a portion of the deltoid activity is lost, the remaining portion of the muscle can be transferred to restore the lost function.

If the entire deltoid muscle is denervated, the trapezius can be transferred distally onto the proximal humerus. Originally described by Bateman[7] and then subsequently modified by Saha,[79] this technique involves a saber incision along the scapular spine. The incision is curved anteriorly along the acromion to expose the entire trapezius insertion including the distal clavicle. Bony cuts in the acromion and the clavicle are then made to release the entire muscle insertion. After mobilizing the trapezius muscle, its bony insertions are transferred onto the proximal humerus and fixed with screws. In this fashion, the insertion of the trapezius muscle is transferred distally to augment shoulder abduction and flexion. By transferring the insertion on the anterior or posterior aspect of the humerus, shoulder rotation may also be restored.[79]

Another structure that has been used as a donor for muscle transfer procedure is the latissimus dorsi.[46,63,85] Advocates of this procedure argue that the muscle provides a more physiologic lever arm for flexion, restores normal shoulder contour, and does not disrupt the coracoacromial arch.[46] The original technique described by Itoh and colleagues involves three separate incisions: (1) laterally on the anterior border of the latissimus dorsi to isolate and harvest the muscle; (2) anterolaterally on midhumerus to expose the deltoid insertion site; and (3) superiorly along the acromion and the distal clavicle to isolate the deltoid origin. Alternatively, in addition to the posterior incision for the latissimus dorsi muscle, a single extended incision can be made in the anterior aspect of the shoulder to expose the deltoid in its entirety (Fig. 36-10). Patients are placed in the lateral decubitus position with the entire arm draped free. First, the latissimus dorsi muscle is isolated in its entirety. While preserving its neurovascular pedicle, the muscle is harvested by releasing its origin and insertion. Next, a bed for the muscle transfer is created by exposing the native deltoid muscle. Tunnels are then created deep and superior to the pectoralis major tendon. Through this tunnel, the latissimus dorsi muscle is transferred to the anterior aspect of the shoulder without disturbing its neurovascular pedicle. The muscle is rotated such that its broad origin lies superiorly along the acromion and the distal clavicle. Its tendinous insertion is then laid on top of the deltoid insertion on the midhumerus. The muscle is tensioned with the arm held in 70 to 80 degrees of flexion and then attached to the underlying deltoid muscle or to the bone using bone tunnels. Although this procedure has not been commonly performed, early reports and anecdotal experience demonstrate that some selective patients can recover near-normal function of their shoulders.[46,63]

Postoperative Considerations

During the immediate postoperative period, the type and duration of immobilization can vary widely depending on the performed procedure. As such, recommendations for postoperative immobilization have varied from a simple sling[85] to a Desault-type cast.[68] Recommendations for the duration of immobilization has similarly varied from 1 week to 2 months.[66,68] In general, more reconstructive procedures will require a prolonged period of immobilization to allow healing. Thus, for example, muscle transfer procedures typically require a more rigid immobilization (splint or spica cast) for longer duration in a position that minimizes tension across the fixation sites.

After the initial period of immobilization, passive exercises are initiated to restore shoulder motion. This also minimizes the formation of scar tissue that can encapsulate the decompressed or grafted nerve. Subsequently, active-motion exercises are gradually instituted. Although supervised physical therapy can be very useful, it is crucial to stress to the patients that they are responsible for their own rehabilitation and to perform some of these exercises on a daily basis as a home regimen. Depending on the type of the lesion and the surgical procedure, sensation may gradually return over 3 to 6 months. Strength may be regained in a similar period, but occasionally may take 12 months or longer until maximal strength is restored.[3,14,17,18,31,46,63,66,68]

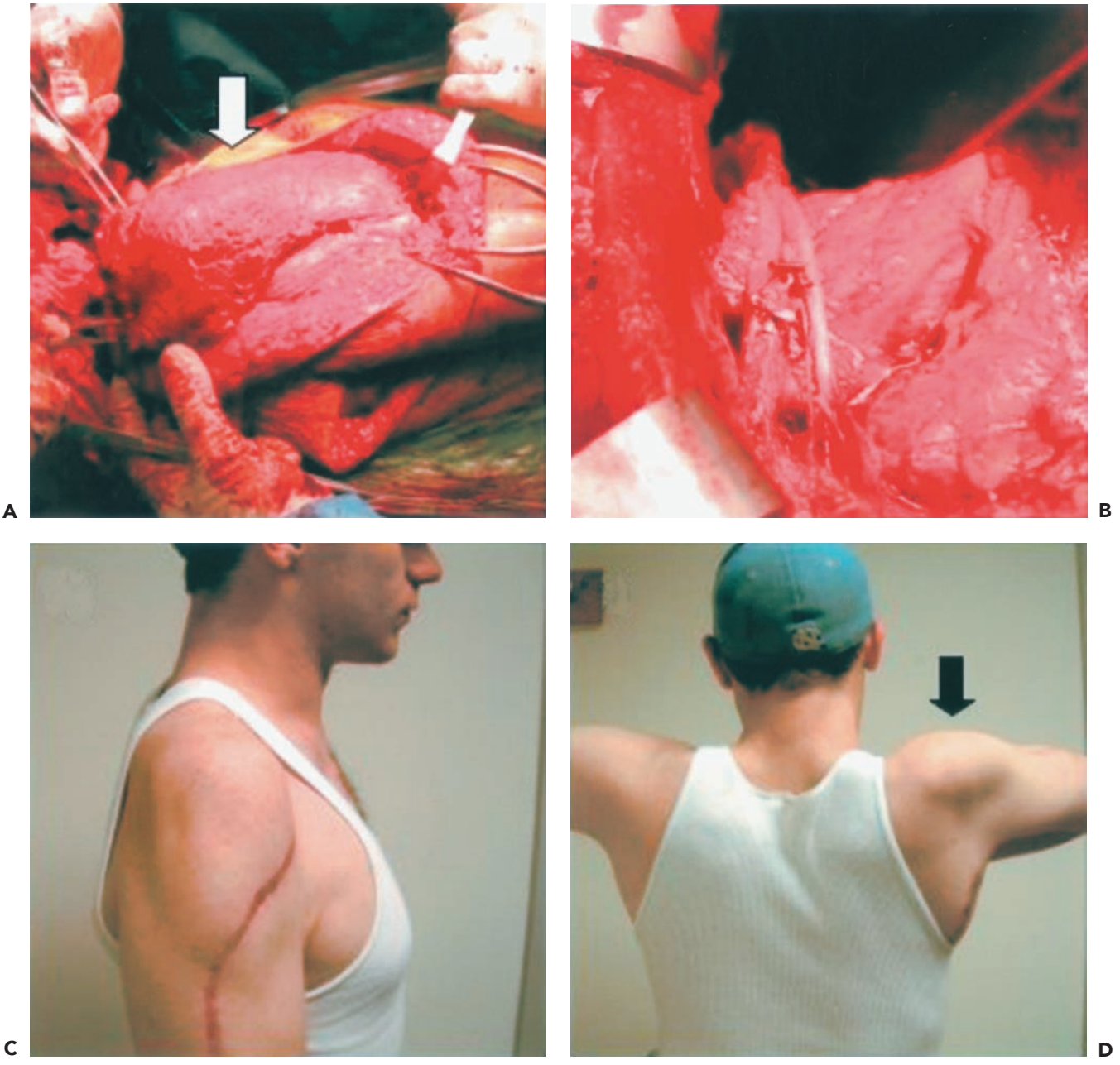
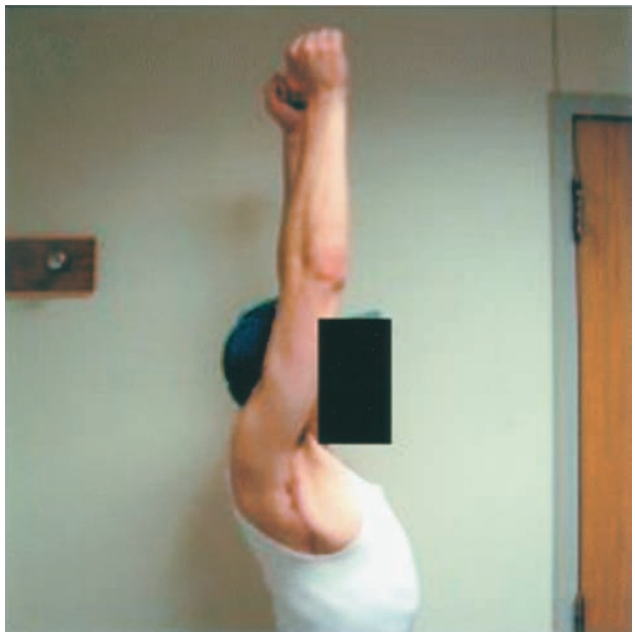


Figure 36-10 Latissimus dorsi muscle transfer for a 21-year-old male with a permanent axillary nerve injury demonstrated in figure 36–8. After isolating the latissimus dorsi muscle (*arrow*), it is released from its insertion and origin. The muscle is then transferred and laid directly over the atrophied deltoid muscle **(A).** During the transfer, the neurovascular pedicle to the muscle is isolated and protected during the procedure **(B).** Six months after the procedure, the patient exhibits restoration of normal shoulder contour **(C),** abduction **(D),** elevation **(E),** and external rotation **(F).**

Results and Complications

For surgical procedures that directly address the axillary nerve, the best outcomes have been reported when they are performed during the early period. Therefore, most authors suggest that decompression, neurolysis, nerve grafting, and neurotization all possess the highest rates of success if they are performed within the first 6 months of the injury. However, there have also been some reports of good functional improvement in patients with intervention within 1 year of injury. In general, surgery delayed beyond 1 year is believed to be associated with suboptimal outcomes.[18,59,68]

The nerve decompression procedure for QSS is generally associated with a good outcome. Francel et al., for example, reported that all five patients in their series



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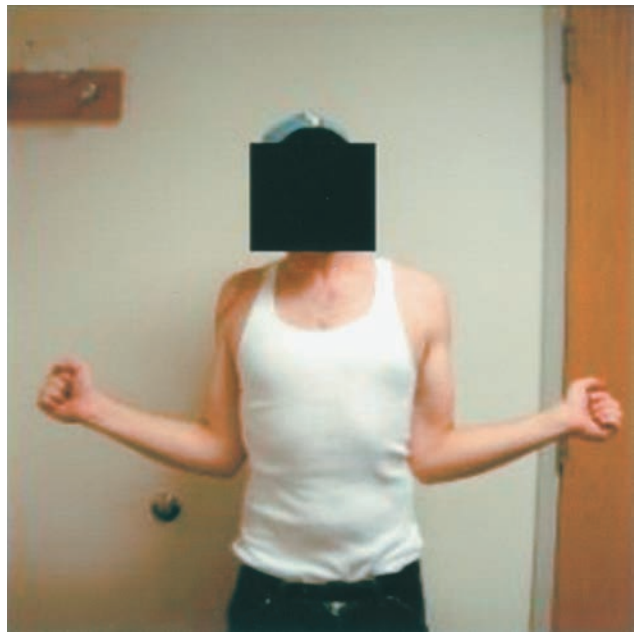

F

Figure 36-10 *(continued)*

experienced improved motion, pain relief, and sensation.[31] In a larger series of patients, Cahill and Palmer noted that only 2 of the 18 patients demonstrated no improvement after the procedure. Of the remaining 16 patients, half of them were completely asymptomatic, while the other half experienced occasional night pain only.[14] Despite these generally good outcomes, as the pathophysiology of this diagnosis is not well understood, some authors still recommend that the procedure be reserved for patients whose symptoms are refractory to nonoperative treatment.[66]

Similar to nerve decompression, neurolysis and nerve grafting procedures are also associated with generally favorable outcomes. Coene and Narakas reported on 27 patients who underwent exploration, neurolysis, and, if necessary, nerve grafting within 6 months of the injury. They found that nine patients recovered full strength while another nine patients recovered to one grade less than full strength. Those who only underwent a neurolysis procedure fared slightly better as 10 of these 13 patients were able to regain either full strength or one grade less than full strength at follow-up. Delaying surgery 1 year or more after the injury resulted in inferior outcomes. Five of these six patients experienced significant residual weakness and less functional recovery than their counterparts who underwent early surgical intervention.[18] In another series of 12 patients who were treated with nerve grafting or neurolysis, Artico et al. reported 50% good and 50% excellent results. They found no difference in the final outcome between the two procedures.[3] Petrucci et al. reported similar success with treating patients with sural nerve grafts, as they noted that eight of the nine patients were able to regain either full strength or one grade less than full strength at a minimum follow-up of 1 year.[68]

Leechavengvongs et al. reported on a series of seven patients who underwent neurotization of the anterior deltoid with the nerve to the long head of the triceps. All patients achieved significant functional recovery with a mean abduction of 124 degrees and all attaining four out of five motor strength. Additionally, there were no subjective complaints regarding any functional deficit from the donor site.[53] Despite these early promising results, however, more long-term outcome data are needed to establish the efficacy of this procedure.

Muscle transfer procedures are typically used to treat patients whose axillary nerve lesion has resulted in a significant and permanent deterioration of the deltoid muscle. As such, it may be expected that these patients experience outcomes that are generally inferior in comparison to those whose nerve lesions have been directly addressed. Itoh et al. reported on 10 cases of latissimus dorsi muscle transfer for deltoid paralysis and found that only six were able to obtain active flexion greater than 90 degrees. Upon further analysis, they noted that the final outcome corresponded with the preoperative function of the rotator cuff muscles. As such, they suggested that concomitant rotator cuff pathology is a relative contraindication for the latissimus dorsi transfer procedure.[46] Subsequently, Narakas modified this procedure by transferring both the latissimus dorsi and the teres major muscle. The latissimus dorsi was still transferred to the anterior deltoid while the teres major was simultaneously transferred onto the infraspinatus

tendon. He noted that the teres major muscle transfer portion of the procedure provided better visualization and mobilization of the latissimus dorsi neurovascular pedicle. Of the six patients who underwent this procedure, five exhibited good to excellent active abduction.[63]

SUMMARY

Injuries to the suprascapular and axillary nerves can have profound effects on shoulder function and cause significant pain and discomfort. Once diagnosed, the prognosis appears to be dependent on the cause and severity of the lesion. Although a trial of nonoperative management is generally recommended for most patients, surgical intervention may be required for a select population of patients. Depending on the cause of the lesion, several procedures exist to correct the pathology and restore nerve function. With appropriate counseling and treatment, a favorable outcome can be achieved for most patients.

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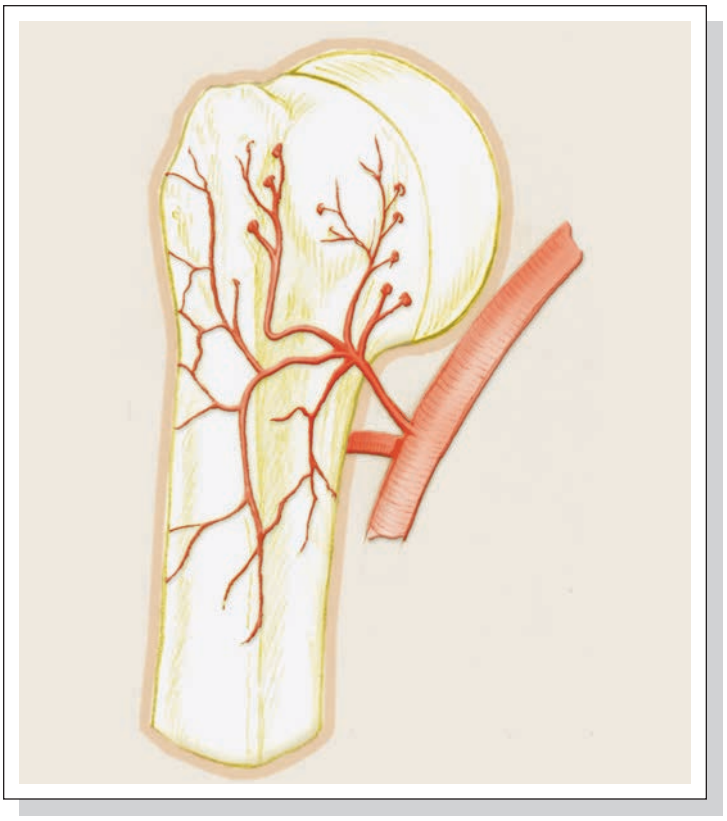
Neoplasia and Infection

VIII



Tumors of the Shoulder Girdle

37

Martin M. Malawer *James C. Wittig* *Kristen Kellar Graney*

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INTRODUCTION

Each bone of the shoulder girdle—the proximal humerus, the scapula, and the clavicle—can give rise to a primary bone tumor or be involved by an adjacent soft tissue sarcoma.[1] The proximal (upper) humerus is one of the most common sites for high-grade malignant bony tumors in both adults and children, and it is the third most common site for osteosarcomas. Chondrosarcomas also commonly involve the shoulder girdle, often arising from the scapula or the proximal humerus. The bones of the shoulder girdle may also be involved secondarily by high-grade soft tissue sarcomas or metastatic tumors that often require resections similar to those used in the treatment of high-grade primary bony sarcomas. Metastatic tumors often involve the shoulder girdle, and because of the extent of bony destruction and the presence of large extraosseous components, the treatment is sometimes similar to that for primary malignant bony sarcomas. For example, hypernephromas (renal cell carcinomas) have a unique propensity to involve the proximal humerus, often as a solitary metastasis. They commonly result in extensive bony destruction with a large soft tissue component.

Three Phases of Surgical Resection

The surgical treatment of a malignant bony tumor involving the shoulder girdle consists of three stages: (a) wide surgical resection of the tumor, (b) reconstruction of the skeletal defect, and (c) multiple muscle transfers to provide soft tissue coverage, stabilize the shoulder girdle, and restore function to the upper extremity.

The aim is to provide a stable shoulder and preserve a functional elbow and hand. Each of the various surgical techniques currently in use for reconstruction of a segmental defect of the humerus or shoulder girdle offer some degree of stability, function, durability, range of motion, and preservation of motor power.

CLASSIFICATION OF SHOULDER-GIRDLE RESECTIONS

Malawer et al. have developed a six-stage surgical classification system.[3] This system is based on current concepts of surgical margins, the relationship of the tumor to anatomic compartments (i.e., intracompartmental vs. extracompartmental), the status of the glenohumeral joint (intraarticular vs. extraarticular), the magnitude of the individual surgical procedures, and the presence or absence of the abductor mechanism (deltoid muscle, rotator cuff muscle, or both).

The six-stage classification is as follows (Fig. 37-1):

Type I: Intraarticular proximal humeral resection
Type II: Partial scapular resection
Type III: Intraarticular total scapulectomy
Type IV: Intraarticular total scapulectomy and humeral head resection
Type V: Extraarticular humeral and glenoid resection
Type VI: Extraarticular humeral and total scapular resection

Each of the six types is further modified according to a major variable: the presence or absence of the main motor group, the abductor mechanism (i.e., deltoid and rotator cuff muscles). The abductors are either present (subtype A) or partially or completely resected (subtype B). The abductor mechanism is almost always resected when there is extraosseous extension of a bone tumor in this area. The loss of any component of the abductor mechanism tends to create a similar functional disability. Regardless of histology or primary bone involvement, subtype A generally entails an intracompartmental resection, and subtype B an extracompartmental resection (Table 37-1).

TUMOR GROWTH AND ANATOMY

Sarcomas, which arise from mesenchymal tissues (mesodermal embryonic layer), grow in a centripetal manner and form ball-like masses and compress surrounding muscle into a pseudocapsule layer. Sarcomas typically respect fascial borders and generally grow along the path of least resistance. This growth pattern is in contrast to that of carcinomas, which are invasive and usually penetrate compartmental borders. The pseudocapsule layer contains microscopic, finger-like projections of tumor referred to as satellite nodules. Sarcomas spread locally along the path of least resistance. Surrounding fascial layers resist tumor penetration and provide boundaries to local sarcoma growth. These boundaries

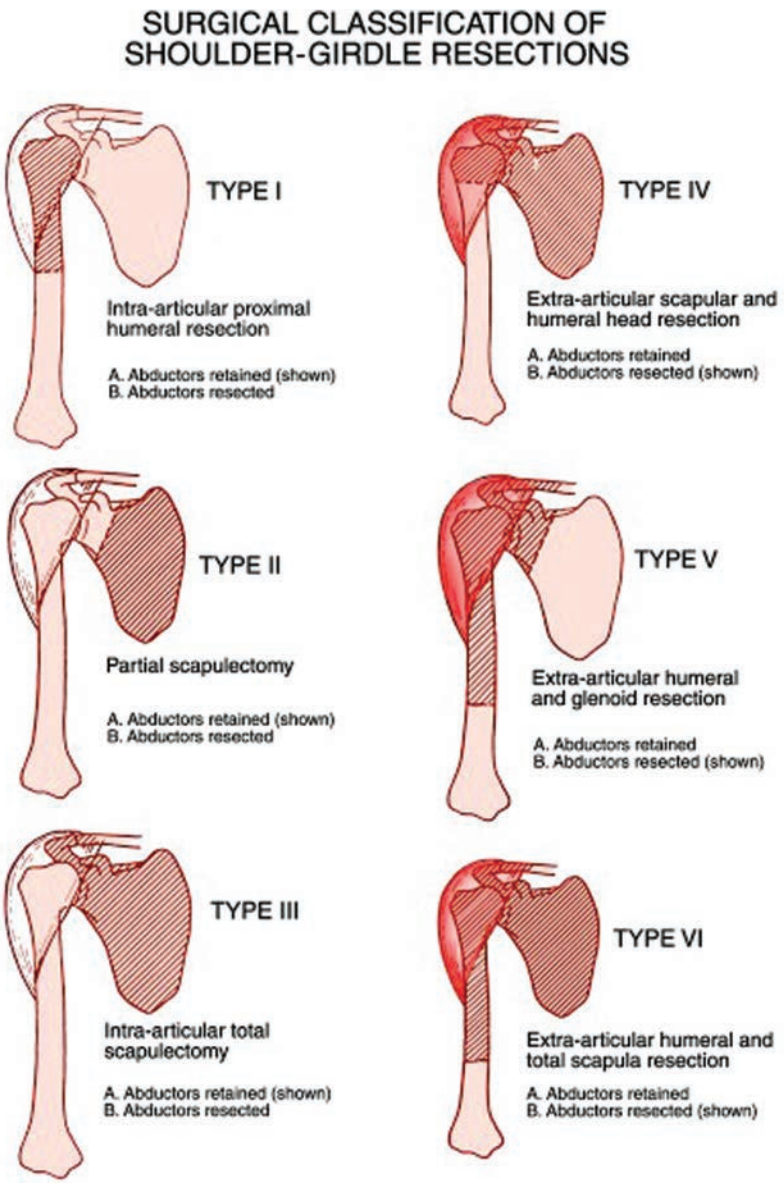


Figure 37-1 Surgical classification of shoulder girdle resections. Shoulder girdle resections are classified as type I to type VI. In general, types I to III are performed for benign or low-grade tumors of the shoulder girdle, whereas types IV to VI are performed for high-grade malignant tumors. In the schematic key, A = abductor muscles retained, whereas B = abductor muscles resected. The main abductors of the shoulder girdle include the rotator cuff musculature and the deltoid muscle. In general, these muscles are resected with high-grade tumors but are retained with low-grade tumors. (From Malawer MM, Meller I, Dunham WK. A new surgical classification system for shoulder-girdle resections. Analysis of 38 patients. *Clin Orthop* 1991;(267):33–44.)

form a compartment around the tumor. A sarcoma will grow to fill the compartment in which it arises; only rarely does a sarcoma extend beyond its compartmental boundaries. With reference to bony sarcomas that extend beyond the cortices into the surrounding soft tissues, the term "functional anatomic compartment" refers to the investing muscles that are compressed into a pseudocapsular layer. These muscles provide the fascial borders of the compartment, which has important surgical implications. A wide resection (i.e., compartmental resection) of a bone sarcoma entails removal of the entire tumor and pseudocapsular layer and must therefore encompass the investing normal muscle layers.

The functional compartment surrounding the proximal humerus consists of the deltoid, subscapularis, and remaining rotator cuff musculature, latissimus dorsi, brachialis, and portions of the triceps.

High-grade sarcomas that extend beyond the bony cortices of the proximal humerus involve and compress the investing muscles that form the compartmental borders and pseudocapsular layer. They grow along the path of least resistance and therefore are directed toward the glenoid and scapular neck by the rotator cuff and the glenohumeral joint capsule. Anteriorly, the tumor is covered by the subscapularis, which bulges into and displaces the neurovascular bundle (axillary vessels and brachial plexus). Only rarely does a proximal humerus sarcoma extend beyond the compartmental borders. In these instances, the tumor usually protrudes through the rotator interval. A wide resection for a high-grade sarcoma must therefore include the surrounding muscles that form the pseudocapsular layer, the axillary nerve, the humeral circumflex vessels, and the glenoid (extraarticular resection).

TABLE 37-1
TYPE OF RESECTION, TYPE OF RECONSTRUCTION, AND FUNCTIONAL OUTCOMES OF 134 TUMORS TREATED BY A LIMB-SPARING RESECTION OF THE SHOULDER GIRDLE

Resection Type	*n*	Proximal Humerus Prosthesis	Scapular Prosthesis	Humeral Head Suspension	Function			
					Excellent	Good	Moderate	Poor
IA	29	29			20	5	4	
IB	7	7			3	3	1	
IIA	5				4	1		
IIB	12				8	4		
IIIA	1			1		1		
IIIB	15		3	12	6	3	4	2
IVA	0							
IVB	8	4	4		4	2	1	1
VA	1					1		
VB	53	53			4	31	11	7
VIA	0							
VIB	3		2			1	2	
Total	134	92	9	13	49	52	23	10

From Bickels J, Wittig JC, Kollender Y, Kellar-Graney K, Meller I, Malawer MM, Limb-sparing resections of the shoulder girdle. *J Am Coll Surg* 2002;194(4):422–435.

Most high-grade scapular sarcomas arise from the region of the scapular neck and body. The compartment consists of all of the muscles that originate on the anterior and posterior surfaces of the scapula. Although not one of the compartmental borders, the deltoid, which attaches to a narrow region of the scapular spine and acromion, may be involved secondarily by a large soft tissue extension. In most cases, the deltoid is protected by the rotator cuff muscles. Because the anatomic origin of most tumors is in the neck, the rotator cuff muscles are compressed into a pseudocapsular layer by sarcomas that arise from the scapula. The subscapularis also protects the neurovascular bundle from tumor involvement. The head of the proximal humerus is contained within the compartment surrounding the scapula. The tumor follows the path of least resistance and typically crosses the glenohumeral joint, grossly or microscopically, to involve the humeral head. Direct tumor extension through joints or articular cartilage is rare and typically occurs as the result of a pathologic fracture. Because of the small size of the glenohumeral joint, the tumor almost always involves the capsule or the synovium. The long head of the biceps tendon, which is intraarticular, is another pathway by which the tumor may cross the joint. Wide resection of a high-grade scapula sarcoma must therefore include the rotator cuff and, in most instances, the humeral head.

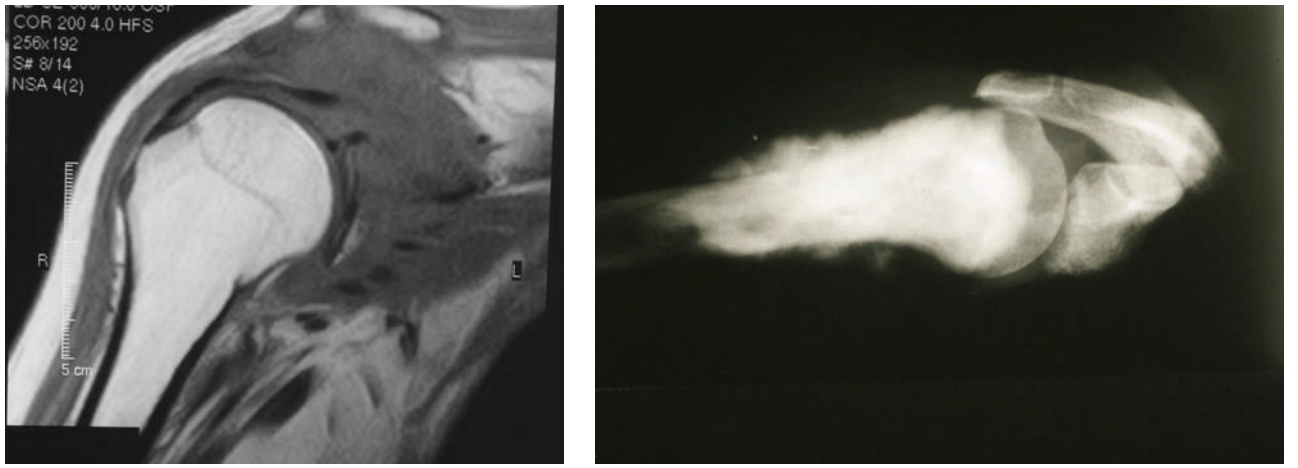
INTRA- VERSUS EXTRAARTICULAR TUMOR EXTENSION

The shoulder joint appears to be more prone than other joints to intraarticular or pericapsular involvement by high-grade bone sarcomas. Figs. 37-2 and 37-3 show the mechanisms of tumor spread. Direct capsular extension, direct tumor tracking along the long head of the biceps, a poorly planned biopsy, and pathologic fracture are mechanisms of glenohumeral contamination and make intraarticular resection for high-grade sarcomas a higher risk than extraarticular resection for local recurrence. A local recurrence in this region often requires a forequarter amputation and may compromise patient survival. (This is in contrast to most clinical experience with resections of the distal femur, which tend to be intraarticular.) Therefore, extraarticular resection is recommended for most high-grade sarcomas of the proximal humerus and scapula.

CLINICAL EVALUATION

History and Physical Examination

Patients with bone sarcomas typically present to their primary care physician with complaints of a dull, aching pain of several months' duration. They often seek medical intervention because the pain has become more severe. This increased pain can be correlated with tumor penetration of cortical bone, irritation of the periosteum, or pathologic fracture. Severe night pain in the affected extremity is common. Some patients may describe regional tenderness, difficulty in moving the arm, or a palpable swelling or mass. Physical examination of the extremity usually confirms the presence of a mass or regional swelling and deformity. Children are especially susceptible to referred pain; for this reason, all regional joints should be examined.



A B

Figure 37-2 **(A)** Magnetic resonance imaging (MRI) scan showing gross tumor involvement within the joint (T1-weighted MRI image). **(B)** Radiograph of an extraarticular resection of the proximal humerus for osteosarcoma. Note that the proximal humerus, glenoid, and distal one-third of the clavicle have been resected en bloc. This procedure is classified as a type VA resection (Malawer classification). Extraarticular resection is often required for high-grade sarcomas of the proximal humerus.

The presenting symptoms for a soft tissue sarcoma are different and nonspecific. Typically, the mass presents as a slow-growing, painless lesion. Tumors arising in the upper extremity are more palpable and identified earlier than those in the lower extremity.

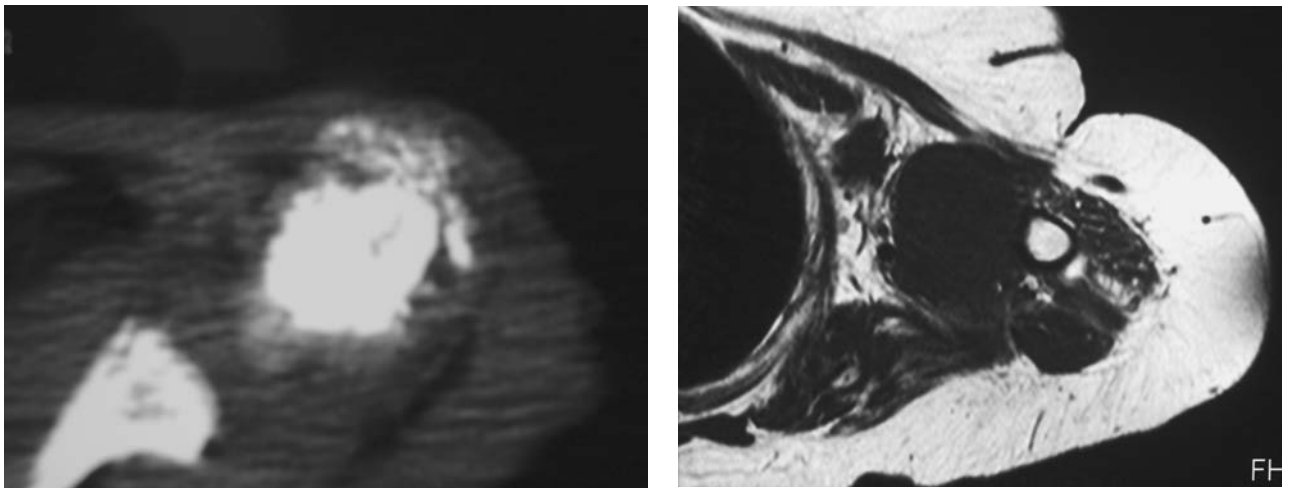
Specialist Referral

All patients with suspected malignancies should be referred to an orthopedic oncologist or a cancer center. A multidisciplinary team approach for patients with malignant tumors is essential to providing the best possible clinical outcome. Patients with aggressive benign tumors (i.e., giant cell tumor, chondroblastoma, or enchondroma) should also be referred.

Unique Anatomic and Surgical Considerations

The local anatomy of a sarcoma determines the extent of the operative procedure required. The following discussion addresses unique considerations of shoulder girdle anatomy that are relevant to surgery in this area.

- The glenohumeral joint generally does not serve as an effective barrier to tumor spread. A lesion may cross the joint by direct extension or indirect mechanisms, as



A B

Figure 37-3 **(A)** Computed tomography scan of the glenohumeral joint showing destruction of the glenohumeral joint by tumor. The typical mechanism of intraarticular involvement by tumor is via direct extension and by capsular involvement. **(B)** Magnetic resonance imaging (MRI) scan demonstrating large tumor component adjacent to the proximal humerus with an adjacent skip nodule or enlarged axillary lymph node. MRI scans of the shoulder girdle are essential in evaluation of neoplastic lesions.

shown (Figs. 37-2 and 37-3). It is often necessary to perform an extraarticular resection for high-grade bone sarcomas of the proximal humerus or the scapula (glenoid region).

- The three major cords of the brachial plexus are in close proximity to the subscapularis muscle, glenohumeral joint, and proximal humerus. Tumors involving the upper scapula, the clavicle, and the proximal humerus often displace the infraclavicular component of the brachial plexus. It may be necessary to sacrifice some of the major nerves if they are encased by neoplasm, or a forequarter amputation may be required.
- The musculocutaneous and axillary nerves are often in contact with or in close apposition to tumors around the proximal humerus, and before proceeding with resection it is necessary to clearly identify both. It is crucial to preserve the musculocutaneous nerve to preserve a functional elbow. The musculocutaneous nerve generally comes from beneath the coracoid and passes through the conjoined tendon or coracobrachialis muscle within a few centimeters of its origin. The position of this nerve does vary, however, and it may lie within 2 to 8 cm of the coracoid. It then passes through the short head of the biceps and into the long head of the biceps before innervating the brachialis muscle.
- The axillary nerve is closest to most large tumors of the proximal humerus. It arises from the posterior cord and, along with the circumflex vessels, courses around the subscapularis muscle and the head and neck of the humerus to innervate the deltoid posteriorly. In patients who have large malignant tumors of the proximal humerus, the axillary nerve usually must be resected because of tumor proximity or involvement, and because it is necessary to remove the deltoid muscle and glenoid to provide a satisfactory margin. With large stage IIB bone sarcomas of the proximal humerus, the axillary nerve and deltoid muscles can rarely be preserved. In contrast, the axillary nerve is usually not involved by scapular tumors and therefore can be preserved along with the deltoid muscle. This allows for functional anatomic reconstruction of the scapula with a prosthetic replacement.
- The brachial artery is surrounded by the three major cords of the brachial plexus and is tethered to the proximal humerus by the anterior and posterior circumflex vessels. A presurgical angiogram is extremely useful to localize the brachial artery and identify the level of the circumflex vessels. Occasionally, one finds anomalous brachial and axillary arteries that would be difficult to identify and explore if not recognized preoperatively. In general, the circumflex vessels are ligated during the initial dissection; this allows the entire axillary artery and the vein and brachial plexus to fall away from the tumor mass. Early ligation of the circumflex vessels is key to the resection of proximal humeral sarcomas.
- The radial nerve courses along the posterior aspect of the axillary sheath and exits from the posterior cord at the inferior border of the latissimus dorsi muscle. Fortunately, most sarcomas are located in the proximal third of the humerus and do not involve this nerve. However, to avoid injury the radial nerve must be isolated and protected prior to tumor resection. Sacrifice of the radial nerve is rarely necessary.

PREOPERATIVE EVALUATION AND IMAGING STUDIES

Appropriate imaging studies are crucial to successful resection of tumors of the shoulder girdle (Fig. 37-4). The most useful preoperative evaluations are computed tomography (CT), magnetic resonance imaging (MRI), arteriography, and three-phase bone scans. For large tumors of the proximal humerus, a venogram may be warranted if there is clinical evidence of distal obstruction.

Computed Tomography

CT is more useful than MRI in determining cortical bone changes, and it is considered complementary to MRI in evaluating the chest wall, clavicle, and axilla. CT is useful for determining the planes of tumor resection. Subtle cortical erosions by adjacent soft tissue sarcomas are better visualized on CT than on MRI. The amount of tumor necrosis can be determined. Often, a reactive rim of calcification can be visualized surrounding tumors that have had a good response to preoperative chemotherapy.

Magnetic Resonance Imaging

MRI is used to determine the extent of soft tissue involvement, especially around the glenohumeral joint, along the chest wall, and into the axillary space. It is often difficult to visualize the suprascapular area in patients with large tumors, which may infiltrate below the subscapularis muscle and exit near the coracoid. MRI is especially useful in identifying the extent of intraosseous tumor, which is necessary to determine the length of the resection. Skip metastases can also be identified, although they rarely occur in this area. MRI is not useful for determining the preoperative tumor response to induction chemotherapy. MRI and bone scan studies accurately demonstrate the soft tissue extension as well as the intraosseous extent of the tumor.

Bone Scan

Bone scintigraphy is routinely used to assess the presence of metastatic and polyostotic bone disease as well as involvement of a bone by adjacent soft tissue sarcomas.

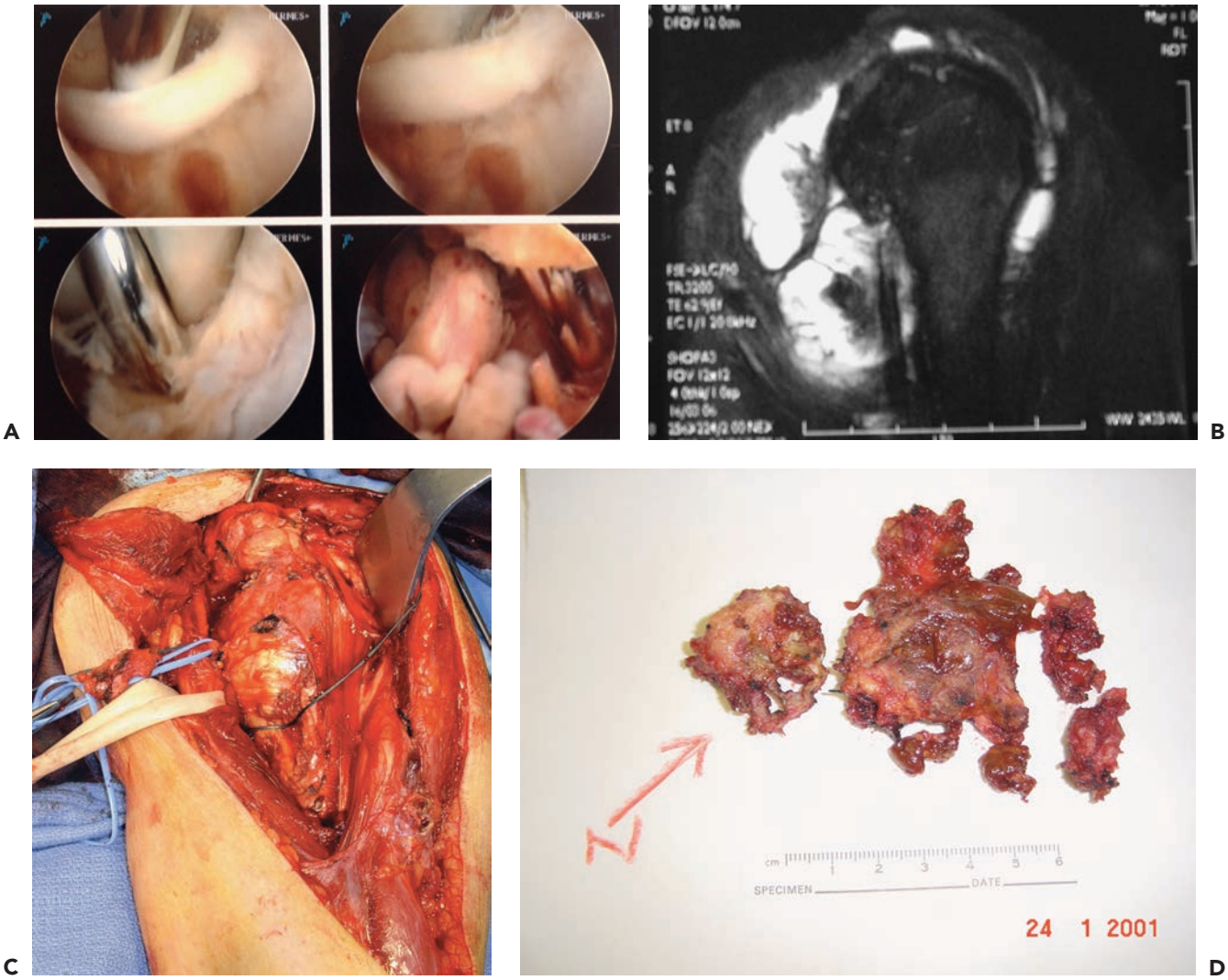


Figure 37-4 Pigmented villonodular synovitis (PVNS) or aggressive synovitis of the shoulder. **(A)** This photograph shows an arthroscopic view of the shoulder joint. Note the brownish pigmentation of the synovium. **(B)** Magnetic resonance imaging scan (T2-weighted fat suppressed) showing a marked distended joint with blood (white area) and bulky synovial disease. **(C)** Intraoperative photograph showing the proximal humerus following detachment of the pectoralis major muscle. Note the large soft tissue mass surrounding the proximal humerus. **(D)** Gross specimen following curettage and resection of tumor (PVNS). Note the brownish pigmentation, which is consistent with hemosiderin deposits and marked histiocytic proliferation.

The appearance of a bone lesion in the flow and pool phases, when assessed by a three-phase bone scan, is useful in determining the biologic activity of the tumor, which may be helpful in determining a diagnosis. Some surgeons utilize the bone scan following induction chemotherapy as an indirect measurement of evaluating tumor response.

Angiography

Angiography is extremely useful and should be done with the arm abducted to determine the relationship of the axillary and brachial vessels to the major tumor, the level of the circumflex vessels, and the presence of any anomalies (Fig. 37-5). The axillary vessels and brachial plexus are often displaced by large tumors in this area. Angiography is also the most reliable means of determining the response to neoadjuvant chemotherapy. The absence of vessels in the tumor or a decrease in tumor vascularity indicates tumor necrosis. If there is a very good angiographic response (i.e., decrease in or absence of tumor blush), it is indicative of a good response to the preoperative chemotherapy. This information is important for determining the extent of surgical margins and also provides prognostic information. For instance, if the tumor has had a good response, it is safe to proceed with

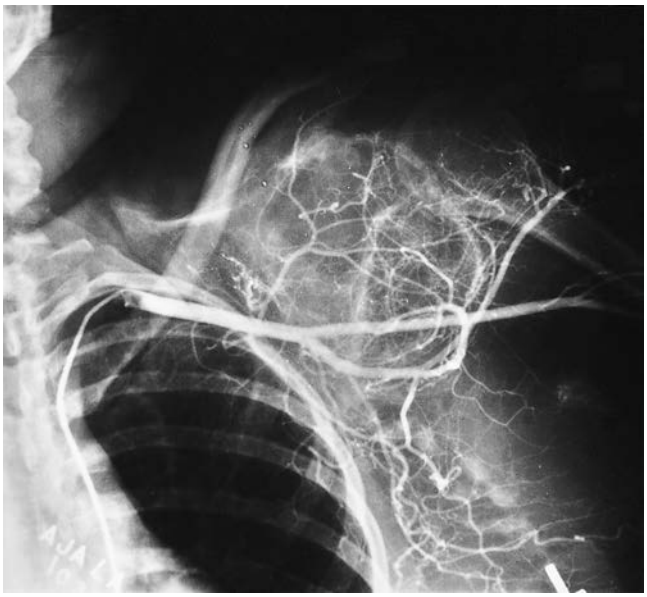


Figure 37-5 Angiogram of a large scapular sarcoma. Angiograms are essential for evaluation of bulky tumors of the shoulder girdle. This scan demonstrates the relationship of the artery to the tumor and the marked vascularity of most tumors. In general, prior to surgery, vascular tumors are embolized.

a limb-sparing resection. The margins may be narrower with less soft tissue resected. If the tumor has had a poor response, the surgeon may elect to take a wider soft tissue margin or perform an amputation. The venous flow phase is useful to demonstrate venous occlusion or tumor thrombi. If there is any suggestion of occlusion, a brachial venogram should be performed (Fig. 37-6A).

Venography

If venous thrombosis or a mural thrombosis is expected, venography should be performed. The most suspicious finding is extremity edema. Axillary vein thrombosis or occlusion is most common with large shoulder osteosarcomas and chondrosarcomas. It is indicative of encasement of the vascular structures by neoplasm and therefore indirectly reflects brachial plexus involvement because of the intimacy of the brachial plexus and the axillary vessels. This finding suggests that the tumor is unresectable (Fig. 37-6B).

Biopsy

Because 95% of bone sarcomas have a soft tissue component, a small needle, or core, biopsy is possible (Figs. 37-7 and 37-8). One exception may be the young patient with a suspected round cell tumor from whom more tissue may be required for cytogenetic and immunohistochemical stains. Another exception would be an older patient in whom a solitary metastatic lesion is suspected and the pathology supports either metastatic carcinoma or a spindle cell sarcoma. This differentiation most often occurs with metastatic renal cell carcinoma. In such a case, a significant amount of tissue may be required to obtain

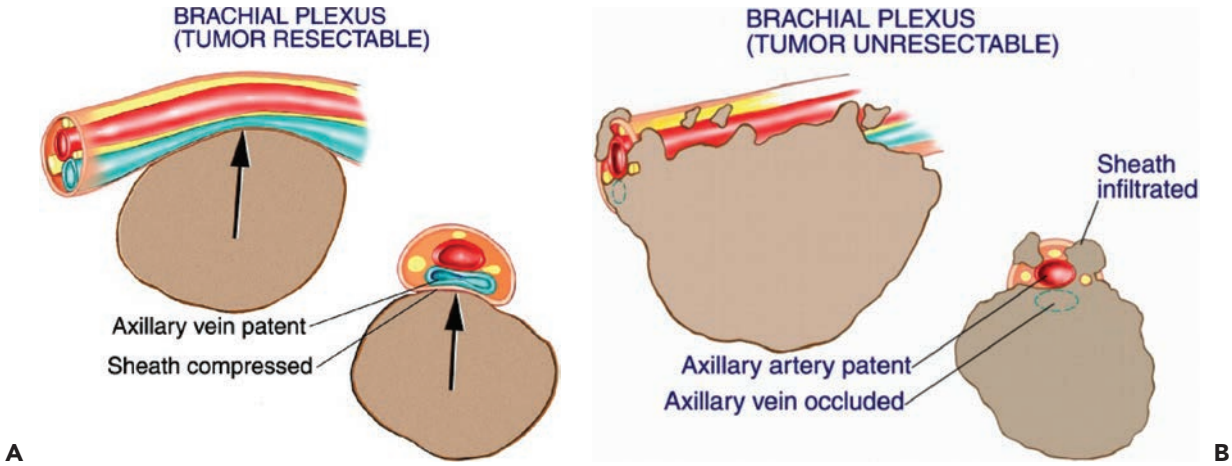


Figure 37-6 **(A)** Schematic diagram showing the relationship of the axillary vessels to tumors arising within the axillary space, either from the scapula, proximal humerus, or axillary space itself. The infraclavicular portion of the brachial plexus is often displaced by a tumor mass. The axillary artery and vein can be seen on angiography and axillary venography, respectively. These vessels are both patent but may be compressed. The clinical implication is that there is no nerve involvement and therefore these tumors are usually resectable. **(B)** Schematic diagram showing an unresectable tumor due to infiltration of the infraclavicular portion of the brachial plexus. An angiogram would still demonstrate a patent artery in this situation, but most importantly, the axillary vein would be occluded and thus the surrounding nerves are infiltrated. Axillary venography has proven to be very important in determining tumor respectability.(From Malawer M, Wittig JC. Resections of the shoulder girdle. In: Malawer MM, Sugarbaker PH, eds. *Musculoskeletal cancer surgery: treatment of sarcomas and allied diseases.* Dordrecht, Netherlands: Kluwer Academic Publishers, 2001:193.)

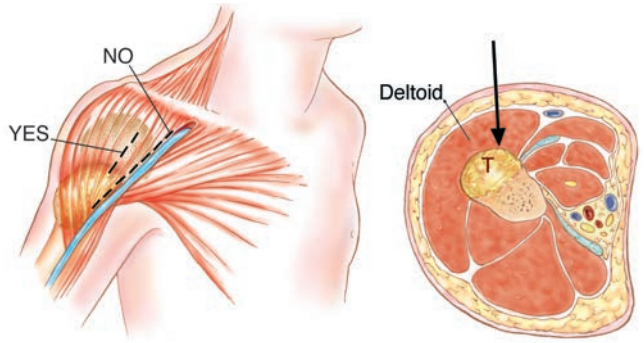


Figure 37-7 Schematic of the shoulder girdle and the preferred site of a needle or a small incisional biopsy for bony tumors. Biopsy should be performed through the anterior one-third of the deltoid to avoid contamination of the pectoralis major and therefore the brachial vessels underneath. A biopsy should never be performed through the deltopectoral interval. Approximately 95% of bony tumors can be correctly identified with multiple cores obtained through a single puncture site performed under computed tomography guidance. (From Malawer M, Wittig JC. Resections of the shoulder girdle. In: Malawer MM, Sugarbaker PH, eds. *Musculoskeletal cancer surgery: treatment of sarcomas and allied diseases.* Dordrecht, Netherlands: Kluwer Academic Publishers, 2001:194.)

immunohistochemical stains that will differentiate the metastatic tumor from a primary sarcoma.

Planning the Biopsy

It is essential to plan and perform the biopsy carefully, because an inappropriate biopsy is a common cause of forequarter amputation. In a patient with a tumor of the proximal humerus, a core biopsy through the anterior third of the deltoid muscle is recommended (see Fig. 37-8). Open biopsies are rarely required and may lead to excessive local contamination. If there is a soft tissue component, which occurs 95% of the time, there is no need to enter the bone. Several samples may be taken from different areas through a single puncture site. Care must be taken to avoid the deltopectoral groove. Contamination of this groove leads to contamination of the pectoralis muscle and, potentially, of the brachial vessels and axillary space (see Fig. 37-7).

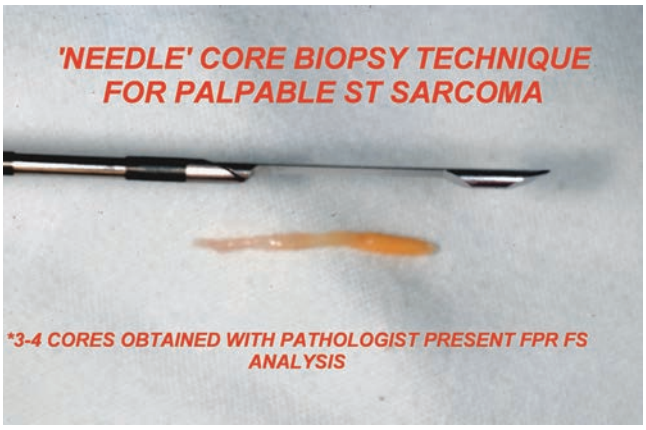


Figure 37-8 Core biopsy showing the needle and specimens that can be obtained. Approximately four or five cores are obtained from the tumor through the same puncture wound by changing the angle of the approach.

Biopsy Technique

The biopsy site is a crucial factor in determining the final operative procedure. For tumors arising within the body of the scapula, a posterior needle biopsy or a biopsy along the axillary border of the scapula is recommended. With lesions involving the scapula and neck, a posterior approach directly through the teres minor is recommended. If an open biopsy is required, a small longitudinal incision in line with the incision that will be used for resection is recommended. Most operative approaches involve an incision along the axillary border of the scapula.

A biopsy of the proximal humerus should be performed through the anterior third of the deltoid, not through the deltopectoral interval. A biopsy through the anterior third of the deltoid results in a limited hematoma that is confined by the deltoid muscle and can be resected with the tumor en bloc (see Fig. 37-7). The axillary nerve innervates the deltoid muscle posteriorly, so the anterior portion of the muscle can be partially resected with minimal loss of function if the remaining deltoid is to be preserved. On the other hand, an open biopsy through the deltopectoral interval will contaminate the pectoralis major muscle and provide a plane for the hematoma to dissect to the chest wall along the brachial vessels. This makes a local resection more difficult and increases the possibility of local recurrence.

Clavicle tumors are biopsied along the length of the clavicle. Unless there is a soft tissue component, a small biopsy is advisable because a needle in this location could injure the brachial plexus and the neurovascular bundle.

Most shoulder-girdle soft tissue sarcomas are easily palpable. Multiple core needle biopsies performed through one puncture site under local anesthesia are recommended. If the mass is not palpable, core biopsies should be performed under fluoroscopic or CT guidance. To obtain multiple specimens from different areas, the surgeon should reintroduce the needle through the same puncture site, varying the angle. Cultures should be obtained routinely, regardless of the suspected diagnosis, because infection may simulate any malignancy. Touch-preps, frozen sections, or both confirm that lesional tissue has been obtained.

INDICATIONS AND CONTRAINDICATIONS FOR SHOULDER-GIRDLE RESECTION

Approximately 95% of high-grade shoulder-girdle malignancies can now be treated safely by limb-sparing surgeries;

forequarter amputation is now rare. The decision to proceed with limb-sparing surgery is based on the location of the cancer and a thorough understanding of its natural history.

Major contraindications for limb-sparing techniques are tumor involvement of either the neurovascular bundle or the chest wall. Relative contraindications include:

- pathologic fracture,
- extensive involvement of the shaft of the humerus,
- infection, and
- tumor contamination of the operative area from hematoma following biopsy or unwise placement of the biopsy incision.

These contraindications are described later in greater detail. General comments on contraindications are as follows:

- A pathologic fracture that has healed during the course of induction chemotherapy is not a contraindication to limb-sparing surgery. The arm is immobilized during this treatment period. With proper surgical treatment, the local recurrence rate is acceptably low and survival rates are not altered.
- The brachial artery is rarely involved by a tumor, although it may be in close proximity to it. The subscapularis, coracobrachialis, and short head of the biceps muscles often separate tumors of the proximal humerus and scapula from the vascular structures and brachial plexus. Occasionally, however, the brachial veins are directly invaded by a tumor and may be the site of tumor thrombi.
- Involvement of the musculocutaneous nerve by the tumor is rare, as is involvement of the three major cords to the brachial plexus, which follow the brachial vessels. The axillary nerve is often involved by tumors arising from the proximal humerus and is therefore resected. Direct tumor extension of the brachial plexus requires a forequarter amputation. Such extension into the plexus occurs most often with axillary or chest wall sarcomas, or metastatic carcinomas or melanoma to axillary lymph nodes.

  If an inappropriate biopsy has contaminated the shoulder girdle, limb-preserving resection is often inadvisable. Today, one of the major causes for amputation of the shoulder girdle is inappropriate biopsy resulting in contamination of the pectoralis major, the chest wall, and the neurovascular structures.
- Infection is a contraindication to limb-preserving surgery. Even with adequate resection, reconstruction of an infected field by arthrodesis, prosthesis, or allograft replacement is extremely risky, considering that all patients with high-grade sarcomas must receive postoperative adjuvant chemotherapy. If an infection cannot be eradicated with the primary resection, amputation is advisable.
- Previous surgeries affect the feasibility of a limb-sparing procedure. The local recurrence rate is increased if a wide resection is attempted following a previous resection around the shoulder girdle.
- On rare occasions, tumors of the scapula or proximal humerus with large soft tissue components invade the chest wall and intermingle with the intercostal muscles and the ribs. This situation usually requires a resection of the adjacent chest wall, but it is not an absolute indication for forequarter amputation because limb-sparing resection may be combined with chest wall resection.
- In the rare instance of lymph node involvement documented by biopsy, a forequarter amputation may be the best way to remove all the axillary nodes as well as the proximal sarcoma. On the other hand, it is not unreasonable to proceed with a limb-sparing resection and an axillary node dissection. This method can provide long-term cure and local control.

Utilitarian Shoulder-Girdle Incision

The utilitarian shoulder-girdle incision was developed by the senior author (MM) to serve as a basic incision for use in all types of shoulder tumors and in all anatomic locations. This incision permits adequate exploration of the bony structures and soft tissues and complete exposure of the axillary vessels and infraclavicular brachial plexus. It consists of three components (Fig. 37-9):

1. Anteriorly, the incision begins at the junction of the medial and middle thirds of the clavicle. This incision extends medial to the coracoid, along the deltoid pectoral interval across the axillary fold, and courses distally along the anteromedial aspect of the arm.
2. The posterior incision begins over the midclavicular region of the anterior incision and travels inferiorly over the lateral aspect of the scapula and curves posteriorly at its tip. Large fasciocutaneous flaps are elevated anteriorly and posteriorly.
3. An incision into the axillary fold can be extended for proximal humerus tumors with axillary extension, for isolated axillary tumors, or for those rare instances when a limb-sparing resection cannot be performed and the procedure must be converted to a forequarter amputation.

PAIN CONTROL

A unique method developed for the postoperative management of pain in patients undergoing major tumor surgery is the use of perineural catheters. This technique involves the direct placement of a silastic (epidural type) catheter within the nerve sheath of the brachial plexus prior to

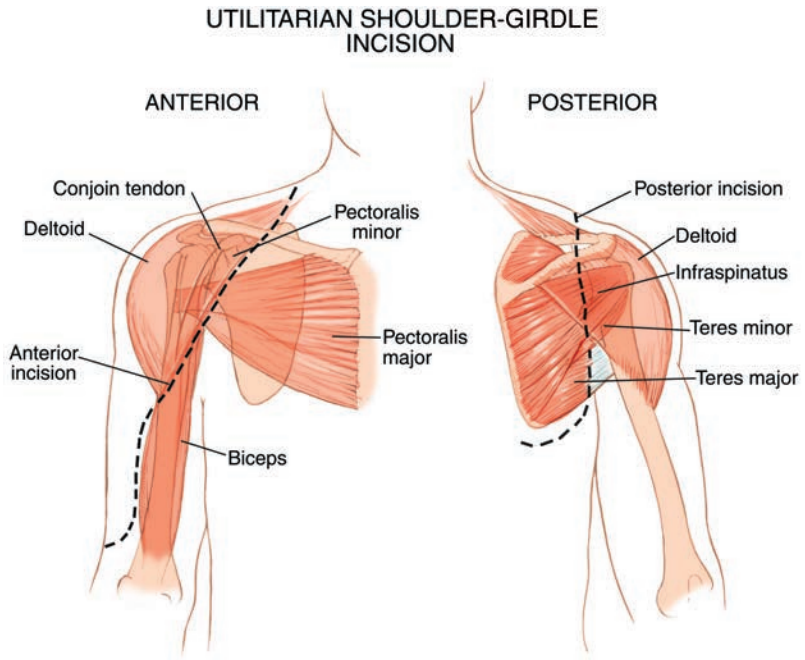


Figure 37-9 Utilitarian shoulder girdle incision. The utilitarian shoulder incision consists of three components: **(1)** A long deltopectoral incision from the midthird of the clavicle through the deltopectoral interval and distally along the medial aspect of the arm. **(2)** A curved incision along the axillary border of the scapula, which permits a large posterior fasciocutaneous flap to be developed to expose the scapula and the periscapular musculature. **(3)** An axillary incision that permits a forequarter amputation in conjunction with the first two incisions. (From Malawer M, Wittig JC. Resections of the shoulder girdle. In: Malawer MM, Sugarbaker PH, eds. *Musculoskeletal cancer surgery: treatment of sarcomas and allied diseases.* Dordrecht, Netherlands: Kluwer Academic Publishers, 2001:191.)

closure of the wound (Fig. 37-10). Twenty milliliters of 0.25% of Marcaine is perfused initially, and then a continuous infusion of 2 to 4 mL/hr of .025% for the immediate postoperative period is given. This technique reduces the postoperative narcotic requirements by about 90%.

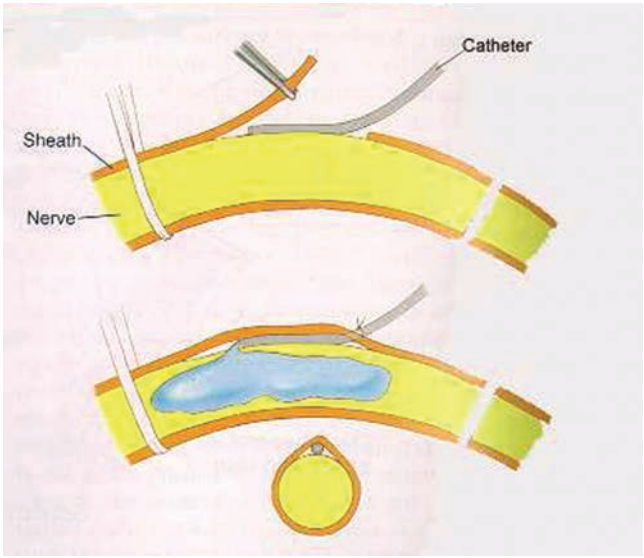


Figure 37-10 Schematic diagram of the perineural catheter utilized for perioperative pain control following most shoulder-girdle resections. At the time of surgery, a small Silastic catheter is placed in the nerve sheath of the brachial plexus. Twenty milliliters of 0.25% bupivacaine are bloused prior to wound closure, and then 4 to 8 mL/hr are infused over the next 48 to 72 hours. This provides excellent pain control for most shoulder-girdle procedures. (From Sugarbaker PH, Bickels J, Malawer M. Above-knee amputation. In: Malawer MM, Sugarbaker PH, eds. *Musculoskeletal cancer surgery: treatment of sarcomas and allied diseases.* Dordrecht, Netherlands: Kluwer Academic Publishers, 2001:360.)

REHABILITATION AFTER SHOULDER-GIRDLE RESECTION

From a rehabilitation perspective, the outcome of resection is clearly superior to that of a forequarter amputation or shoulder disarticulation. Patients undergoing shoulder-girdle resection retain hand function and good elbow function, but they lose some shoulder function, mainly abduction. Shoulder-girdle resection is less disfiguring than amputation and is associated with only minimal pain and edema. Generally, patients' acceptance of the outcome of their surgery is good to excellent.

Rehabilitation begins with an orientation program that often features pictures of patients who have undergone the procedure and demonstrations of what one can do postoperatively. Preoperatively, a shoulder mold is fashioned using the involved shoulder, provided its contours are not distorted. The cosmetic shoulder helps preserve the symmetry and appearance of the shoulder contour and can support a bra strap or heavy overcoat.

The patient uses a sling postoperatively, and motion is restricted until the incision is healed. The sutures are removed about 2 weeks after surgery. Edema is controlled with an elasticized glove or elastic stockinet. Active motion of the elbow and hand is initiated to preserve strength and range of motion and to help minimize edema.

If the incision heals per primam, assistive elbow motion is started within the confines of the sling as soon as the suction catheters have been removed. At about 2 weeks, the sling is removed for passive shoulder range of motion (ROM) and pronation and supination of the wrist. The patient should continue to use the sling intermittently after the incision is healed, primarily for upright activities

in which arm support increases comfort. Once the arm is out of the sling, full ROM of elbow (flexion, extension, pronation, and supination) is performed. Passive ROM to the shoulder (flexion, abduction, and external and internal rotation and pendulum exercise) with the help of a family member or physical therapist is recommended.

Rehabilitation depends on the type of reconstructive technique. In general, patients with endoprosthetic intraarticular allografts or composite allograft reconstruction undergo the same rehabilitation program. Those treated by arthrodesis, allograft, or autograft are immobilized for 4 to 5 weeks to allow early bony union to take place.

TUMORS OF THE PROXIMAL HUMERUS

Despite the complexity of these cases, limb-sparing surgery for both high- and low-grade sarcomas of the proximal humerus is possible in approximately 95% of cases. Forequarter amputation is indicated mainly for large, fungating tumors, tumors with secondary infections, cases in which there is chest wall involvement, and patients who have had a failed attempt at limb-sparing resection. Preoperative neoadjuvant chemotherapy may allow fracture healing if there is significant tumor necrosis.

Most low-grade sarcomas of the proximal humerus can be treated by type I excision with minimal functional deficit. High-grade sarcomas require a modified Tikhoff-Linberg resection (type V). Intraarticular and synovial involvement is more common with high-grade chondrosarcomas and with osteosarcomas of the shoulder girdle than with such tumors at other anatomic sites. Thus, extraarticular, rather than intraarticular, resections are recommended for high-grade tumors of the proximal humerus. Prosthesis, allograft, or allograft prosthetic composite can be used for reconstruction following a marginal resection (type I) for a low-grade lesion. Arthrodesis is rarely performed today. Following resection of a high-grade lesion (stage IIB), the aim is to provide a stable shoulder that will preserve function in the elbow and hand. Regardless of the type of reconstruction planned, the magnitude of the surgical resection depends on the grade of the tumor and its anatomic extent.

A major consideration in the preoperative evaluation and surgical planning is the intraosseous extension of the tumor within the bone marrow. The humerus is shorter than the femur and tibia, the two most common sites of sarcomas, and large tumors of the humerus often require resection of a significantly larger portion of the bone. It is not unusual to resect 50% to 80% of the humerus. Tumors arising within the diaphysis may require a total humeral resection and replacement of the glenohumeral and elbow joints. The surgeon must have various lengths and diameters of intramedullary stems at hand. The final decision about the extent of resection needed is made at the time of surgery.

The abductor mechanism (i.e., the deltoid muscle and the rotator cuff) normally covers the shoulder joint. These structures are usually resected in patients with high-grade proximal humeral sarcomas. Following the resection, joint coverage and stability are essential to eliminate dead space, decrease the risk of infection, and maintain good elbow and hand function. The key muscle transfers in the reconstruction are the pectoralis major, the biceps, and the latissimus dorsi; these must be identified and preserved during the resection.

Specific tumors of the Proximal Humerus (Table 37-2)

Benign Tumors

Giant Cell Tumor

Giant cell tumor (GCT) is a locally aggressive tumor with a low metastatic potential. It occurs slightly more often in females than in males. This tumor is thought to arise in the metaphyseal–epiphyseal junction, and large tumors may extend into the metaphysis or, more rarely, the diaphysis. The descriptor "benign" was first applied to GCT to differentiate it from other bony malignancies that required amputation. GCT is now considered a benign aggressive lesion, although 3% to 5% are primarily malignant or will undergo malignant transformation either after radiation therapy or following several local recurrences (Fig. 37-11).

GCTs are eccentric lytic lesions without matrix formation. They have well-defined borders and a sharp transition between the tumor and host bone. Periosteal elevation is rare unless accompanied by a pathologic fracture. The typical GCT comprises two basic cell types. The stroma consists of polygonal to somewhat spindle-shaped cells. Scattered diffusely through the stroma are benign osteoclast-like giant cells. Extensive hemorrhage, fracture, or previous surgery can alter the usual pathologic picture of GCT and make it resemble that of a primary bone sarcoma. Cystic areas with surrounding hemosiderin pigment and xanthoma cells correspond to the grossly observed cyst. Approximately 5% of all GCTs occur around the shoulder girdle.

Treatment of GCT is surgical removal (curettage) along with administration of an adjuvant cytotoxic agent such as phenol, zinc chloride, alcohol, hydrogen peroxide, or carbolic acid, or, as the authors prefer, curettage and application of a physical adjuvant such as cryosurgery. Treatment of GCT with curettage, burr drilling, and application of cryosurgery has achieved a local recurrence rate of less than 5% (see Cryosurgery section). Type I resection for GCTs is rarely necessary and is reserved for those tumors in which there is insufficient bone remaining for reconstruction with polymethylmethacrylate (cementation). We recommend the treatment of GCT of the proximal humerus with curettage and cryosurgery.

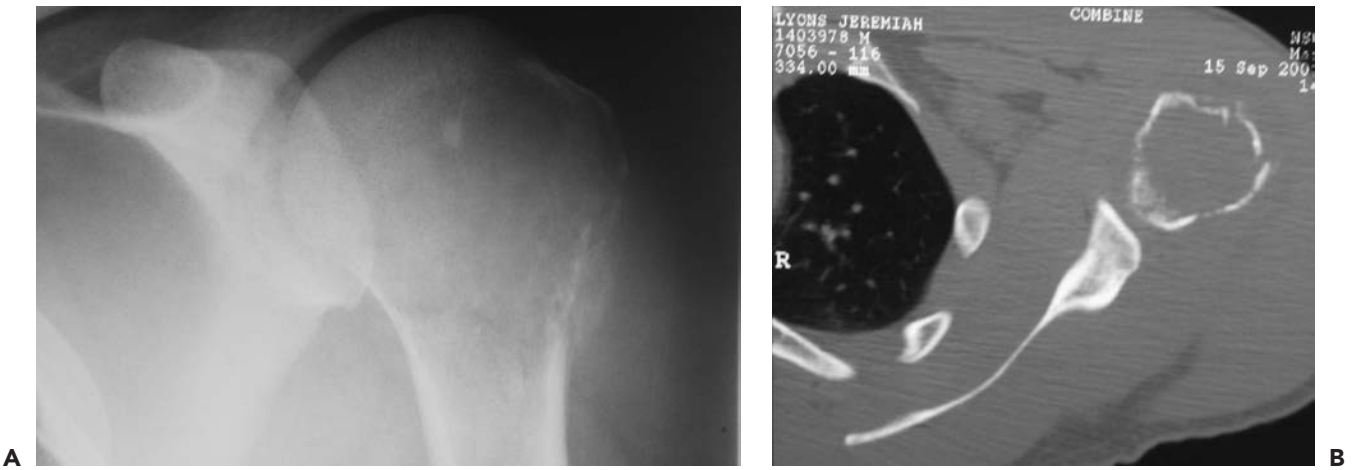
TABLE 37-2
HISTOGENESIS AND ANATOMIC SITE OF TUMORS IN 72 PATIENTS TREATED BY LIMB-SPARING SURGERY OF THE SHOULDER GIRDLE

Type of Tumor	Scapula	P. Humerus	Total
Chondrosarcoma	4	13	17
Osteosarcoma	4	24	28
Ewing's sarcoma	1	1	2
Giant cell tumor	1	0	1
Osteochondroma	2	0	2
Fibrosarcoma	2	0	2
Fibromatosis	3	0	3
Hemangiopericytoma	2	0	2
Synovial sarcoma	0	1	1
Leiomyosarcoma	0	1	1
Malignant fibrous histiocytoma	1	3	4
Hypernephroma	1	3	4
Hemangiosarcoma	0	1	1
Pleomorphic sarcoma	1	1	2
Paget's sarcoma	0	1	1
Osteoblastoma	0	1	1
TOTAL	**22**	**50**	**72**

Presented at Surgical Grand Rounds at Georgetown University Medical Center May 2001. Washington DC (not published)

Chondroblastoma (Codman's Tumor)

Chondroblastoma is a benign aggressive tumor that shows a marked predilection for the epiphysis of the bone. This tumor was originally described by Codman as occurring in the proximal humeral epiphysis (thus named Codman's tumor). It is composed of round and spindle cells, some of which resemble immature chondrocytes or chondroblasts, hence the name *chondroblastoma*. Chondroblastomas account for less than 1% of all bone tumors. They are one-fifth as common as GCTs. Most patients are skeletally immature when this tumor occurs: 95% of patients are between 5 and 25 years of age, and most of these tumors occur in teenagers. If a chondroblastoma occurs after skeletal maturity, one must be very suspicious of a clear cell chondrosarcoma. Males are affected twice as often as females. Patients usually present with mild pain that may have been present for several months. About one-third of patients have a joint effusion or fluid



A B

Figure 37-11 Giant cell tumor of the proximal humerus. **(A)** Plain radiograph demonstrating a lytic lesion of the proximal humerus with poorly defined borders and with cortical destruction. There is no matrix formation. **(B)** Computed tomography scan showing marked cortical thinning and destruction. Needle biopsy showed a giant cell tumor. Giant cell tumors of the proximal humerus are very rare and represent less than 1% to 2% of all giant cell tumors.

in the joint, and swelling and limitation of the joint may occur.

Ninety-eight percent of chondroblastomas are located in the epiphysis of the bone. Mature cartilage is present only focally in some tumors. Chondroblastomas are almost always confined to the original bone, but on rare occasions they can penetrate the cortex and enter the joint or the soft tissues. Chondroblastomas may also undergo aneurysmal changes with secondary aneurysmal bone cyst (ABC) formation. In this setting, the tumor undergoes necrosis with hemorrhage and large cystic spaces that are filled with bloody fluid compose a significant portion of the tumor. Since they are aggressive, these tumors can cause extensive destruction of the bone. Treatment is similar to that of GCT and includes a thorough curettage of the tumor and tumor cavity.

Cryosurgery is recommended to eradicate microscopic tumor cells. The tumor cavity may be packed with cement and/or bone graft and may require metallic internal fixation. There have been rare reports of chondroblastoma metastasizing to the lungs; however, patients can still be cured with metastasectomy. In some cases, metastases do not appear for up to 30 years following the initial diagnosis. There are reported cases of chondroblastomas treated with radiation that have later undergone malignant transformation to fibrosarcomas and osteosarcomas. Thus, one should refrain from using radiation to treat this type of tumor. The first procedure should be undertaken with great care to ensure local control of this tumor.

Enchondroma

An enchondroma (sometimes called a central chondroma) is a benign intramedullary tumor of cartilage. It accounts for about 10% of all benign bone tumors. These tumors are believed to arise from cartilaginous nests of cells that are displaced from the growth plate during development. Enchondromas occur in persons of virtually all ages, but 60% of patients are between 15 and 40 years of age. Some patients complain of pain due to stress fractures through the area of the tumor. In other cases, the tumors are asymptomatic and are discovered incidentally on radiographs taken for other reasons. The tumors may also appear as hot spots in patients undergoing a skeletal scan for other reasons. Increased uptake on a bone scintigraphy is not a sign of malignancy but of enchondral ossification, which normally occurs in enchondromas. The proximal humerus is commonly affected.

Enchondromas must be differentiated from chondrosarcomas, and it is particularly difficult to differentiate a low-grade chondrosarcoma (also known as an enchondrosarcoma) from an enchondroma. Special clinical and radiographic criteria are used to differentiate the two entities. In general, low-grade chondrosarcomas are greater than 5 cm in size and cause endosteal cortical erosion through the cortex of the bone. They also typically occur in more-proximal locations than enchondromas do. The presence of pain and the absence of fracture should be regarded as highly suspicious for a low-grade chondrosarcoma. Tumors arising in the digits are rarely malignant. Chondrosarcomas, however, can arise from enchondromas. Thus, enchondromas should be followed with yearly radiographs and treated if they begin to cause pain, to erode into the inner cortex, or to increase to a size that suggests malignancy. Biopsy does not help distinguish a low-grade chondrosarcoma from an enchondroma. Most enchondromas are treated with bone graft and/or polymethylmethacrylate (PMMA). This tumor should not be biopsied because it is pathologically very difficult to distinguish between a low-grade chondrosarcoma and an enchondroma. The two lesions look very similar under a microscope, and if a biopsy from a small area of the lesion is performed, the results will be inconclusive. Thus, the entire tumor should be curetted if there is pain and endosteal scalloping. If radiographic and clinical information suggest that this may be a low-grade chondrosarcoma, then cryosurgery may be performed. The bone may require reconstruction with cement and/or bone graft and stabilization with intramedullary pins. Rarely is a primary prosthetic replacement required.

Unicameral Bone Cysts

A unicameral bone cyst (UBC) is a benign cavity of bone that is filled with clear or bloody fluid. Other terms for this lesion include benign bone cyst and simple bone cyst. UBCs constitute 3% of all primary bone lesions. Most occur in the proximal or midhumerus. UBC does not appear to be a true tumor, and its cause is unknown. It may occur secondarily to intraosseous hypertension and failure of venous outflow in the region of the bone. There is a male predilection of 3:1. Sixty-five percent of UBCs occur in teenagers, and 20% occur in the first decade of life. The patient usually presents with pain. Despite popular belief, fracture of the bone cyst usually does not result in permanent healing. Preferred treatment is aspiration of the cyst followed by high-pressure injection with cortisone and Renografin. The procedure is performed under fluoroscopy to ensure that the entire cavity is filled and that the fluid enters the venous outflow, thus restoring circulation in the area of the cyst. The procedure is done under general anesthesia. The majority of cysts are cured following the first injection; however, some require several injections. Two-thirds of patients are cured with three injections or less. Patients who are not cured in this manner can be treated with curettage and bone grafting or bone-filling substitute. Patients with fractures through the area of the cyst are immobilized until the fracture heals and are then treated with aspiration and injection. Aspirated bone marrow or growth factors may be injected into the cyst to aid with healing (Fig. 37-12).

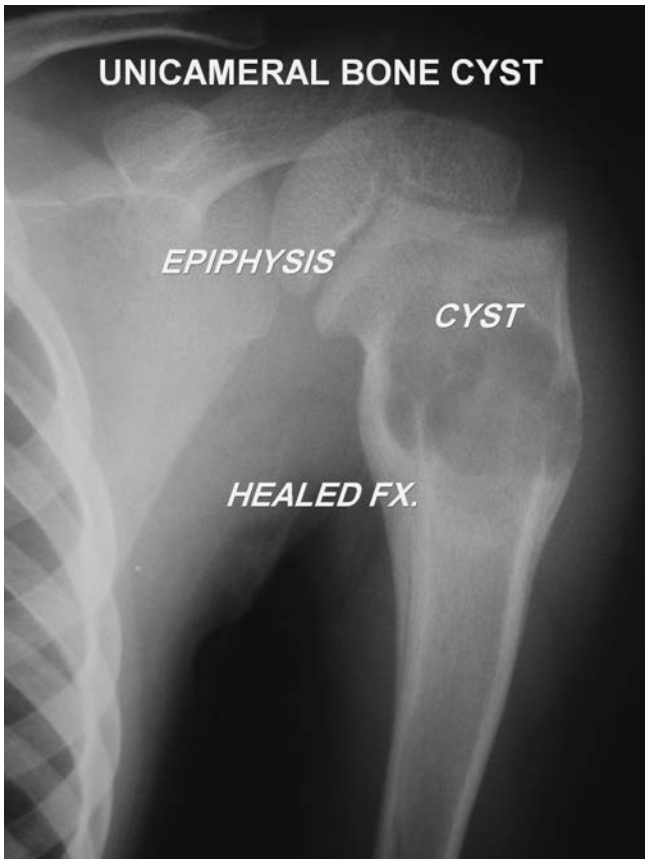


Figure 37-12 Healed unicameral bone cyst (UBC) of the proximal humerus. UBCs are typically asymptomatic until a fracture occurs. Surgical treatment is postponed until the fracture is healed. UBC can be treated by several modalities including aspiration and injection with methylprednisolone, curettage, and filling of the defect with one of many bone substitutes. Alternatively, autogenous bone graft can be utilized.

Malignant Tumors

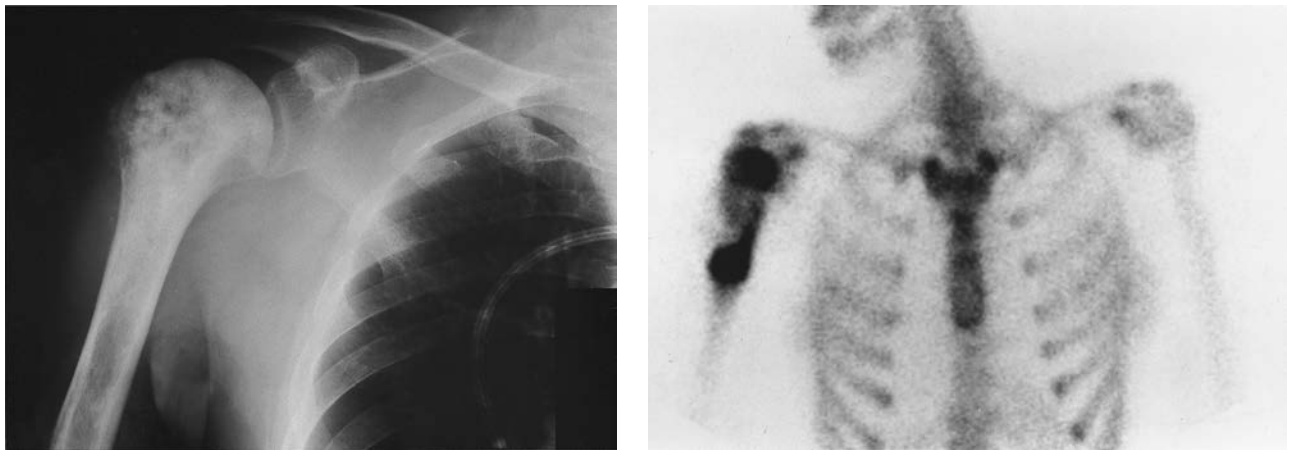
Osteosarcoma

Osteosarcoma is a high-grade, malignant spindle cell tumor that most often arises within a bone. Its distinguishing characteristic is the production of "tumor" osteoid or immature bone directly from a malignant spindle cell stroma.

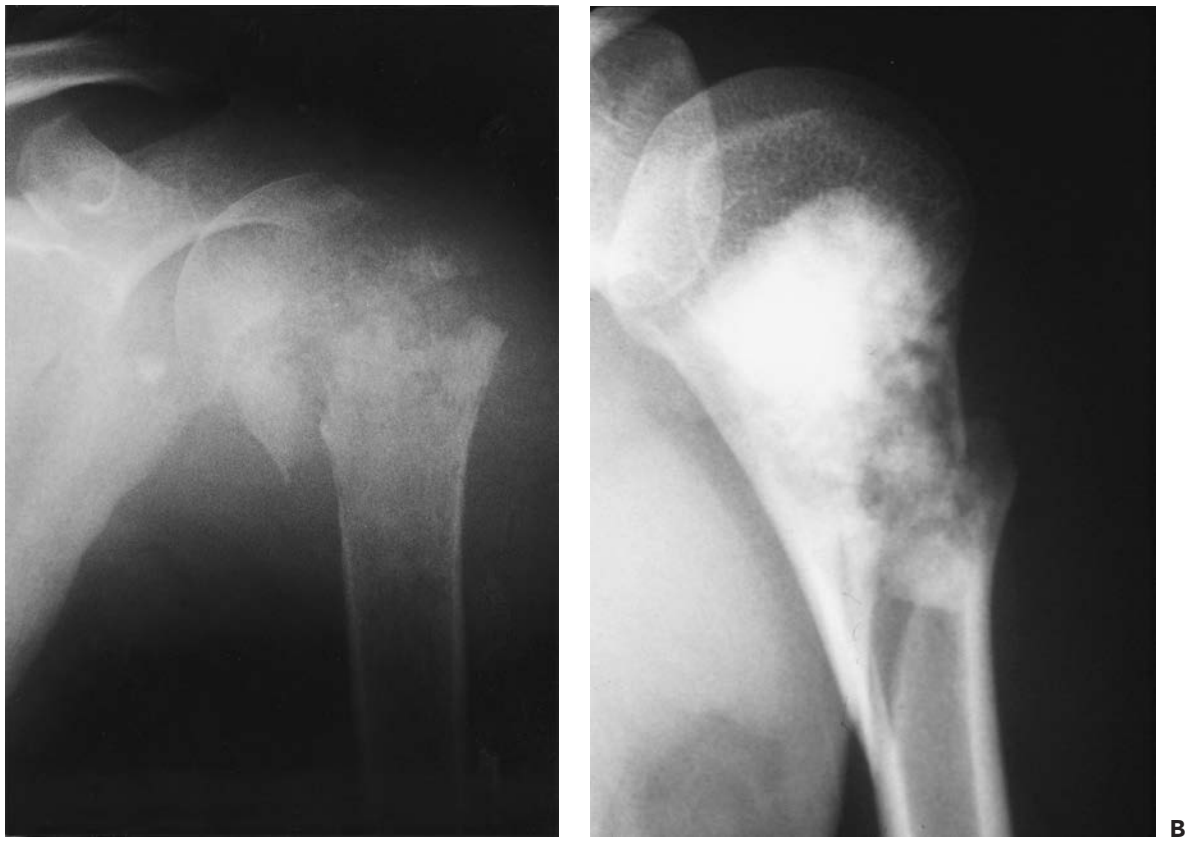
Osteosarcoma typically occurs during childhood or adolescence, with the peak incidence to be between 10 and 19 years of age. The overall incidence is 2.1 cases per million people per year. When osteosarcoma occurs in patients older than 40 years, it is usually associated with a preexisting condition, such as Paget's disease, irradiated bones, multiple hereditary exostosis, or polyostotic fibrous dysplasia.

Bones of the knee joint and the proximal humerus are the most common sites, accounting for 50% and 25%, respectively, of all osteosarcomas. Between 80% and 90% of osteosarcomas occur in the long tubular bones, and the axial skeleton is rarely affected. With the exception of serum alkaline phosphatase (AP) levels, which are elevated in 45% to 50% of patients, laboratory findings are usually not helpful. Furthermore, elevated AP per se is not diagnostic, because it is also found in association with other skeletal diseases. Pain is the most common complaint. Night pain gradually develops and is a hallmark of skeletal involvement. Physical examination demonstrates a firm, soft mass fixed to the underlying bone with slight tenderness. No effusion is noted in the adjacent joint, and motion is normal. Incidence of pathologic fracture is less than 1%. Systemic symptoms are rare.

The proximal humerus is the third most common site for osteosarcoma. Osteosarcomas in this area tend to have a poorer prognosis than those around the knee, and most have significant extraosseous components (Figs. 37-13 and 37-14). Plain radiographs suggest the correct diagnosis. All



Figures 37-13 Osteosarcoma of the proximal humerus. **(A)** Plain radiograph showing marked sclerosis of the proximal one-third of the humerus with cortical destruction. The tumor extends from the proximal one-third of the diaphysis to the subchondral bone. **(B)** Bone scan (late phase) of the proximal humerus showing marked bony uptake. This bone scan alone is not characteristic of an osteosarcoma, but it is indicative of the extension of the lesion. Note that there is only minimal extraosseous component demonstrated.

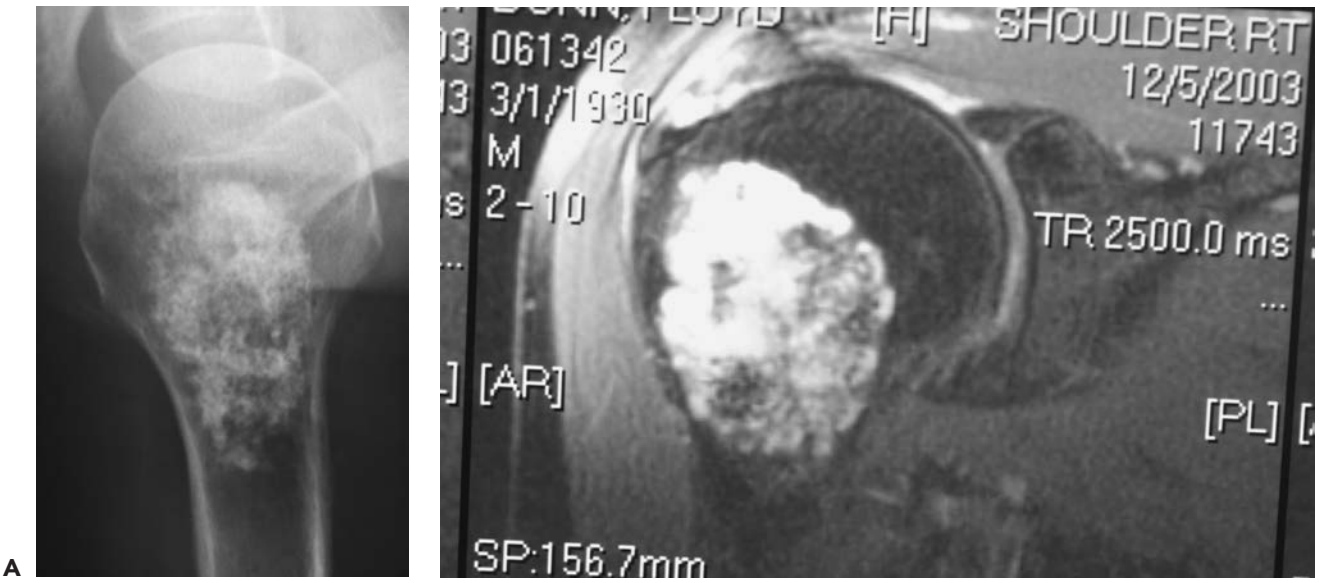


Figures 37-14 Osteosarcoma of the proximal humerus with a pathologic fracture. (A) Plain radiograph showing a pathologic fracture prior to treatment with induction chemotherapy. (B) The same patient following 4 months of induction chemotherapy, prior to attempted limb-sparing surgery. Note that the pathologic fracture has healed and that the tumor has undergone ossification (both positive prognostic indicators). Pathologic fracture through an osteosarcoma classically required a forequarter amputation. Today, with induction chemotherapy, most pathologic fractures will heal, indicating significant tumor necrosis.

staging studies are performed prior to biopsy. If the axillary vessels are free of tumor, a limb-sparing procedure, preferably an extraarticular resection, is generally indicated. A modified Tikhoff-Linberg procedure (type VB) provides adequate resection of the proximal humerus for high-grade sarcomas. This includes en bloc removal of 15 to 20 cm of the humerus and shoulder joint, with the deltoid, rotator cuff, and portions of the biceps and triceps muscles. Reconstruction involves suspension of the arm, motor reconstruction, and provision of adequate soft tissue coverage.

Extraarticular resection of the glenohumeral joint by medial scapulectomy is safer than intraarticular resection. A modular prosthesis is used for reconstruction. Soft tissue reconstruction and suspension are essential to stabilize the shoulder, prevent infection, and avoid postoperative pain, instability, and fatigue. Static suspension of the prosthesis is accomplished with Dacron tape. Dynamic suspension is accomplished by transferring the biceps tendon to the clavicle. The prosthesis is covered with the pectoralis major and latissimus muscles. Hand and wrist function is normal after resection. Shoulder abduction and flexion is minimal; however, rotation is preserved. Latissimus dorsi transfer and scapulothoracic motion permit external rotation. The pectoralis major enables internal rotation. Cosmesis is acceptable and can be enhanced with the use of a shoulder pad.

Alternatively, resection of the proximal humerus for osteosarcomas can be performed by an intraarticular resection that preserves the glenoid and the adjacent deltoid muscle. The problems associated with this procedure include significant local recurrence and instability of the reconstructed prosthesis or allograft. When the glenoid and deltoid are preserved in this procedure, minimum margins are obtained along the shoulder joint, deltoid muscle, and axillary nerve. Because of this serious drawback, this technique is not recommended by the surgical author (MM). Less than 5% of osteosarcomas of the proximal humerus (usually those without an extraosseous component [stage IIA]) can be treated by an intraarticular resection. When an intraarticular resection is performed, the senior author recommends reconstruction of the glenohumeral ligaments with a Gore-Tex aortic graft.



A B

Figures 37-15 Chondrosarcoma (enchondrosarcoma) of the proximal humerus. **(A)** Plain radiograph of the proximal humerus showing a large area of calcification with adjacent cortical thinning and destruction. There is a lytic component in addition to the calcified component. **(B)** Magnetic resonance imaging scan showing typical calcification appearance. Calcification is often described as small nodules and curly cues.

Chondrosarcoma

Primary (central) and secondary (peripheral) chondrosarcomas commonly occur in the proximal humerus. Peripheral lesions tend to be large but low grade, whereas central lesions tend to be higher grade. Stage I tumors of the proximal humerus can be treated by excision (type I) with minimal functional deficit (Fig. 37-15).

High-grade sarcomas require a modified Tikhoff-Linberg resection (type V) or, rarely, a forequarter amputation. Intraarticular and synovial involvement with high-grade cartilaginous lesions are more common in this location than in other sites. A prosthesis is recommended for reconstruction following a marginal resection (type I) for a low-grade sarcoma.

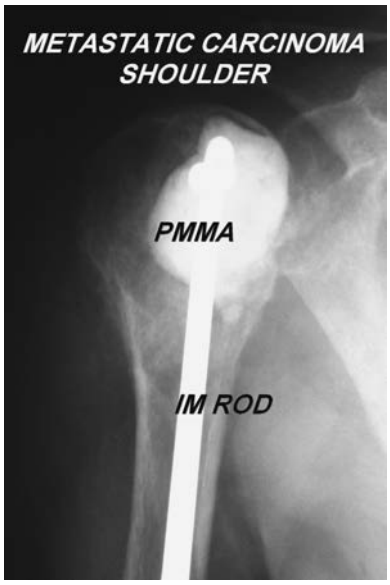
Ewing's Sarcoma

Treatment of Ewing's sarcoma follows the guidelines for other high-grade bone sarcomas of the humerus, even though fewer than 10% of Ewing's sarcomas involve the proximal humerus. The flat bones, specifically the scapula and clavicle, are the most common sites for Ewing's sarcoma. Ewing's sarcomas often decrease dramatically in size following preoperative chemotherapy, in which case the deltoid and axillary nerve may be preserved. Surgery should never precede induction chemotherapy. Often, there is no detectable soft tissue component; in such cases, a type I resection may be indicated. Ewing's sarcomas may dramatically decrease in size following induction chemotherapy. For this reason, intraarticular resections (type I) are recommended. Radiation therapy is not recommended in patients treated with a prosthesis or an allograft because it often leads to severe local complications, such as restriction of motion, infection, severe lymphedema, and secondary amputation.

Metastatic Carcinomas

All carcinomas can metastasize to the proximal humerus (Fig. 37-16). Many large metastatic tumors with marked bony destruction may be resected by a primary resection and prosthetic replacement. Hypernephroma, which is extremely vascular and has a predilection for this location, may present a unique problem of uncontrollable bleeding. Radiography often reveals marked destruction and ballooning, much like that seen in ABCs and primary sarcomas. Simple biopsy may lead to severe hemorrhage. Preoperative angiography with embolization is recommended. The anterior and posterior circumflex vessels should be ligated prior to any surgical procedure. If curettage and cementation are not feasible because of severe bony destruction, an intraarticular resection with prosthetic replacement (type I) is indicated.

Most metastatic carcinomas of the proximal humerus can be treated with radiation therapy rather than surgery. If there is a pathologic fracture, then curettage through a deltopectoral interval should be performed. The defect is reconstructed with PMMA, intramedullary pins, an intramedullary rod, or a long-stem hemiarthroplasty. Bone graft is not used to fill a defect if a metastatic carcinoma is present.



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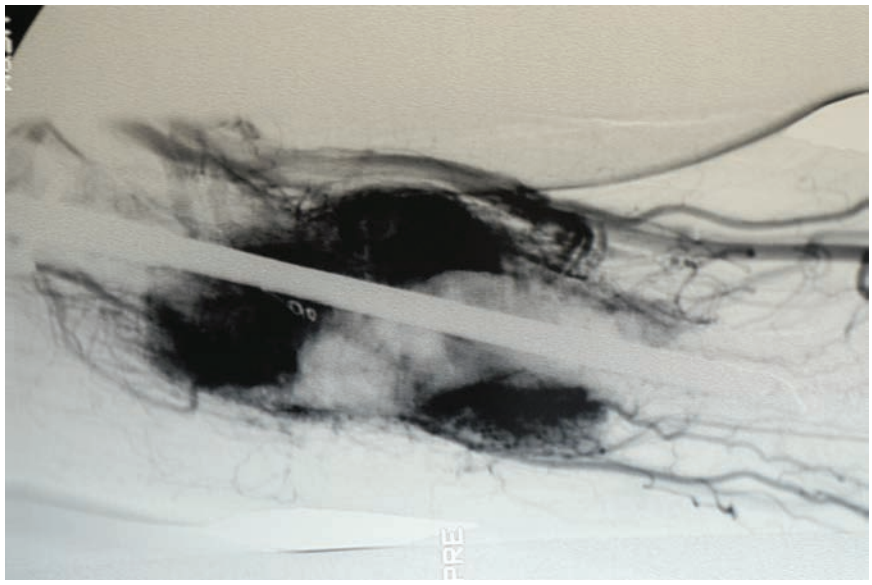

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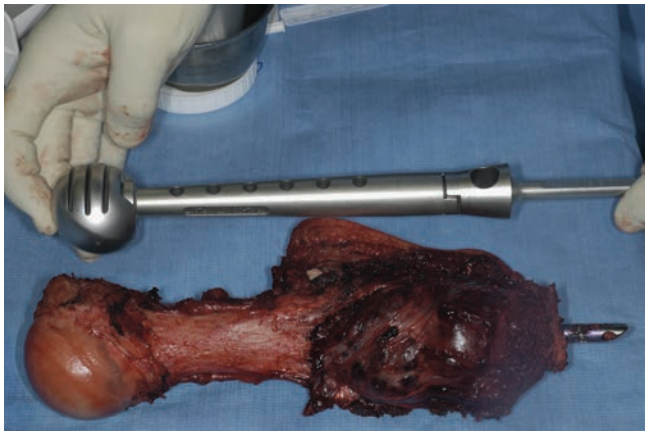


C

Figure 37-16 **(A)** Metastatic carcinoma of the proximal humerus treated by intramedullary rod fixation and cementation. Polymethyl methacrylate (PMMA) is used with all metastatic lesions of the humerus when surgery is required. Cementation provides immediate fixation and may prevent secondary loosening if the metastatic tumor recurs. This has become more of an issue as patients are living longer with improved therapies. **(B)** Large recurrent tumor (metastatic hypernephroma) of the humerus. Angiogram shows tumor recurrence around an intramedullary rod. Hypernephromas are often difficult to treat and are very vascular. Radiation has minimal effect on preventing a recurrence. We prefer primary resections of metastatic hypernephromas, specifically those that occur around the humerus. **(C)** A specimen of the proximal two-thirds of the humerus following resection for the recurrent hypernephroma. The segmental prosthesis that will replace the resected bone is shown. IM = intramedullary.

CRYOSURGERY

Cryosurgery extends the margin of curettage and makes it equivalent to that of wide resection. Compared with other techniques, cryosurgery with composite fixation (PMMA combined with intramedullary rods) not only preserves joint function, but also significantly decreases the rate of local tumor recurrence. Although a relatively simple procedure, cryosurgery can cause a significant morbidity if performed inappropriately. Effective and safe procedures must follow these consecutive steps: (1) adequate exposure of the tumor, (2) meticulous curettage and burr drilling, (3) soft tissue mobilization and protection before introduction of liquid nitrogen to the tumor cavity, (4) internal fixation of the tumor cavity, and (5) protection of the operated bone throughout the healing period (Fig. 37-17).

The technique for curettage and cryosurgery is as follows:

- All gross tumor is removed with hand curettes.
- After the tumor tissue is curetted away from the inner wall of the lesion, the reactive wall reveals an irregular contour. This irregularity makes it virtually impossible to remove all the tissue; therefore, high-speed burr drilling is achieved using a Midas Rex or Black Max.
- Liquid nitrogen is applied to the cavity utilizing liquid nitrogen or argon probes or, more often, the Marcove direct-pour method. All bony perforations are identified and sealed, and the surrounding skin, soft tissues, and neurovascular bundle are protected by mobilization and shielding with Gelfoam.
- Using the direct-pour method, liquid nitrogen is poured through a stainless steel funnel into the tumor cavity. Care is taken to fill the entire cavity. The Gelfoam immediately freezes and forms a seal around the funnel. Thermocouples are used to monitor the freezing effect within the bone cavity, cavity wall, and adjacent soft tissue, as well as the rim of bone 1 to 2 cm from the periphery of the cavity. The surrounding soft tissues are continuously irrigated with warm saline solution to decrease the possibility of thermal injury. The liquid nitrogen is left to evaporate, and then spontaneous thaw is allowed to occur over 3 to 5 minutes. Once the

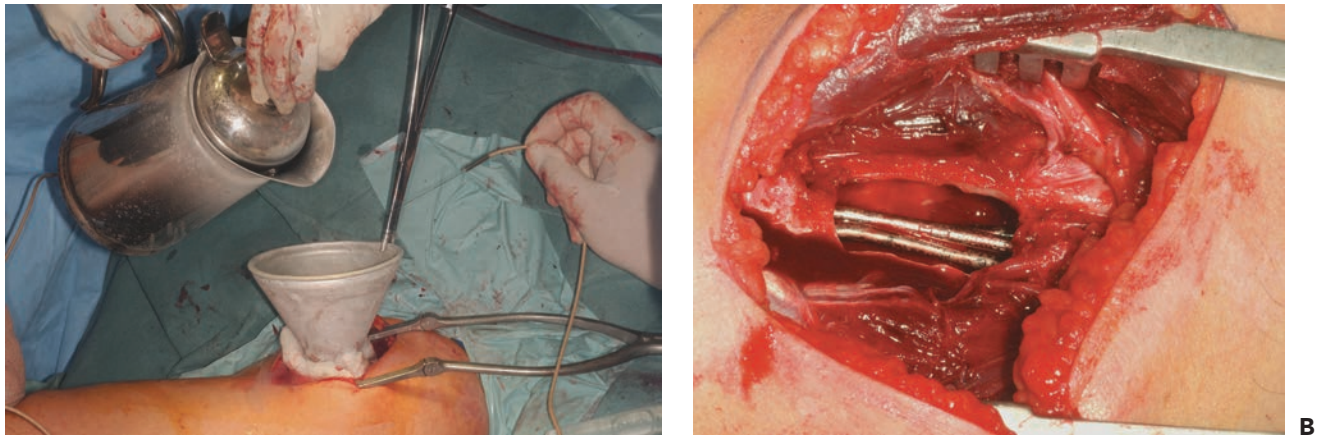


Figure 37-17 Cryosurgery. **(A)** This operative photograph shows the early technique of cryosurgery as developed by Dr. Ralph Marcove using the direct-pour method of liquid nitrogen directly within the tumor cavity. Presently, we use cryoprobes for smaller lesions and the direct-pour method for larger lesions of bone. **(B)** Reconstruction of the curetted defect. It is necessary to reconstruct large defects following curettage resection, especially for giant cell tumors and chondrosarcomas. We prefer the technique of composite reconstruction using Steinmann pins to support the cavity with polymethyl methacrylate cementation.

temperature of the cavity rises above 0°C, the cycle is considered complete. Two to three freeze-thaw cycles are administered, with a saline irrigation occurring between cycles.

Reconstruction is achieved utilizing PMMA, internal fixation, and subchondral bone graft, which provides immediate stability and structural support for large defects and allows early rehabilitation of the adjacent joint.

TECHNIQUE OF TYPE I RESECTION AND RECONSTRUCTION

If an intraarticular (type I) resection is to be performed, the axillary nerve and the deltoid muscle must not be invaded by tumor and must be preservable (Figs. 37-18 to 37-21):

- The utilitarian shoulder incision is utilized.
- The pectoralis major is detached and retracted to expose the axillary vessels and nerves.
- The neurovascular bundle must be dissected out carefully and retracted away from any soft tissue component of the tumor.
- The musculocutaneous nerve, radial nerve, and axillary nerve must all be preserved to ensure optimal hand, arm, and elbow function.
- The pectoralis major muscle is detached and reflected toward the chest wall.
- The pectoralis minor muscle and the conjoined tendon are then released from the coracoid.
- The proximal humerus is now exposed and resection can take place.
- To reconstruct the defect, a Gore-Tex aortic graft is sutured to the remaining portion of the glenoid with Dacron tape.
- The humeral head of the prosthesis is inserted within the Gore-Tex and then sutured through holes in the prosthesis using Dacron tape.

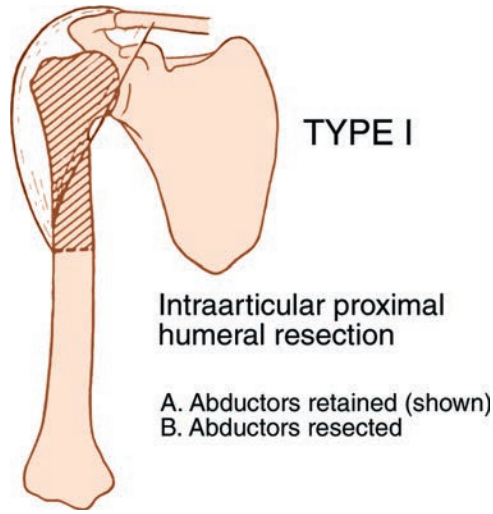


Figure 37-18 Type I shoulder girdle resection. Intraarticular proximal humeral resection with preservation of the deltoid muscle. Type I resections are usually performed for low-grade malignant tumors, such as chondrosarcomas and aggressive giant cell tumors. In general, type I resections are not performed for high-grade sarcomas. (From Malawer M, Wittig JC. Resections of the shoulder girdle. In: Malawer MM, Sugarbaker PH, eds. *Musculoskeletal cancer surgery: treatment of sarcomas and allied diseases*. Dordrecht, Netherlands: Kluwer Academic Publishers, 2001:184.)

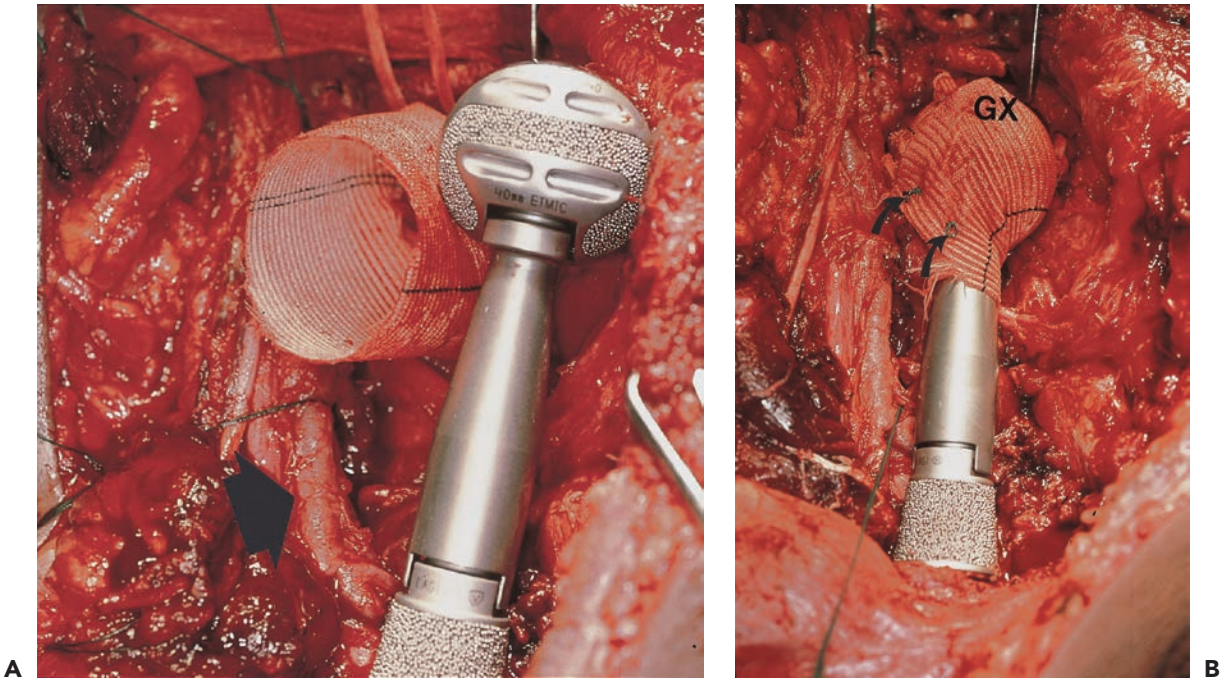


Figure 37-19 Gore-Tex reconstruction in conjunction with a proximal humeral replacement following a type I resection. Gore-Tex graft reconstruction of the capsule is essential to reconstruct shoulder stability. **(A)** The Gore-Tex is sutured to the remaining glenoid rim with Ethibond and Dacron tape. The prosthesis is then placed within this graft and sutured in place with Dacron tape through the Gore-Tex and the holes on the prosthesis. **(B)** Completion of Gore-Tex graft capsular reconstruction. Note the close relationship of the axillary nerve and the infraclavicular portion of the brachial plexus.

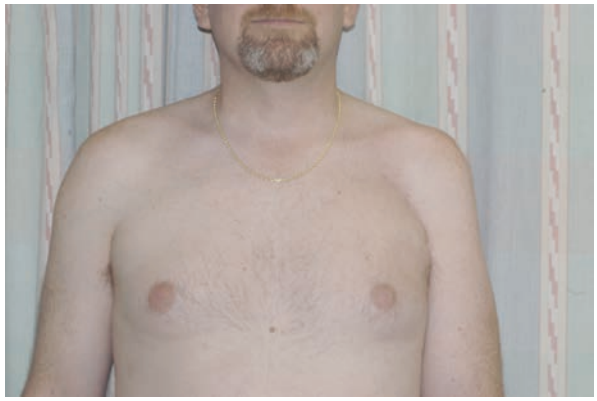
- The remaining capsular structure is sutured to the Gore-Tex to reinforce the capsule and the rotator cuff, as well as to prevent shoulder dislocation or subluxation.

TECHNIQUE OF EXTRAARTICULAR PROXIMAL HUMERAL RESECTION AND PROSTHETIC RECONSTRUCTION (TYPE VB)

The technique of extraarticular proximal humeral resection and prosthetic reconstruction may be modified for an intraarticular resection. We recommend an extraarticular (type VB) resection (Fig. 37-22). In general, the deltoid muscle and axillary nerve cannot be preserved for high-grade sarcomas. The anterior and posterior components of the utilitarian shoulder-girdle incision are utilized (see previous text). Three osteotomies are required for an extraarticular resection.

Resection (Figs. 37-23 and 37-24)

- Place the patient in a lateral position, allowing some mobility of the upper torso.
- Prepare the skin down to the level of the midline anterior and posterior to the umbilicus, and cranially past the hairline.
- Start the incision over the junction of the inner and middle thirds of the clavicle, continue along the deltopectoral groove, and then move down the arm over the medial border of the biceps muscle.
- Excise the biopsy site, leaving a 2- to 3-cm margin of normal skin. Do not open the posterior incision until the anterior dissection is complete.
- For exploration of the axilla, open the skin through the superficial fascia.
- Dissect the skin flap anteriorly off the pectoralis major muscle to expose its distal third, and uncover the short head of the biceps muscle. The key to exposure of the anterior shoulder girdle and axilla is the detachment and mobilization of the pectoralis major muscle with partial mobilization medially toward the chest wall.
- Dissect the pectoralis major muscle overlying the axilla free of fat, so that its insertion on the humerus can be visualized. Divide this muscle just proximal to its tendinous insertion on the humerus, and use a suture to tag the portion of muscle remaining with the patient.
- Identify the axillary sheath and visualize the coracoid process. To expose the axillary sheath along its full extent, divide the pectoralis minor, the short head of the biceps, and the coracobrachialis muscles at their insertion on the coracoid process. Tag all proximal muscles with a suture for later identification and use in reconstruction.



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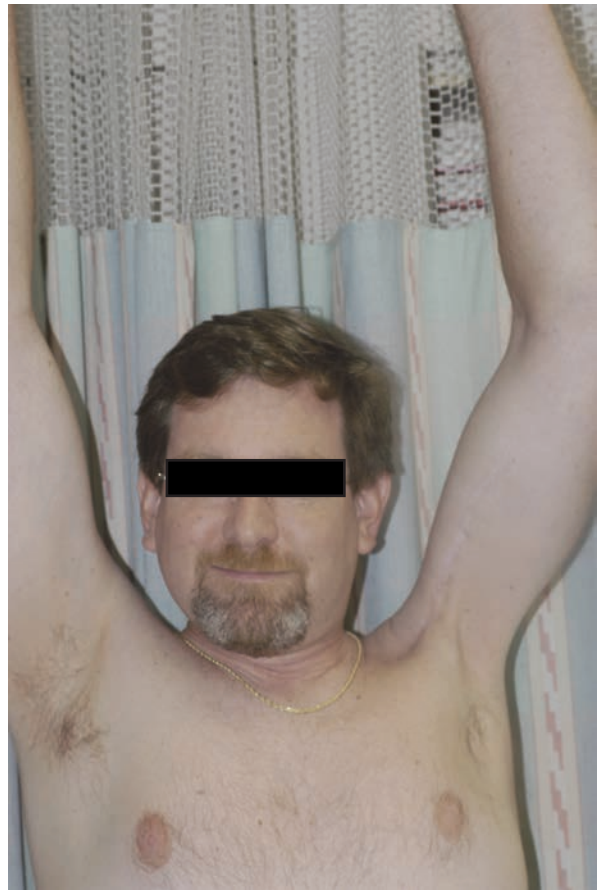

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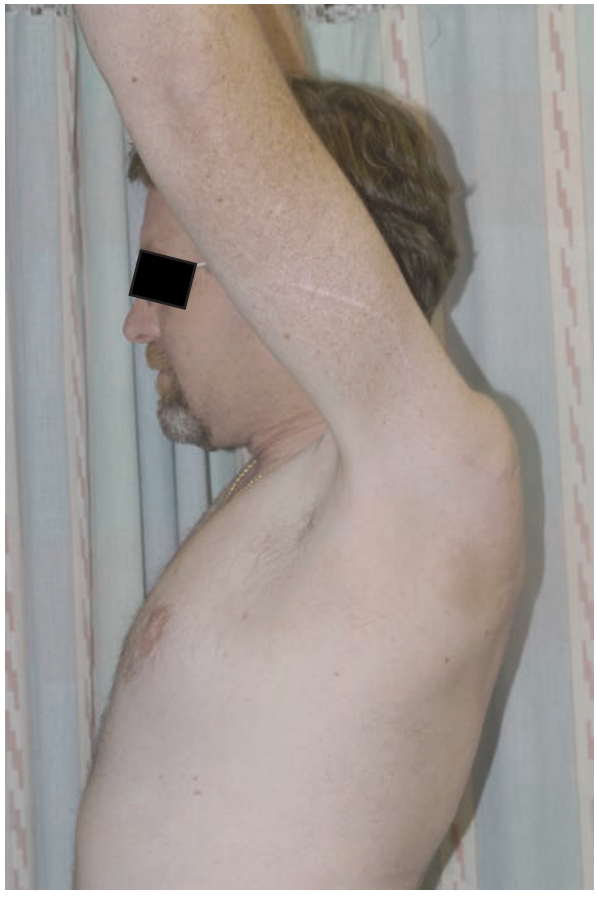

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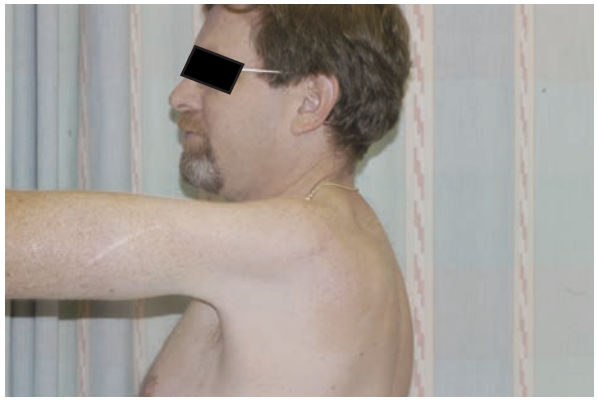

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Figure 37-20 **(A–D)** Clinical photographs of a patient 5 years after a type I proximal humeral resection with Gore-Tex graft reconstruction. Functionally, there is minimal loss of abduction. There is normal internal and external rotation and forward flexion. The Gore-Tex graft avoids subluxation or dislocation, which has been a common problem reported in the literature following proximal humeral replacement.

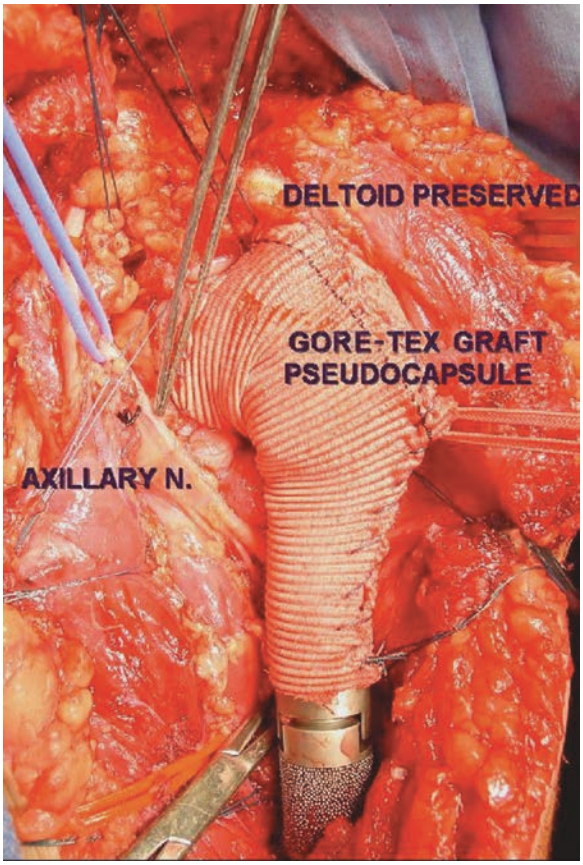


Figure 37-21 Intraoperative photograph showing Gore-Tex reconstruction of the shoulder joint capsule following an intraarticular resection of the humerus. The segmental prosthesis can be seen. The Gore-Tex is sutured to the glenoid and then to fixation holes on the proximal humerus. Gore-Tex reconstruction is always utilized following an intraarticular resection to prevent secondary subluxation or dislocation of the prosthesis.

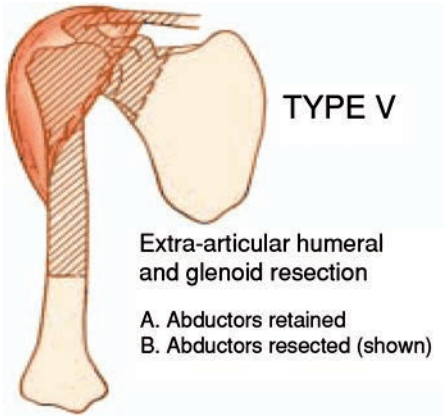


Figure 37-22 Schematic diagram of a type VB shoulder girdle resection. This represents an extraarticular humeral and glenoid resection with removal of the abductor muscles. (From Malawer M, Wittig JC. Resections of the shoulder girdle. In: Malawer MM, Sugarbaker PH, eds. *Musculoskeletal cancer surgery: treatment of sarcomas and allied diseases.* Dordrecht, Netherlands: Kluwer Academic Publishers, 2001:185.)

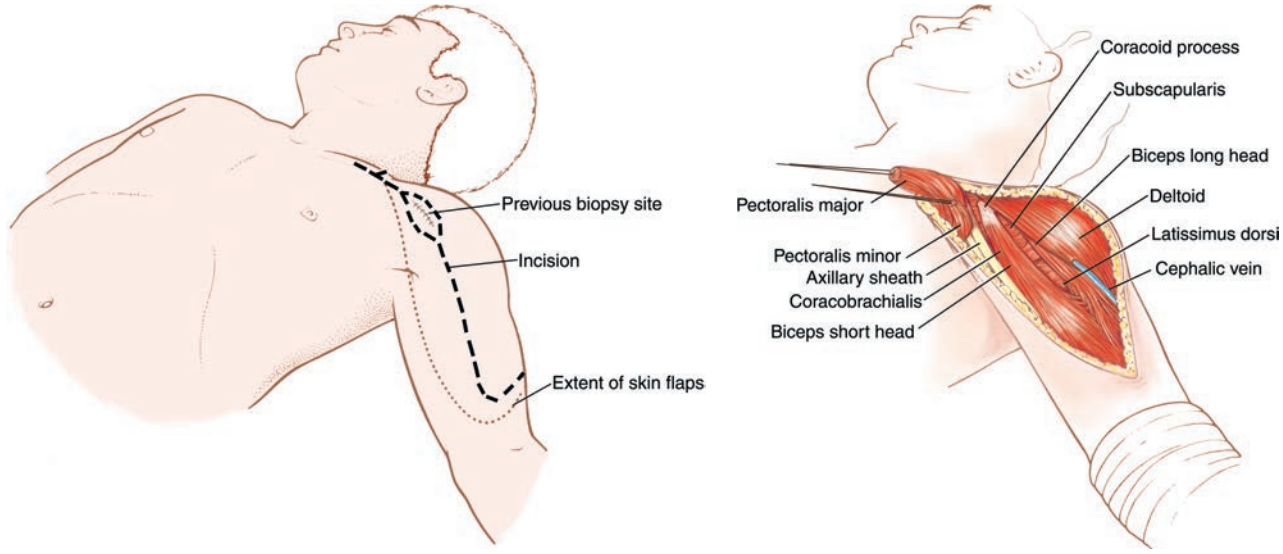


Figure 37-23 Surgical technique of a proximal humeral resection (see text). **(A)** Anterior incision extends from the midclavicle, through the deltopectoral interval, and down the arm as shown. (From Malawer M, Wittig JC. The Tikhoff-Linberg procedure and its modifications. In: Malawer MM, Sugarbaker PH, eds. *Musculoskeletal cancer surgery: treatment of sarcomas and allied diseases.* Dordrecht, Netherlands: Kluwer Academic Publishers, 2001:531.) **(B)** The important first step is identification of and release of the pectoralis major from the proximal humerus and reflecting it upon the chest wall. This exposes the axillary structures. It is important to carefully dissect and identify the infraclavicular portion of the brachial plexus and the nerves (axillary and musculocutaneous) prior to ligation. (From Malawer MM. Tumors of the shoulder girdle, technique of resection and description of a surgical classification. *Orthop Clin N Am* 1991;22:7–35.)

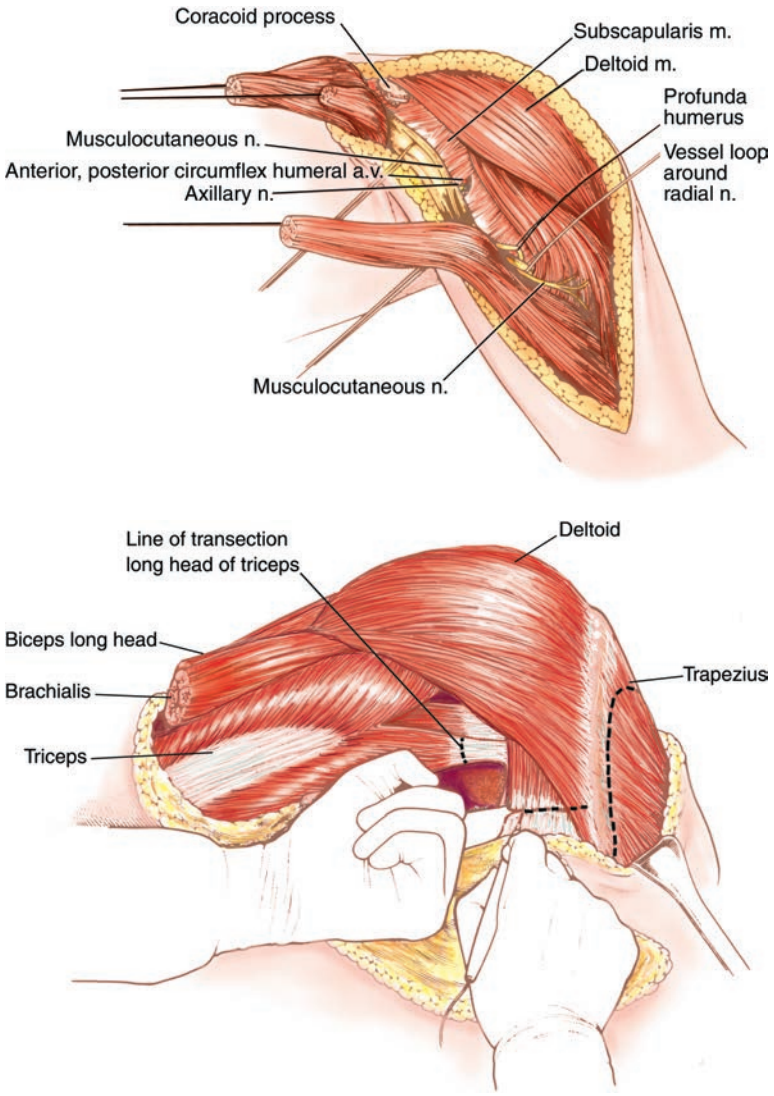


Figure 37-24 **(A)** Second stage of release of musculature of the axillary space. The coracobrachialis is released from the coracoid and the pectoralis minor is released and reflected toward the chest. This exposes the axillary fascia and the axillary vessels. Care is needed to dissect out the individual nerves prior to ligation. The axillary nerve is ligated as well as the anterior and posterior circumflex vessels at the lower border of the subscapularis muscle. The radial nerve is identified, mobilized, and preserved. Radial nerve resection is rarely required. (From Malawer M, Wittig JC. The Tikhoff-Linberg procedure and its modifications. In: Malawer MM, Sugarbaker PH, eds. *Musculoskeletal cancer surgery: treatment of sarcomas and allied diseases*. Dordrecht, Netherlands: Kluwer Academic Publishers, 2001:532.) **(B)** Posterior view showing identification and mobilization of the posterior aspect of the glenoid in which the glenohumeral joint is resected with a Gigli saw (see text). (From Wittig JC, Kellar-Graney KL, Malawer MM, Bickels J, Meller I. Limb-sparing surgery for high-grade sarcomas of the proximal humerus. *Tech Shoulder Elbow Surg*; 2(1):54–69.)

- Before exploring the neurovascular bundle, develop the skin flaps just minimally. If the tumor is found unsuitable for limb-salvage surgery, more-extensive flap dissection would lead to tumor contamination of the skin needed for forequarter amputation.
- In dissecting the neurovascular bundle, pass vessel loops around the neurovascular bundle near the proximal and distal ends of the dissection. Medial traction on the neurovascular bundle allows visualization of the axillary nerve, the posterior circumflex humeral artery, and the anterior circumflex humeral artery. (It is rare to preserve the axillary nerve in large stage IIB sarcomas of the proximal humerus, but if the tumor is small and intraosseous, the nerve can be preserved.) Ligate and divide these three structures.
- If the neurovascular bundle is tumor free, proceed with dissection for a limb-salvage procedure.
- Isolate and preserve the musculocutaneous nerve. It is rarely necessary to sacrifice this nerve to preserve tumor-free margins of resection.
- Divide the deep fascia between the short and long heads of the biceps muscle; this permits easy visualization of the musculocutaneous nerve.
- Identify the radial nerve at the lower border of the latissimus dorsi muscle, where it passes around and behind the humerus in its midportion (spiral groove) into the triceps muscle group.
- Pass a finger around the humerus to move the nerve away from the bone.
- Trace the ulnar nerve down the arm. Divide the intermuscular septum between the biceps and the triceps over the nerve to see it clearly.
- If performing an extraarticular resection, divide the muscle groups anteriorly to expose the neck of the scapula. Separate the short and long heads of the biceps to expose the humerus. Determine the site for the humeral osteotomy, and transect the long head of the biceps and brachialis muscles at this level.
- Identify the inferior border of the latissimus dorsi muscle and make a fascial incision that makes it possible to

pass a finger behind the latissimus dorsi and teres major muscles several centimeters from their insertion.
- Transect the latissimus dorsi and teres major muscles using electrocautery.
- Rotate the humerus externally to expose the subscapularis muscle, which is transected at the level of the coracoid process. Take care not to enter the joint space. Tag the portions of these muscles that will remain with the patient for future reconstruction. Transecting these muscles exposes the anterior portion of the neck of the scapula.
- Now move to the posterior aspect of the patient. Rotate the table, if desired, to provide better visualization.
- Begin the posterior incision anteriorly over the junction of the middle and lateral thirds of the clavicle. Continue down over the lateral third of the scapula past the lower edge of this bone.
- Develop a skin flap by dissecting the skin and subcutaneous tissue between the anterior and posterior incisions from the underlying deltoid muscle down to the level of the midhumerus.
- If removing the entire scapula (type VI resection), make the posterior incision longer and curve it posteriorly to allow the skin flap to expose muscle over the entire scapula.
- Divide the posterior muscle group. Divide the thick fascia that joins the posterior border of the deltoid muscle to the infraspinatus muscle and scapular spine. Leave the deltoid muscle intact to cover the tumor mass.
- Transect the trapezius muscle from its insertions on the scapular spine and acromion.
- Pass the index finger beneath the teres minor up to the area of the planned scapular osteotomy. Transect the supraspinatus, infraspinatus, and teres minor muscles over the neck of the scapula, thus allowing the plane of transection through the neck of the scapula to be exposed. Tag all transected muscles proximally.
- While shielding the radial and ulnar nerves, transect the triceps muscles at the level selected.
- Perform clavicular, scapular, and humeral osteotomies as follows (Fig. 37-25)
  (a) Divide the clavicle at the junction of its middle and inner thirds. This is usually accomplished with a Gigli saw.
  (b) Divide the scapula through its surgical neck medial to the coracoid process, also using a Gigli saw.
  (c) Perform the clavicular and scapular osteotomies before the humeral osteotomy.
  (d) Remove the entire specimen, taking care to protect all the neurovascular structures at each osteotomy site.
- If resecting the entire scapula, take the skin flap back to the medial edge of the scapula. Divide the rhomboid, levator scapula, and trapezius muscles from their insertions on the scapula. It is unnecessary to divide the teres major, teres minor, supraspinatus, infraspinatus, and subscapularis muscles when performing a full scapular resection.
- Transect the humerus 4 to 6 cm distal to the tumor, as determined by preoperative bone scan.
- Obtain frozen sections of tumor margins and touch preparations for cytologic examination of the marrow at the site of the osteotomy.

Proximal Humerus Prostheses

A modular replacement prosthesis is used for large segments of the proximal humerus. The design features of this device are summarized below.

Proximal Humeral Endoprosthesis (MRS, Stryker Orthopedics, Mahwah, NJ)

- Modular components, including stem, body, and humeral head
- Polished intramedullary stems for cement fixation available in multiple diameters and lengths
- Facing reamer to create a perfect seat for the stem–bone interface that protects the stem from bending stresses
- Porous coating (circumferential) at the prosthesis–bone junction for ingrowth of extracortical bone graft and soft tissue to seal the bone–cement–stem interface. Incorporation of extracortical bone graft also protects the prosthetic stem by sharing bending and loading stresses.
- Humeral heads (available in two sizes) with porous coating and metal loops or holes to facilitate muscle and tendon attachment and soft tissue ingrowth

The coracoid and acromion are not recreated on the prosthesis since they serve no function.

TECHNIQUE OF RECONSTRUCTION WITH MODULAR SEGMENTAL PROSTHESIS (MRS)

Endoprosthetic replacement, which has been in use since 1973, is the most common technique for reconstructing large proximal humeral defects (Figs. 37-26 to 37-29). It may be used for both intraarticular and extraarticular defects (i.e., when retaining the glenoid as well as when resecting it with the tumor). Alternatives (although not recommended by the senior author) to reconstructing the defects include allografts, allograft/prosthesis composites, dual fibulas, and vascularized fibulas. Only endoprosthetic replacement will be described in this chapter.

Originally, each patient received a custom-made prosthesis. In 1988, Howmedica (Rutherford, NJ) developed the modular replacement system (MRS), which has since undergone several improvements (see Fig. 37-27). The first MRS prosthesis placed in this location was performed in 1988 in Washington, D.C., for a large stage IIB osteosarcoma of the proximal humerus.

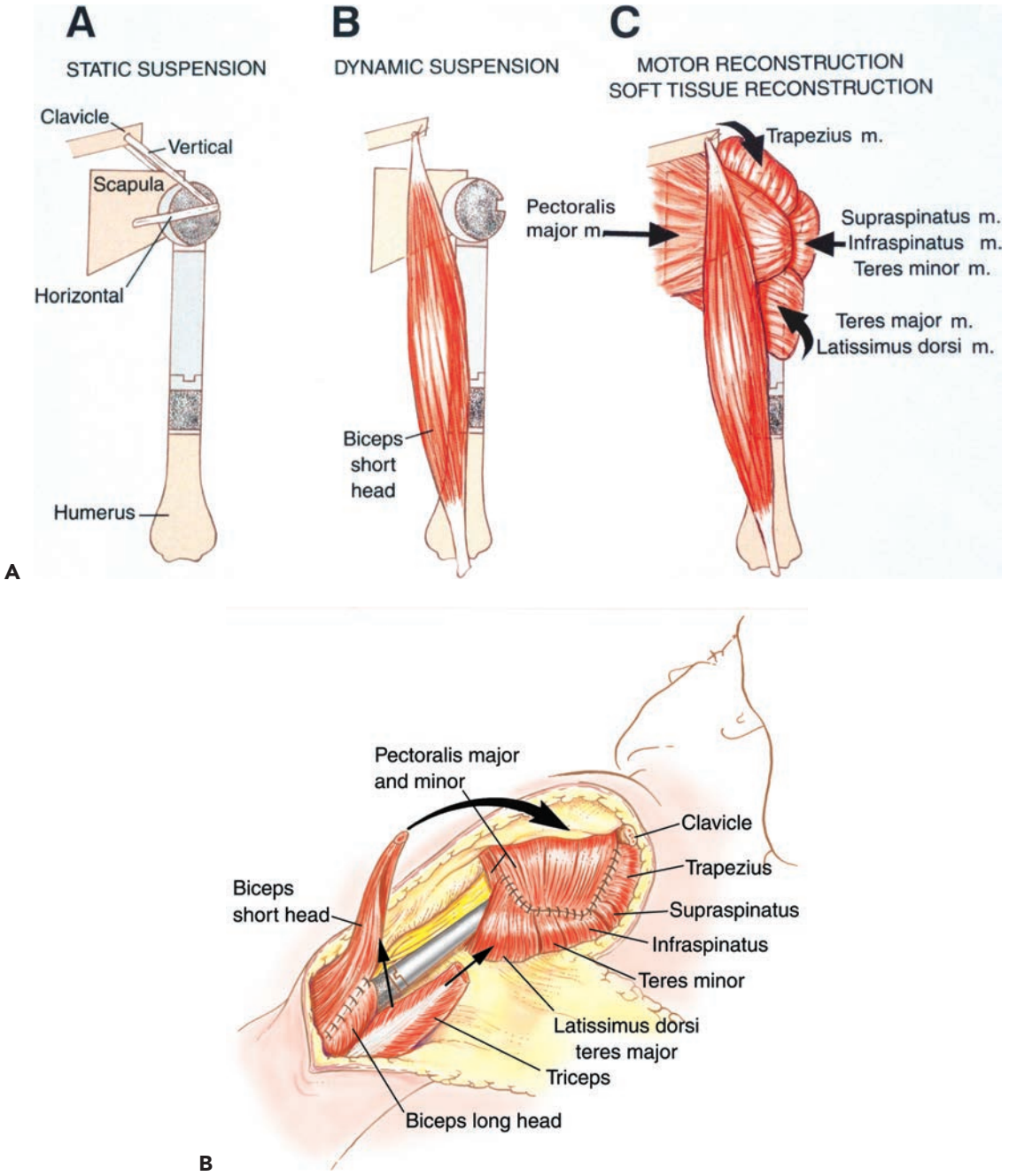


Figure 37-25 Schematic showing the three phases of reconstruction. **(A)** Schematic diagram showing the intraoperative reconstruction of the shoulder girdle musculature. The pectoralis major is sutured to the osteotomized end of the scapula. A prosthesis is placed anterior to the scapula and is covered by the pectoralis major muscle. The remaining muscles are sutured to the pectoralis major, reconstructing the shoulder girdle. The biceps and triceps are tenodesed together and to the muscles. **(B)** The pectoralis major has been sutured to the scapula following prosthetic reconstruction with Dacron tape. The biceps and triceps are then tenodesed to each other to complete the reconstruction. (From Rubert CK, Malawer MM, Kellar KL. Modular endoprosthetic replacement of the proximal humerus. Indications, surgical technique, and results. *Semin Arthroplasty* 1999;10(3):142–153.)

An MRS is used for both intra- and extraarticular reconstructions. The reconstruction is combined with multiple muscle transfers to reconstruct the resected soft tissues. For high-grade bone sarcomas, the deltoid and axillary nerve, along with the glenohumeral joint, are routinely removed (type VB resection). Low-grade tumors are treated with an intraarticular resection and preservation of the abductor mechanism.

Soft tissue reconstruction is essential to cover the prosthesis and create shoulder stability. This is accomplished

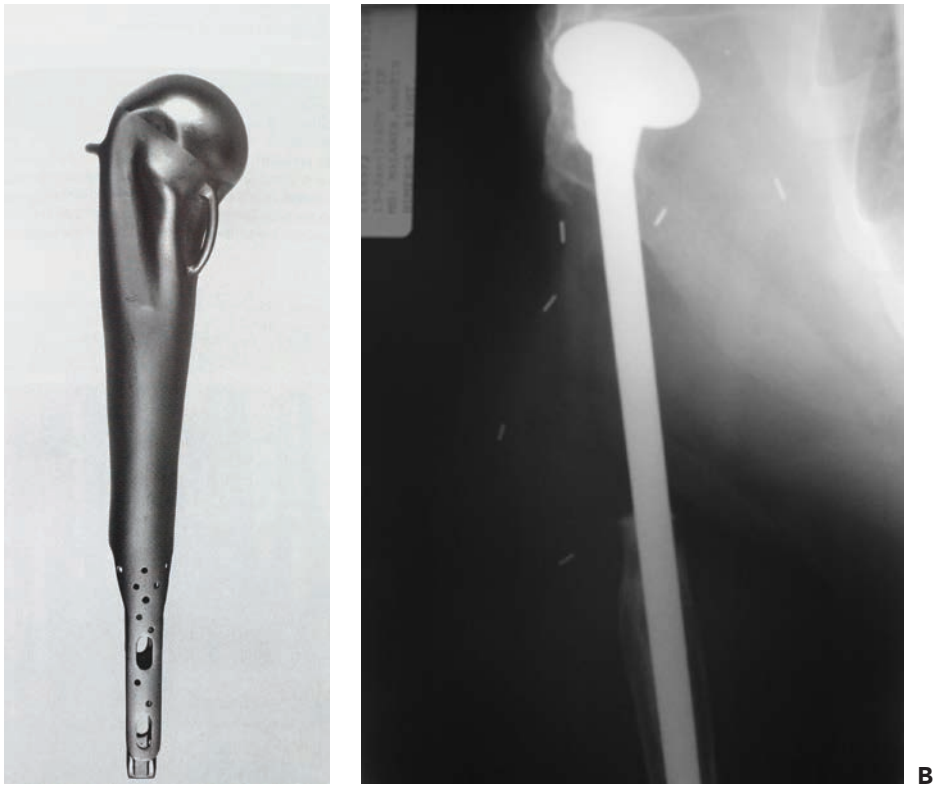


Figure 37-26 Historical prosthetic devices utilized for proximal humeral reconstruction. **(A)** Custom prosthesis with external phalanges for the proximal humerus utilized during the 1960s and 1970s. **(B)** A custom long-stemmed Neer prosthesis utilized by Dr. Ralph Marcove for proximal humerus reconstructions in the 1970s.

through a technique of dual suspension that entails static and dynamic reconstruction. Dacron tape is used to secure the prosthesis horizontally to the scapula and vertically to the clavicle through drill holes. The two sets of Dacron tape provide mediolateral and craniocaudal stability. Dynamic suspension, provided by transfer of the short head of the biceps muscle to the stump of the clavicle, allows elbow flexion. This also restores elbow flexion. Transfer of the trapezius also provides for vertical suspension.

Preservation and transfer of the pectoralis major, trapezius, supraspinatus, infraspinatus, teres minor, teres major, and latissimus muscles provide mobility of the shoulder. These muscle groups offer dynamic support, assist in suspension of the prosthesis, and provide soft tissue coverage, which is essential in preventing skin problems and secondary infection.

Endoprosthetic replacement is highly predictable and successful. There are minimal problems with subluxation following adequate soft tissue reconstruction. Malawer et al.,[3] who have the most extensive experience with replacing the proximal humerus with the MRS, report 95% survival of the prosthesis as determined by Kaplan-Meier analysis at 10 years.

TECHNIQUE OF TOTAL HUMERAL RESECTION

Total humeral resection (i.e., removal of the shoulder and elbow joints) and replacement is an unusual procedure. It is indicated when the tumor involves a large component of the medullary shaft.

Anatomic considerations relative to the proximal humeral component are similar to those previously described. Considerations relating to the midshaft and distal humerus center on the relationship of the tumor to the brachial artery and nerves. Angiography is required to determine the relationship to the brachial vessels medially and the antecubital fossa. MRI and bone scan are used to identify the extent of the humeral involvement, which, in turn, determines whether total humeral resection is required. The entire humerus should be removed in patients who have round cell tumors of the humerus and diaphysis.

The surgical approach is similar to that of a type V resection using multiple muscle transfers and Dacron tape. The resection is similar to that used for lesions of the proximal humerus but continues down to the elbow joint, which is opened anteriorly after mobilizing the brachial vessels and the median nerve through the antecubital fossa (Fig. 37-30).

Resection

- Explore the vessels proximally, release the circumflex vessels proximally, and identify the musculocutaneous, axillary, and radial nerves.
- Mobilize the brachial vessels throughout the length of the arm into the antecubital space to protect them and

Figure 37-27 Modular segmental replacement prostheses. These prostheses consist of three components: a stem of varying diameter, bodies of varying lengths, and humeral heads of various circumferences. Prostheses shown are the trials (holes within the bodies, not to be implanted).

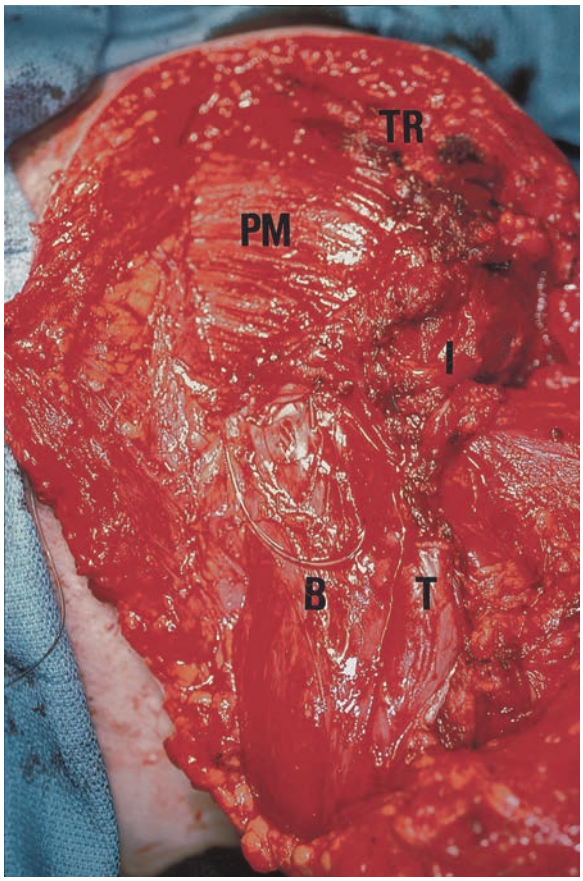


Figure 37-28 Operative photograph showing a completed reconstruction following an extraarticular reconstruction. The prosthesis is covered by muscle. The pectoralis major is the main muscle in the reconstruction and is sutured to the osteotomized scapula. The biceps and triceps are tenodesed to themselves and to the pectoralis major. The remaining musculature of the rotator cuff is tenodesed to the pectoralis major. The trapezius muscle is mobilized and brought down and sutured to the pectoralis major. Intraoperative photograph of completed reconstruction of the proximal humerus. B = biceps; I = infraspinatus; PM = pectoralis major; T = triceps; TR = trapezius.

the accompanying medial nerve. The ulnar nerve passes posteriorly through the intramuscular septum and can easily be identified in the midarm.

- Identify the radial nerve as it passes around the humerus and into the interval between the biceps and brachioradialis muscle, where it becomes the posterior interosseous nerve. Identify and preserve all these nerves, as well as the brachial artery and vein.
- Keep the triceps tendon attached to the olecranon.
- Perform anterior exposure of the elbow joint.
- Explore and identify the brachial vessels and the median nerve.
- Open the capsule of the elbow joint circumferentially; this makes it possible to fit the elbow component and seat it into the olecranon. Avoid a posterior approach to the elbow. Detach the flexor and extensor muscles from their origins on the humeral condyles. Retract the biceps, but do not detach it from its insertion onto the radial tuberosity.

TECHNIQUE OF RECONSTRUCTION WITH TOTAL HUMERAL ENDOPROSTHETIC REPLACEMENT

The senior author recommends total humeral endoprosthetic reconstruction utilizing an MRS with a custom elbow component.

- Use one of the several elbow devices available. An intramedullary stem fixation with PMMA is widely preferred.
- Reattach the forearm flexor and extensor muscles to holes in the prosthesis.
- Transpose the ulnar nerve anteriorly to avoid irritation from the prosthesis. Repair the biceps to the adjacent soft tissue.
- Take care to interpose the capsule between the prosthesis and the neurovascular structures anteriorly.

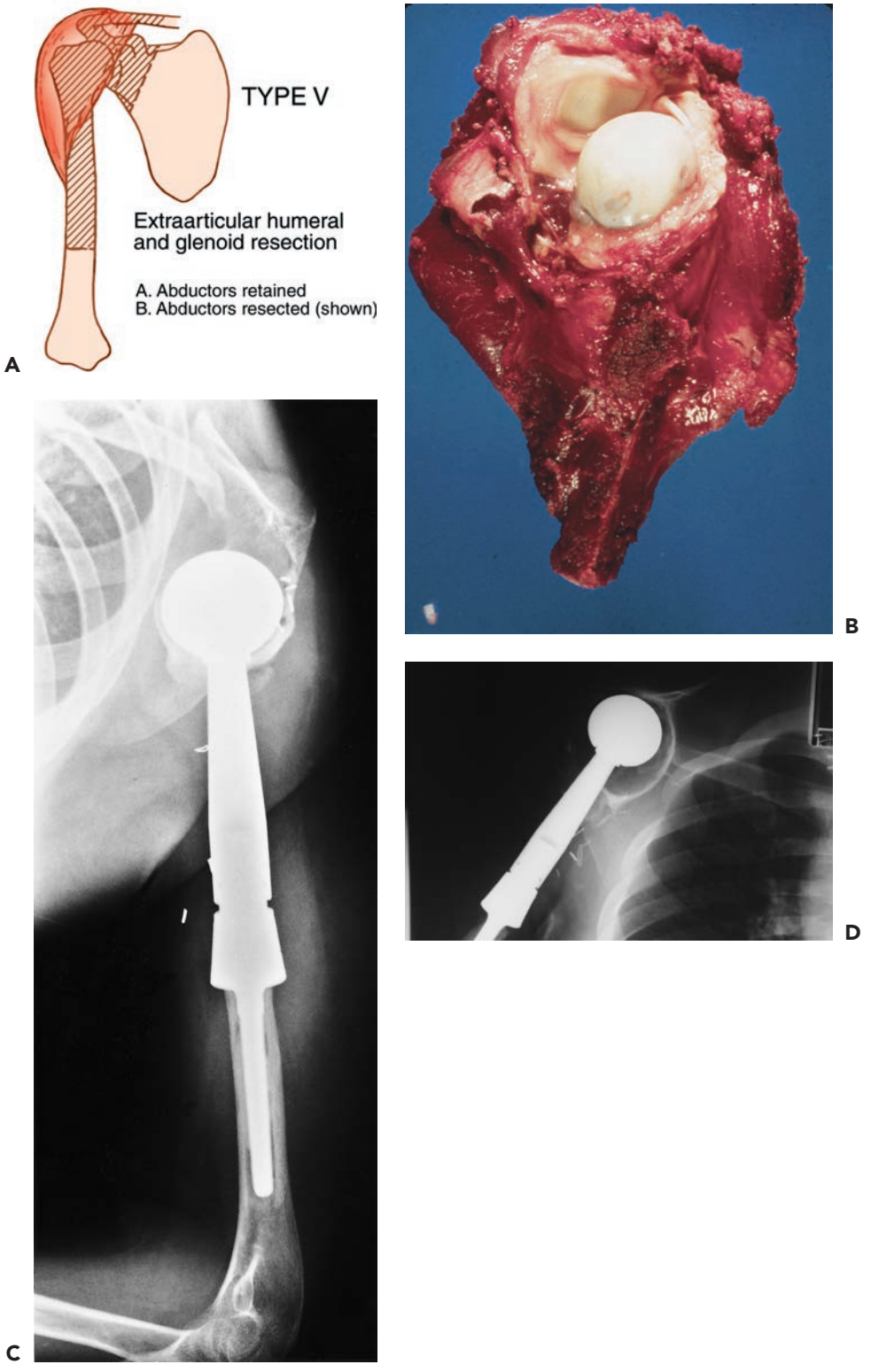


Figure 37-29 **(A)** Schematic of type V resection. This is the most common type of resection for high-grade sarcomas of the proximal humerus. (From Malawer M, Wittig JC. Resections of the shoulder girdle. In: Malawer MM, Sugarbaker PH, eds. *Musculoskeletal cancer surgery: treatment of sarcomas and allied diseases.* Dordrecht, Netherlands: Kluwer Academic Publishers, 2001:186.) **(B)** Gross specimen following an extraarticular resection. Note that the glenohumeral joint has been opened. **(C)** Plain radiograph following an extraarticular resection approximately 5 years postoperatively. Note the new (pseudo) glenoid bone formation around the humeral head. This is a typical finding. **(D)** Plain radiograph following a type VB proximal humeral resection and reconstruction with a modular segmental prosthesis. Note that the prosthesis is placed anterior to the scapula (see reconstructive technique). This is a typical resection for high-grade sarcomas of the proximal humerus. OR Plain radiograph at 5 years follow-up showing metaplastic bone forming a new glenoid. This is a common finding.

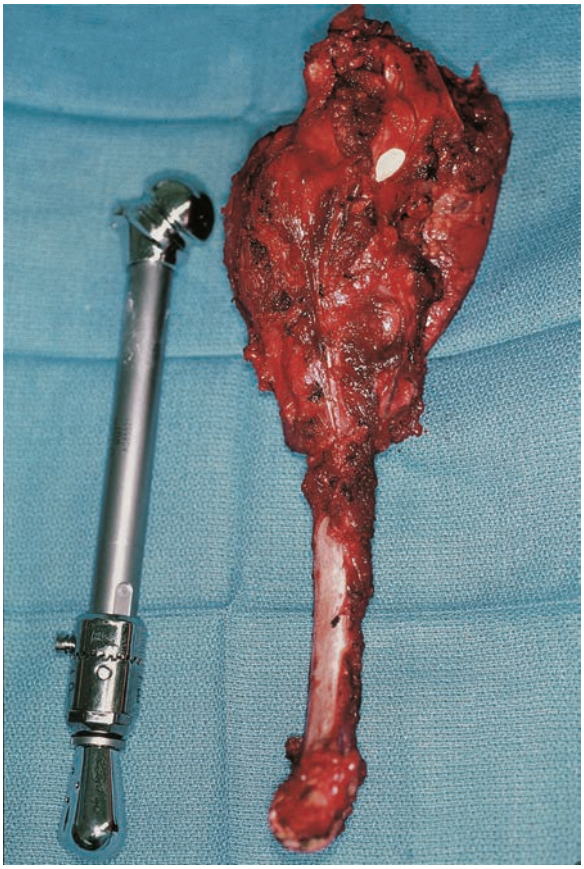


Figure 37-30 Total humeral resection for a large osteosarcoma with intramedullary extension.

Experience with total humeral prostheses is limited, but the duration of these prostheses is reliable. The most critical considerations following total humeral replacement are the potential for arterial thrombosis and occlusion, nerve compression, or neurapraxia.

Rehabilitation

A sling or plaster splint must be worn longer following these procedures than following proximal humeral resections. This is because of the need to allow for soft tissue healing around the elbow and the shoulder girdle. Elevation is required for the first 72 hours. Rehabilitation must focus on both the shoulder and the elbow joints. Fortunately, it is possible to preserve most of the musculature of the shoulder girdle, which allows for an extremely stable shoulder girdle as well as preservation of most of the elbow musculature.

TUMORS OF THE SCAPULA AND PERISCAPULAR AREA

Clinical Characteristics

Tumors of the scapula present with pain, a mass, or both, and they may become quite large before they are brought to the surgeon's attention. Chondrosarcoma is the most common primary malignancy of the scapula. Secondary chondrosarcomas occur from an underlying osteochondroma, but fewer than 2% of osteosarcomas arise from the scapula. In children, the most common malignant scapular tumor is Ewing's sarcoma. Soft tissue sarcomas may involve the suprascapular or the infraspinous musculature and, secondarily, the scapula. Most soft tissue sarcomas of the scapular region occur in adults. In very rare cases, radiation sarcomas of the scapula develop secondary to radiotherapy for breast carcinoma.

Among the unique anatomic considerations associated with this area is that during the early stages of development, a cuff of soft tissue surrounds tumors arising within the scapula. As sarcomas enlarge, they may develop a large axillary component and invade the axillary vessels and brachial plexus. Tumors arising from the neck or glenoid usually involve the periscapular tissue and the glenohumeral joint; this is especially true of chondrosarcomas, osteosarcomas, and Ewing's sarcomas. Important anatomic areas to evaluate for extension are the chest wall, axillary vessels, proximal humeral and periscapular tissues, and rotator cuff. The axillary lymph nodes should be carefully examined, even though they are usually negative. Large suprascapular tumors extend into the anterior and posterior triangles of the neck, making resection difficult or contraindicated, except for palliation.

It is usually possible to satisfactorily treat soft tissue sarcomas arising in the periscapular musculature by removing the adjacent tissue en bloc while preserving the scapula, then following with radiotherapy. Occasionally, a soft tissue sarcoma arising from the deeper structures will involve or encase the scapula, requiring combined scapular resection. If the tumor is distal to the scapular spine, a partial (type IIB) or total (type IIIB) scapulectomy may be adequate. Involvement of the suprascapular musculature or rotators requires an extraarticular resection (type IV).

Specific Tumors of the Scapula

Certain tumors of the scapula and periscapular area require special management (Fig. 37-31). Chondrosarcomas, for example, commonly arise from the scapula; for this reason, any large cartilaginous lesion of the scapula in an adult should be approached with a high index of suspicion. These lesions tend to be low grade and have a large extraosseous component. Cartilage tumors approaching the glenohumeral joint may directly involve the joint space and readily implant on the articular cartilage. In such cases, an extraarticular resection is generally recommended, with no attempt to perform an intraarticular resection. A Tikhoff-Linberg resection (type IV) usually is curative.

Osteosarcomas, of which about 1.5% occur in the scapula, require a limb-sparing resection (type IV) or a forequarter amputation. The limiting factors in performing a limb-sparing procedure are the size and extent of the

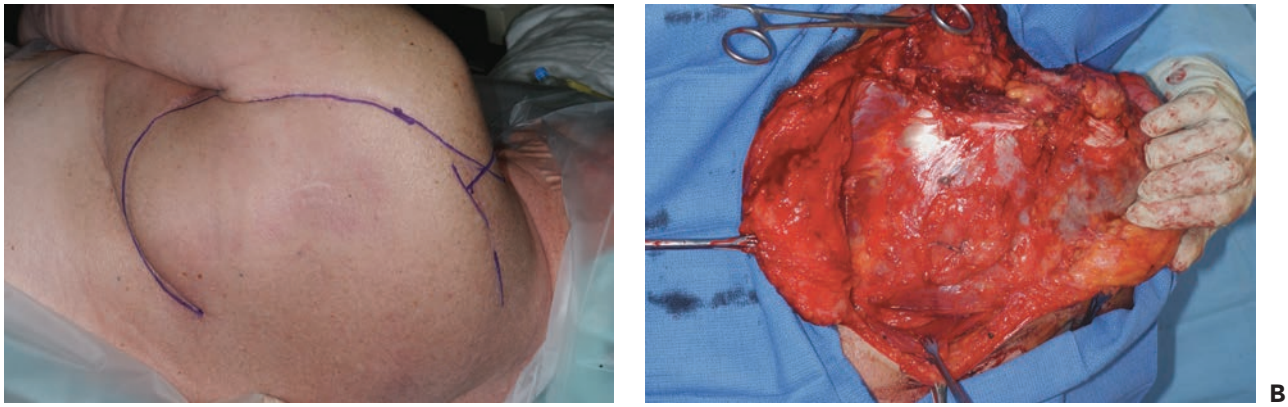


Figure 37-31 **(A)** A large periscapular tumor involving the body of the scapula. A long curvilinear incision is utilized to develop a posterior fasciocutaneous flap to expose the scapula, chest wall, and the supraclavicular area. **(B)** Intraoperative view following fasciocutaneous flap development. A large scapular mass is seen involving the teres and infraspinatus muscles.

extraosseous component. Neurovascular involvement requires a forequarter amputation. Chest wall involvement should be determined before surgery; if present, a partial chest wall resection en bloc with ablation of the primary tumor is necessary.

The traditional treatment for Ewing's sarcoma arising in the scapula has been radiation therapy and chemotherapy, and it has produced excellent functional results. However, the treatment of Ewing's sarcoma is undergoing reevaluation. Recently, total scapulectomy (type IIIA or B), with or without prosthetic replacement, has been recommended in lieu of radiation therapy. Surgery has become increasingly common with the hope of increasing local control, decreasing the morbidity of radiation (especially of late secondary osteosarcomas), and increasing patient survival. The surgery should be planned after induction chemotherapy. Staging should be done in the same manner as in patients with other high-grade sarcomas.

GCTs and ABCs often cause marked ballooning and destruction of the scapula. Small lesions may be treated by intralesional curettage. If the neck of the scapula is not involved, it is possible to perform a partial scapulectomy with minimal loss of function. Large lesions should be treated with total scapulectomy (type IIIA) while preserving most adjacent muscles. Reconstruction involves suspending the scapula from the clavicle by a static and dynamic reconstruction. This is an excellent indication for scapular prostheses, which have recently been developed.

TECHNIQUE OF TIKHOFF-LINBERG (TYPE IVB) RESECTIONS

The Tikhoff-Linberg procedure (extraarticular total scapular proximal humeral resection, type IV) consists of en bloc removal of the scapula, distal clavicle, and proximal humerus and preservation of the arm. The Tikhoff-Linberg procedure was the first true limb-sparing procedure of the upper extremity. The originally description was published in English in 1928 and was performed for periscapular soft tissue sarcomas.[2] Today, the indications for this procedure are low- and high-grade scapular (bony) sarcomas and periscapular and suprascapular soft tissue sarcomas (Fig. 37-32).

Careful preoperative evaluation is imperative. CT and MRI can help determine possible chest wall involvement, and angiography is crucial to determine axillary vessel involvement (Fig. 37-33). Contraindications to the Tikhoff-Linberg procedure are involvement by the tumor of the neurovascular bundle and of the chest wall, both of which require forequarter amputation.

It is important to carefully evaluate the interval between the tumor and vessels; this may require surgical exploration prior to resection. If this interval is clear, the resection may proceed. The surgical team must be prepared to convert from a limb-sparing procedure to a forequarter amputation should the tumor be found to involve the neurovascular bundle. The most medial margin, the paraspinal muscles, and the base of the neck must be explored if there is any possibility of their involvement. It is difficult to evaluate these anatomic areas thoroughly from preoperative studies alone.

Resection includes all the muscles arising from the scapula and inserting on the proximal humerus and an extraarticular excision of the glenohumeral joint. Occasionally, it is possible to preserve the deltoid muscle and the axillary nerve. The deltoid should be preserved whenever possible because it facilitates reconstruction, and soft tissue reconstruction is essential for a stable shoulder.

The surgical guidelines are as follows:

- The utilitarian incision is used, utilizing a combined anterior and posterior approach. The anterior incision is used to explore the axillary vessels, brachial plexus, and axilla.

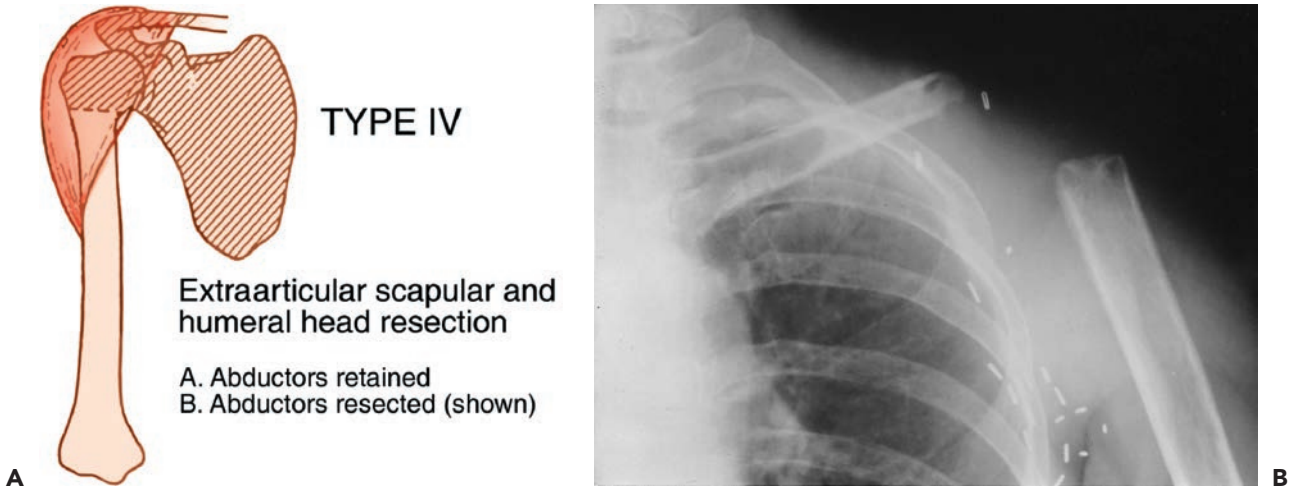


Figure 37-32 **(A)** Type IVB extraarticular scapula and humeral head resection. This is the original type of shoulder-girdle resection as described by Tikhoff and Linberg (see text). The Tikhoff-Linberg resection was the first limb-sparing surgical procedure performed around the shoulder girdle. Resection included the scapula, distal end of the clavicle, and the proximal humerus. Classically, the proximal humerus was suspended from the clavicle by the remaining muscles and/or heavy sutures. (From Malawer M, Wittig JC. Resections of the shoulder girdle. In: Malawer MM, Sugarbaker PH, eds. *Musculoskeletal cancer surgery: treatment of sarcomas and allied diseases.* Dordrecht, Netherlands: Kluwer Academic Publishers, 2001:190.) **(B)** Radiograph following a type IVB resection with soft tissue reconstruction only. Today, we would utilize a scapular prosthesis if the necessary musculature had been retained (see text).

- The pectoralis major muscle is released from the humerus. The pectoralis minor and conjoin tendon are released to permit exposure of the neurovascular structures.
- The glenohumeral joint is exposed anteriorly. The circumflex vessels as well as the axillary nerve are ligated. The joint covered by the subscapularis muscle is not opened.
- The axillary vessels and the brachial plexus are explored and gently retracted anteriorly. The pectoralis major has previously been detached and reflected toward the chest wall for adequate visualization of these structures.
- The posterior portion of the incision allows the release of all muscles that attach the scapula. The rhomboids, trapezius, and levator scapulae muscles are transected. The scapula is then lifted from the chest wall, which permits release of the serratus anterior muscle. It is important to palpate this interval early to determine any chest wall involvement by tumor.
- The glenohumeral joint is removed in an extraarticular manner through the anterior and posterior incisions. The osteotomy is performed below the level of the joint capsule.

To achieve both static and dynamic support, suspension of the proximal humerus is obtained by suturing of the remaining clavicle with Dacron tape (Genzyme Surgical Product Co., Fall River, MA) and muscle transfers. The long and short heads of the biceps and coracobrachialis are sutured through drill holes to the remaining clavicle, and the pectoralis muscle is rotated to cover the defect and to provide stability (Figs. 37-34 and 37-35).

In general, functional results are the same as those following a Tikhoff-Linberg resection (type IVB) and total scapulectomy (type IIIA/B). Patients retain hand function and good elbow function. The shoulder should be stable and no external orthosis should be required. A molded shoulder pad improves cosmesis (Fig. 37-36).

TECHNIQUE OF TOTAL SCAPULECTOMY

Total scapulectomy (intraarticular scapular resection, type III A or B) is indicated primarily for low-grade sarcomas (stage IA/B) of the body of the scapula that involve the suprascapular area, low-grade sarcomas of the glenoid, and soft tissue sarcomas that involve the scapula. Preoperative considerations are similar to those for a Tikhoff-Linberg resection. The neurovascular structures and chest wall must be free of disease. If the tumor extends anteriorly or laterally and involves the rotator cuff or the glenoid, an extraarticular resection (type IVB) should be performed. The skin flaps are similar to those obtained from the posterior limb during a Tikhoff-Linberg resection.

Other guidelines for total scapulectomy are as follows:

- Utilize two incisions: anterior and posterior portions of the utilitarian incision. The anterior incision is used to mobilize the axillary vessels and nerves, especially if there is a large anterior component arising from the scapula. The posterior incision permits exposure of the scapula, rhomboids, latissimus dorsi, and teres muscles.

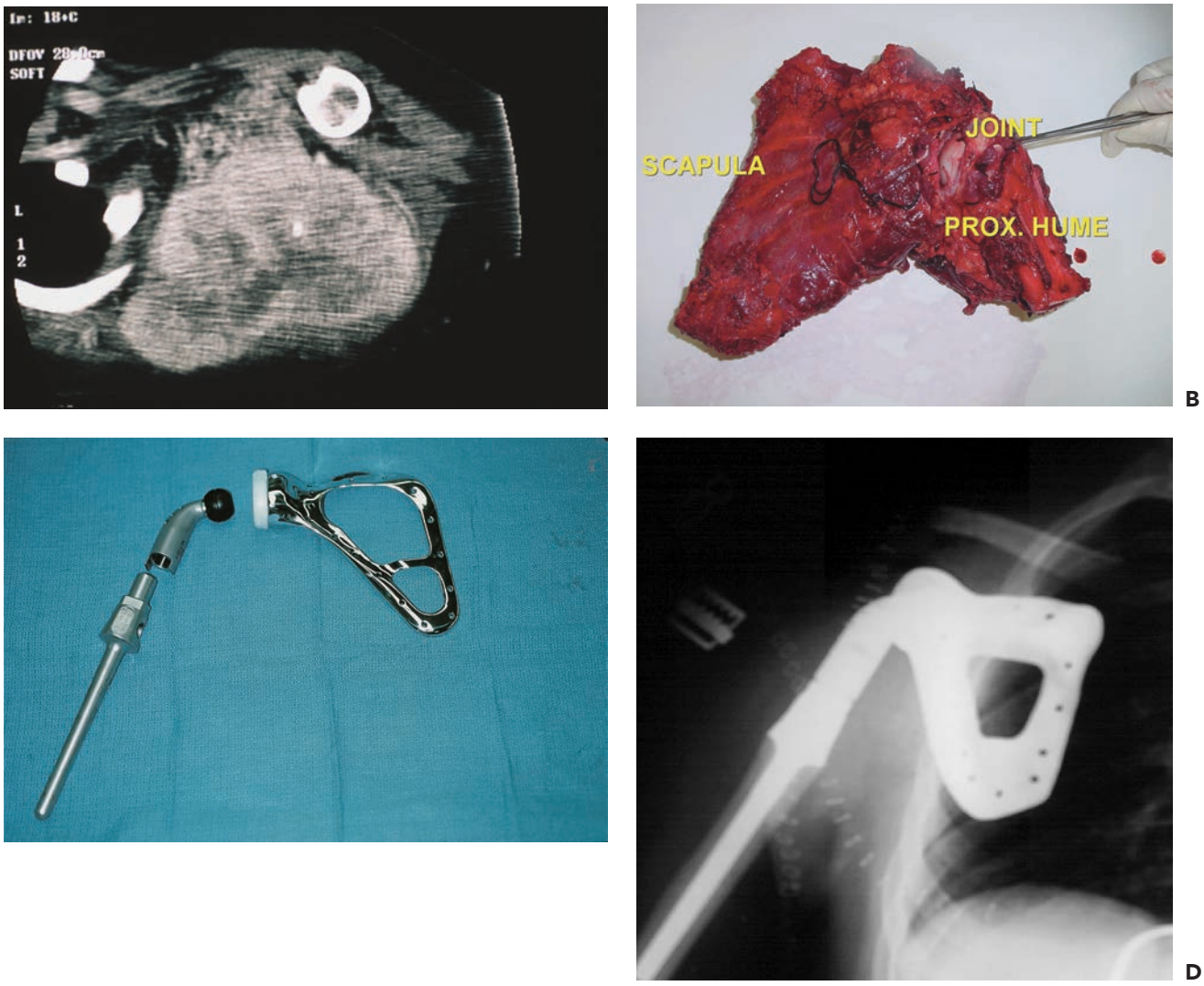


Figure 37-33 **(A)** Computed tomography scan showing a large tumor of the scapula, which is filling the axillary space and involves the proximal humerus. **(B)** Gross specimen following resection of the scapula and proximal one-third of the humerus. This is an extended Tikhoff-Linberg resection (type IVB). **(C,D)** Modular proximal humeral prosthesis and snap fit scapular component (Stryker Orthopedics, Mahwah, NJ).

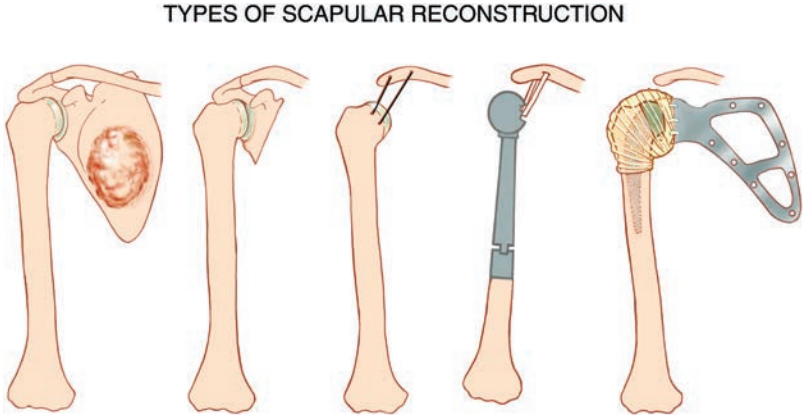
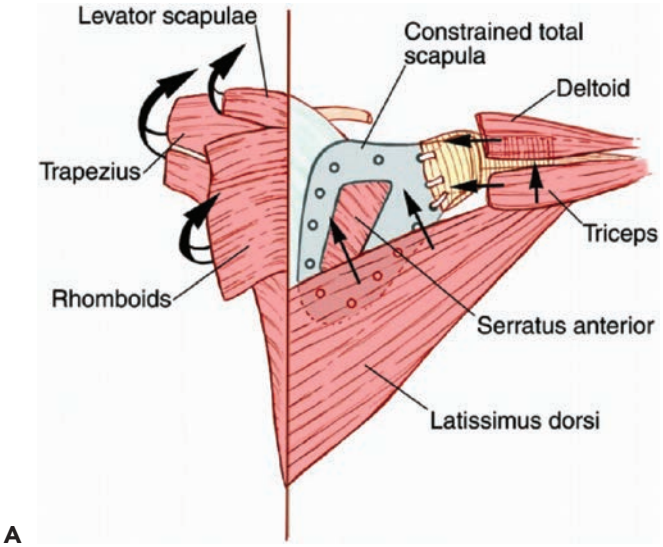


Figure 37-34 Types of scapular reconstruction. The authors prefer the use of a scapular prosthesis if the criteria (see text) can be met in lieu of a flail or hanging shoulder.



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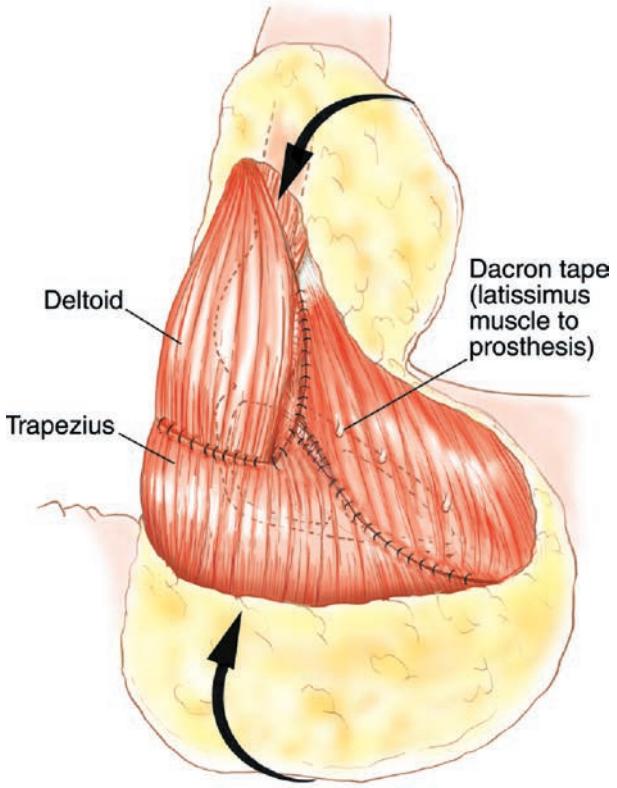

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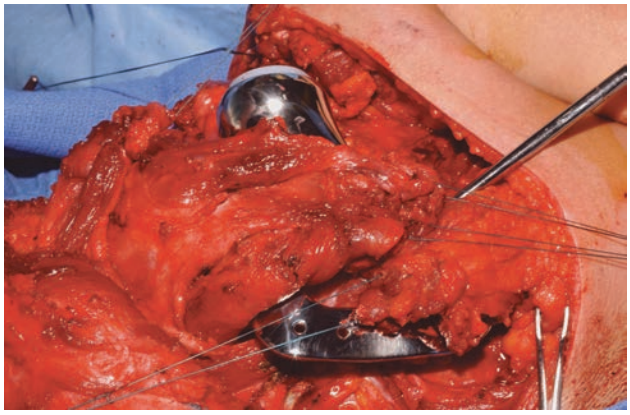

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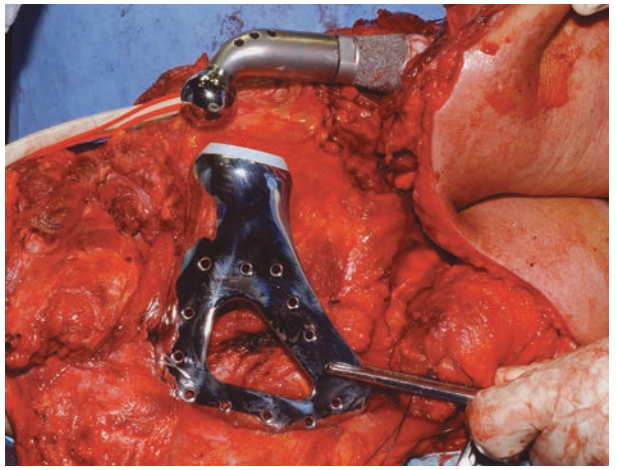

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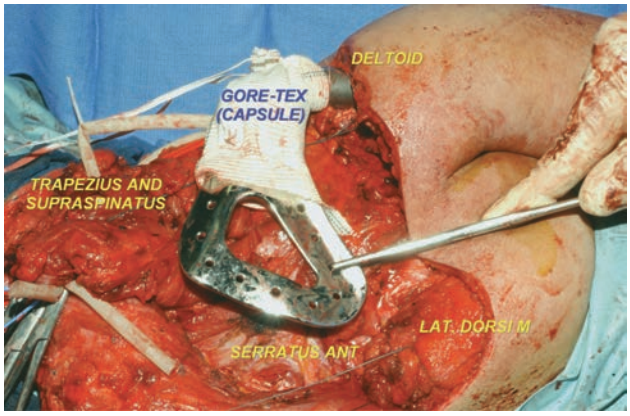

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Figure 37-35 **(A)** Schematic of muscle transfers for a scapular endoprosthesis and proximal humeral component. (From Wittig JC, Bickels J, Wodajo FM, Kellar-Graney KL, Malawer MM. Constrained total scapula reconstruction after after resection of a high-grade sarcoma. *Clin Orthop* 2002;397:143–155.) **(B)** Schematic muscular reconstruction required for shoulder-girdle endoprosthesis. The deltoid and trapezius must be retained to cover the prosthesis superiorly. The inferior portion of the body of the prosthesis is covered by the rhomboids and the latissimus dorsi. In general, the rhomboids and the latissimus muscles are often retained following type IVB resection. The major determinant of the use of a scapular prosthesis is the preservation of the deltoid and trapezius muscles. (From Malawer MM, Wittig JC, Rubert CK. Scapulectomy. In: Malawer MM, Sugarbaker PH, eds. *Musculoskeletal cancer surgery: treatment of sarcomas and allied diseases*. Dordrecht, Netherlands: Kluwer Academic Publishers, 2001:568.) **(C)** Intraoperative photograph of muscle reconstruction over a scapular endoprosthesis (see text). **(D,E)** Intraoperative photographs showing Gore-Tex graft reconstruction.

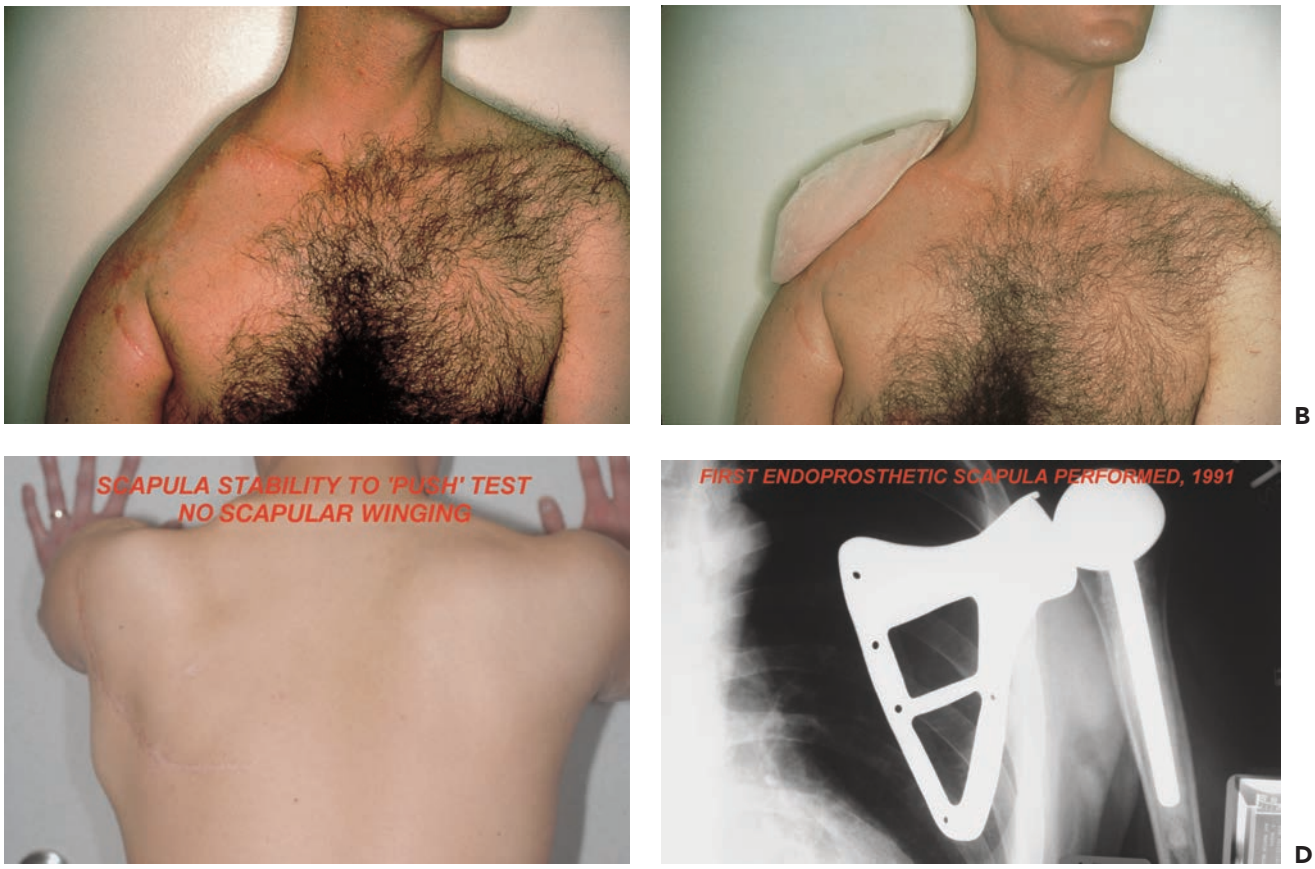


Figure 37-36 Cosmetic appearance following a Tikhoff-Linberg resection. **(A)** Typical clinical appearance. **(B)** Cosmesis is achieved by a contoured shoulder pad. **(C)** Cosmesis following scapular reconstruction with an endoprosthesis. **(D)** Plain radiograph showing a scapula and glenohumeral replacement. Gore-Tex is utilized to recreate the shoulder joint and reattach the humerus to the scapular neck.

- Transect all muscles away from the bone, starting at the lowest point inferiorly.
- Approach the neurovascular structures from the back, as the scapula is retracted away from the chest in a cephalad direction. Take care to avoid injuring the musculocutaneous and axillary nerves near the coracoid and around the subscapularis muscle.
- Be prepared to convert this approach (type III) to a Tikhoff-Linberg resection (type IVB) if the anterior or medial margins are questionable.

Soft tissue reconstruction is mandatory to provide stability and to avoid a flail extremity. Employ a dual-suspension technique using Dacron tape from the clavicle for static support and reattaching the biceps and triceps muscles through drill holes. Tenodesing the deltoid to the pectoralis major and trapezius muscles is essential to provide stability.

The functional results are similar to those with a standard Tikhoff-Linberg resection. If significant soft tissue remains, this defect occasionally can be reconstructed with a total scapula prosthesis. The important muscles for this purpose are the latissimus dorsi, rhomboids, and trapezius.

TECHNIQUE OF PARTIAL SCAPULECTOMY

A partial scapulectomy is indicated for low-grade or benign lesions involving only the body of the scapula. It preserves a cuff of infraspinatus, subscapularis, and serratus anterior muscle. Reconstruction consists of suturing together these muscles to close the dead space and reconstituting the points of origin and insertion of these muscles. A sling is required for 5 to 7 days.

Functional loss after a partial scapular resection (type II) is minimal; in fact, shoulder motion and strength are nearly normal. Total scapular resection (type IIIA/B) causes a significant loss of shoulder motion, but elbow and hand function are normal. The major limitation is the loss of shoulder abduction. Shoulder-girdle function is similar to that following total scapular resection and a Tikhoff-Linberg resection (type VB).

Soft tissue reconstruction is the key to establishing shoulder stability. A compressive arm stocking should be worn immediately after surgery to prevent swelling. The patient should be encouraged to flex the elbow but to

avoid extension until the wound has healed. The patient must wear a sling for 2 to 4 weeks, by which time the transferred muscles provide a stabilizing force to the entire upper extremity. Forward and backward flexion of approximately 30 to 45 degrees is obtained. The goal of rehabilitation is to strengthen the transferred pectoralis major, latissimus dorsi, and trapezius muscles around the shoulder as well as the elbow flexors. A shoulder pad contributes to cosmesis and restores symmetry.

TECHNIQUE OF SCAPULA ENDOPROSTHETIC RECONSTRUCTION

Experience with total scapular replacement, although still limited, is increasing. If most of the musculature is retained (type IIIA), it usually is possible to reconstruct the defect with a custom scapular prosthesis. The most common indications for this procedure are large (stage III) GCTs, low-grade chondrosarcomas, and Ewing's sarcomas following induction chemotherapy. Successful reconstruction poses three primary challenges: (a) replacing the humeral joint, (b) stabilizing the scapula prosthesis within the humeral component (i.e., creating a new glenohumeral joint), and (c) providing soft tissue attachments to both the scapular and humeral components to ensure stability as well as active motion.

Types of Prostheses (Fig. 37-37)

A custom prosthesis is utilized for reconstruction of the scapula. The design features of these prostheses are summarized below:

- Nonconstrained or semiconstrained design (first- and second-generation prostheses)
- Holes along the periphery of the prosthetic scapular body for reattachment of the scapular stabilizing muscles (levator scapulae, rhomboids, and trapezius)
- Holes along the base of the prosthetic scapular neck for capsular reconstruction with Gore-Tex graft
- The body of the scapula is open to permit adjacent muscle tenodesis.
- No attempt is made to recreate the coracoid, acromion, or scapula spine. These structures would create wound complications and closure difficulties.

Technique of Constrained Total Scapula Endoprosthesis Reconstruction

The technique, surgical anatomy, and indications and results of the constrained scapula prosthesis are as follows:

- Tenodese the scapula prosthesis to the remaining trapezius, rhomboids, and latissimus dorsi muscles.
- Place the prosthesis in a pocket between the rhomboids and serratus anterior.
- Use Dacron tapes to tenodese the prosthesis to these muscles. Tenodese the trapezius to the prosthesis and to the deltoid muscle.
- Rotate the latissimus dorsi to cover the prosthesis in entirety with soft tissues.
- Suture a Gore-Tex graft over the proximal humeral component before snapping it into the glenoid for additional stability.
- Reconstruct the glenohumeral joint by sewing an aorta Gore-Tex (W.L. Gore & Associates, Flagstaff, AZ) graft over the scapula neck and the proximal humerus. This

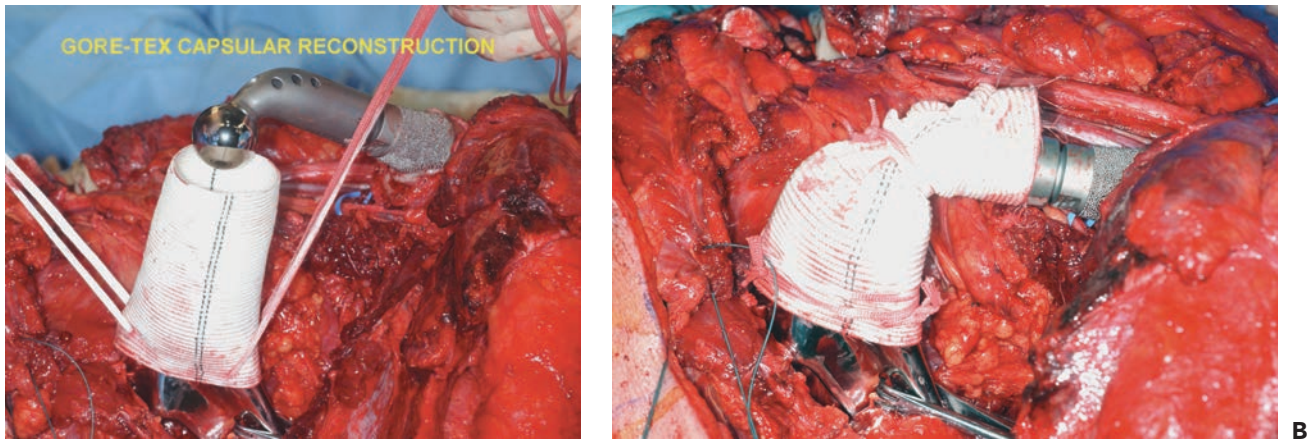


Figure 37-37 Gore-Tex reconstruction in conjunction with a bipolar snap fit head. **(A)** Reconstruction with Gore-Tex graft of a new capsule. Gore-Tex is initially sewn to the neck of the scapula. **(B)** Prosthesis is reduced into the glenoid. Note that the Gore-Tex graft has been placed proximally around the glenoid and scapular component. The Gore-Tex is brought over the proximal humeral component and sutured into place with 3-mm Dacron tape. Completion of the capsular reconstruction is achieved with the Gore-Tex graft. The rhomboid muscles and latissimus dorsi are then closed over the body of the prosthesis.

permits stabilization of the joint and provides additional stability.
- Reattach and tenodese the remaining rhomboids, latissimus dorsi, and teres muscle to the prosthesis and to themselves to achieve reliable and functional soft tissue reconstruction.
- Advance the preserved deltoid proximally and suture it to the trapezius; then close the pectoralis major muscle anteriorly over the new joint.

Wittig et al.[4] reported no infections or local recurrence and superior cosmesis when compared to the traditional Tikhoff-Linberg procedure. It was emphasized that it is essential to retain the deltoid, trapezius muscles, and axillary nerve. All patients had a stable shoulder and good to excellent hand and elbow function, thus providing a functional extremity. Most patients had good forward flexion, abduction, and external and internal rotation. Elevation, protraction, and retraction of the shoulder were preserved.

Wodajo et al.[5] reported a retrospective comparison of patients undergoing scapular resection and reconstruction with and without an endoprosthesis. Patients with endoprosthetic reconstruction had higher Musculoskeletal Tumor Society (MSTS) scores than did patients with no endoprosthesis (86% and 62%, respectively). The former group also had a larger arc of abduction than the latter (60% to 90% and 10% to 20%, respectively) and improved cosmesis. Most bony sarcomas of the scapula are contained by the two surrounding muscles, the infraspinatus posteriorly, and the subscapularis muscle anteriorly and do not involve the deltoid and trapezius muscles. Therefore, most tumors of the scapula, irrespective of size, are amenable to endoprosthetic replacement.

AXILLARY TUMORS

Overview

Several types of malignant tumors may involve the axillary space and may require surgical resection. Primary sarcomas occur within the muscles (i.e., the pectoralis major, latissimus dorsi, teres major, and subscapularis) that make up the borders of the axillary space and rarely within the axillary fat itself. Large metastatic deposits to the regional lymph nodes occasionally create large, matted masses that may require resection. The most common are metastatic melanoma and recurrent breast carcinoma. In addition, certain primary tumors arise from the brachial plexus, either the nerves or the vessels. These include leiomyosarcomas of the axillary vein and neurofibrosarcomas of the adjacent nerves. Secondary extraosseous extension from large tumors of the proximal humerus or scapula often goes into the axillary space and require imaging evaluation and surgical resection. Tumor involvement of the brachial plexus and the major vessels is the main determinant of resectability (Fig. 37-38).

The key to adequate and safe surgical resection of axillary tumors is the complete visualization and mobilization of the infraclavicular portion of the brachial plexus (i.e., the axillary artery and vein and the cords that surround them). Multiple imaging studies are required, but the final decision to proceed with a limb-sparing surgery is made during intraoperative exploration of the axillary space. Gross tumor involvement of the brachial plexus and/or the major vessels is an indication for amputation or for abandoning the attempt for resection. Contraindications to axillary space resection include involvement of the neurovascular bundle and of the adjacent chest wall.

The technique of axillary space exploration via the transpectoralis approach, as described here, has been developed by the authors and has been found to be the most useful in this determination and in the surgical approach.

Unique Anatomic Considerations

Anatomic Borders

The axilla is a pyramid-shaped space between the chest wall and the arm. The apex of the pyramid is formed by the junction of the clavicle and the first rib. The superior border is determined by the scapula. This apex is approximately 1 to 2 cm medial to the coracoid process. The anterior wall of the axilla is formed by the pectoralis major muscle, and the posterior wall is formed by the subscapularis, the teres major, and the latissimus dorsi muscles. The chest wall and the serratus anterior muscle form the medial wall of the triangle. The humeral shaft is covered by the muscle fibers of the coracobrachialis, and the short head of the biceps defines the lateral wall. The axilla is triangular shaped from both the coronal and axial views.

The most significant structures of the axillary space are the axillary artery and vein, which are surrounded by the cords of the brachial plexus as they enter from the apex and pass through the axillary space medial to the coracoid to the medial aspect along the humeral shaft. This space is filled with a fair amount of fat and lymph nodes that follow the axillary vessels. The space is bounded anteriorly by the deep clavicular pectoralis fascia that arises from the clavicle and covers the deep fat below the pectoralis major muscle. Inferiorly, the fascia wraps around the base of the axilla. Identification of this layer is extremely important prior to entering the deeper structures.

Pectoralis Major and Conjoined Tendon Muscles

Two major muscles form the gateway to the axillary space. Lying just below the clavicular-pectoralis fascia, they are the pectoralis minor muscle, which arises from the chest wall and attaches to the coracoid, and the conjoined tendon, which arises from the coracobrachialis and short

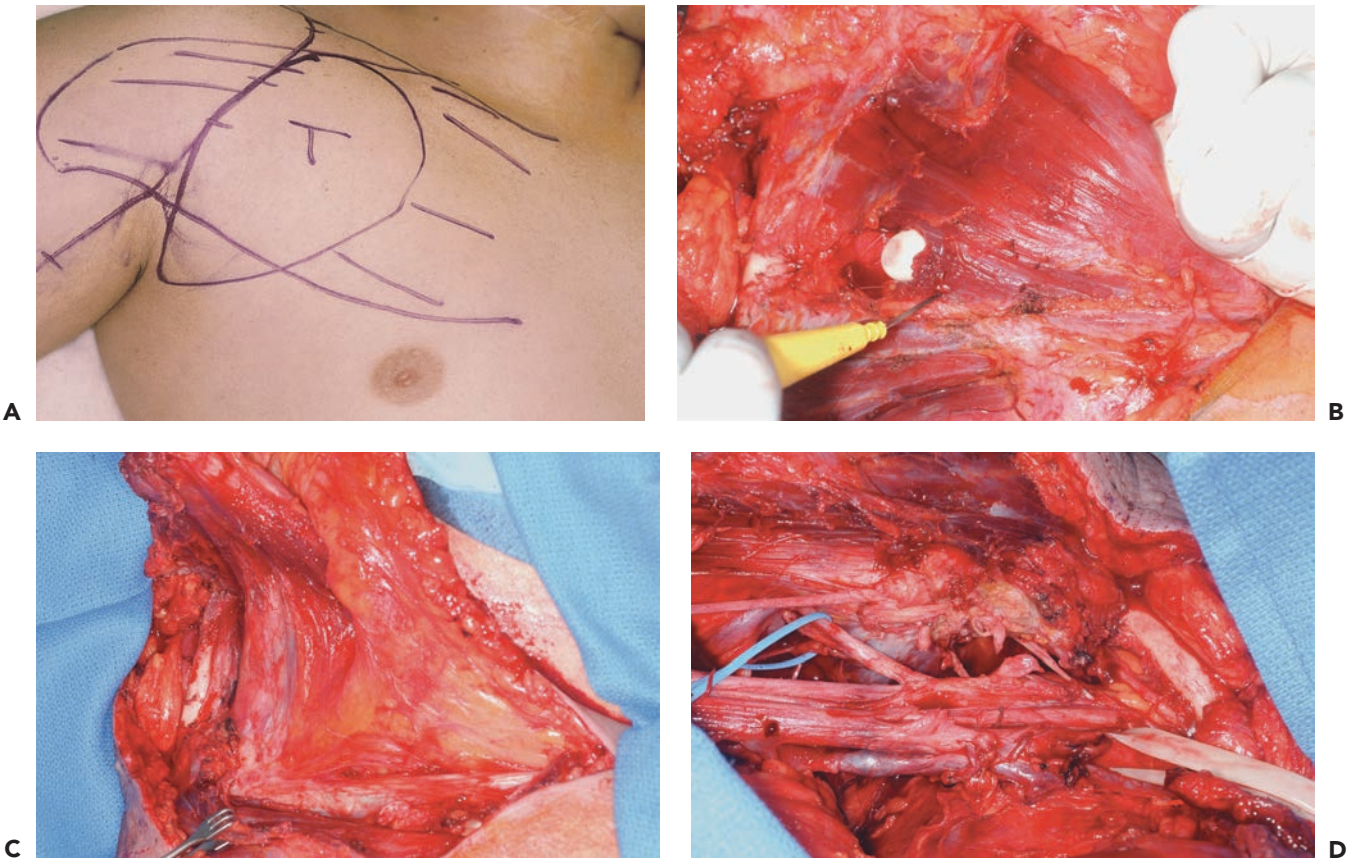


Figure 37-38 Axillary tumor. **(A)** Clinical photograph of a large axillary tumor and its relationship to the pectoralis major muscle. The initial step in the axillary exposure is release of the pectoralis major muscle. **(B)** Intraoperative view of release of the pectoralis major muscle, which provides access to the axilla. The pectoralis is released from the clavicle and its humeral insertion. Care is taken to protect the underlying vessels, which are often displaced anteriorly by a large axillary tumor. **(C)** The second layer of muscles and the axillary fascia are seen once the pectoralis major muscle is detached and rotated toward the chest wall. The second layer of muscle consists of the coracobrachialis and pectoralis minor by inserting on the coracoid. Surgically, both of these muscles are released prior to opening the axillary fascia. **(D)** Completion of exposure in the infraclavicular portion of the brachial plexus. The axillary nerve is within the vessel loop.

head of the biceps from the medial aspect of the humerus. These two muscles must be identified in the clavicular-pectoralis fascia during the surgery. Their identification is the key to accurate identification and dissection of all structures that are located deeper within the axillary fat.

Infraclavicular Brachial Plexus (see Fig. 37-38)

The infraclavicular portion of the brachial plexus is the most significant anatomic component; therefore, it must be thoroughly evaluated and its anatomy completely understood. The axillary artery and vein are contained within a single sheath and are surrounded by the cords of the infraclavicular plexus. The lateral, posterior, and medial cords of the plexus are found at the level of the pectoralis minor muscle. These cords occur in the sheath around the axillary artery and vein. At the lower border of the pectoralis minor muscle, these cords give rise to the five major nerves of the extremity: median, ulnar, radial, musculocutaneous, and axillary nerves.

The lateral cord gives rise to the musculocutaneous nerve, which travels along the medial aspect of the conjoined tendon, moves into the muscle belly of the coracobrachialis, and then enters the short head of the biceps. This nerve is the first to be identified during the exploration. It is located in the superficial axillary fat inferior to the coracoid process. The posterior cord gives rise to the axillary nerve, which travels deep in the space and passes inferior to the glenohumeral joint and the subscapularis muscle. The axillary nerve innervates the deltoid muscle. The main portion of the posterior cord becomes the radial nerve, which travels posterior to the sheath and exits the axillary space along with the axillary sheath. The medial cord gives rise to the median nerve, which is found on the lateral aspect of the sheath and exits the inferior aspect of the axillary space along the sheath. The ulnar nerve arises from the median cord and travels along the most

medial aspect of the sheath and exits distally, along with the sheath. The ulnar nerve is the most common nerve to be involved by tumors arising inferior to the brachial plexus because of its medial position along the sheath. This nerve often displays the first symptom (i.e., weakness or neuropathic complaints) of brachial plexus involvement.

Axillary and Brachial Arteries

The axillary artery is a continuation of the subclavian artery as it passes below the clavicle and the first rib. As it exits the axillary space just distal to the take-off of the circumflex vessels, it is termed the brachial artery. This transition occurs anteriorly at the level of the inferior pectoralis major and teres major muscles. The axillary artery consists of three segments: (a) the portion between the clavicle and pectoralis minor, (b) the area under the pectoralis minor, and (c) the segment between the inferior lateral pectoralis minor border to the point of exit below the teres major muscle. Tumor involvement may occur secondary to lymph node metastases in any of these three locations. The most common sites for axillary sarcomas are the second and third segments. Metastatic carcinomas involving the axillary space can involve any of these areas; however, they most often present as large, matted tumor masses between areas two and three.

Radiographic Evaluation

Three-dimensional imaging of the axillary space is important for accurate tumor localization and surgical planning. CT, MRI, angiography, and three-phase bone scans are used in the same way as in other anatomic sites. In addition, venography of the axillary and brachial veins is essential to the evaluation of tumors of the axilla and brachial plexus.

Computed Tomography

CT is most useful in evaluating the bony walls of the axilla, specifically, the humerus, glenohumeral joint, and scapula. Soft tissue tumors are well defined by CT scans. CT scans with intravenous contrast will aid in the definition of the axillary vessels.

Magnetic Resonance Imaging

MRI is extremely useful in determining the extent of a soft tissue mass in the axillary space and the involvement of the underlying serratus anterior and/or the anterior and posterior walls of the axillary space (pectoralis major, subscapularis, latissimus dorsi, and teres major muscles).

Angiography

Angiography should be part of the evaluation of all tumors of the axillary space. This technique will demonstrate any vascular displacement (very often inferior or anterior) and vascular anomalies of the axillary vessels. In addition, it can provide useful information on any response to induction chemotherapy (e.g., decrease in tumor vascularity).

Venography

Venography is one of the most accurate means of determining brachial plexus and axillary sheath involvement or infiltration by tumor. The arterial wall is thick and almost never shows signs of occlusion, whereas the axillary vein is a thin-walled structure that is easily compressed and infiltrated by tumor. Therefore, occlusion is almost synonymous with vascular sheath and brachial plexus involvement. A venogram should be routinely performed to evaluate the axillary vein. A positive venogram showing occlusion of the vein, in combination with neurologic pain and weakness, is almost always pathognomonic of axillary sheath and brachial plexus involvement by tumor. The triad of axillary vein occlusion, distal motor weakness, and neuropathic pain is a very reliable predictor of tumor infiltration of the brachial plexus sheath.

Biopsy

Biopsy of axillary tumors should be performed utilizing a needle or fine needle aspiration (FNA) technique. If a metastatic lesion is most likely, then FNA is the more appropriate means of identifying carcinoma cells. If a sarcoma is suspected, a needle or core biopsy should be performed. The biopsy site should be inferior through the base of the axillary space and not through the pectoralis major muscle or near the vascular sheath. This can easily be performed under CT guidance. The biopsy site must be removed in its entirety during resection of the tumor. Deep-seated lesions near the chest wall should be approached in this manner. Anterior lesions occasionally may be approached through the lower portion of the pectoralis major.

Surgical Management

Guidelines

1. Use of an anterior utilitarian incision with axillary extension. This incision extends along the deltopectoral interval with preservation of the cephalic vein. It then curves inferiorly and distally over the base of the axilla.
2. Detachment of the pectoralis major muscle. This muscle is detached from its insertion on the humerus and is reflected toward the chest wall while maintaining its vascular pedicles. This permits exposure of the entire axillary space and fascial sheaths.
3. Development of an anterior axillary fascial plane (clavicopectoralis fascia). This thick layer of fascia contains

the entire axillary space and structures. It is extremely well defined. This plane must be developed prior to any further dissection.

4. Release of the pectoralis minor and conjoin tendon. The pectoralis minor and conjoin tendon form the anterior muscle layer within the axillary space. Release of these muscles is key to exposure of the vascular sheath, the brachial plexus, and the numerous vascular branches feeding any large tumors.
5. Initial identification of the musculocutaneous and axillary nerves. The musculocutaneous nerve comes around the lower border of the coracoid under the pectoralis minor muscle. The axillary nerve comes off deeper from the posterior cord and travels toward the shoulder joint.
6. Mobilization of the axillary sheath and brachial plexus. Proximal and distal control of the vascular sheath is obtained prior to tumor dissection. Once the deep fascia is opened and the pectoralis minor muscle is released, the sheath is found very easily by palpating the axillary fat. Vessel loops are placed around the entire sheath; there is no need to dissect the individual components.
7. Resection of tumor. All the feeding branches entering into the mass are serially ligated and transected. Axillary fat is left around the tumor mass as the only true margin.
8. Closure. The pectoralis minor and conjoin tendon are reattached to the coracoid process.
9. Insertion of catheter. An epineural catheter is placed in the axillary sheath for postoperative pain relief.
10. Closure of the empty space. Often following resection, there is a large empty space that is prone to collect fluid and may lead to wound complications and dehiscence. The latissimus dorsi may be released from its insertion onto the humerus and inserted into the defect and sutured to the subscapularis muscle.
11. Suspension and adduction. The arm is suspended and kept adducted at the side of the body to close off this space. Multiple drains are used for 4 to 7 days.

Surgical Technique

- Place the patient in a supine semilateral position. Prepare and drape the arm, shoulder girdle, and chest. Full mobilization of the ipsilateral extremity is essential.
- Use a deltopectoral incision. This incision starts over the junction of the inner and middle thirds of the clavicle, continues along the deltopectoral groove, and curves distally over the anterior axillary fold (inferior border of the pectoralis major muscle).
- Open the superficial fascia, ligate or preserve the cephalic vein, and raise medial and lateral fasciocutaneous flaps.
- Detach the pectoralis major muscle from its insertion to the proximal humerus, leaving at least 1 cm of the tendon for reattachment. Take care to protect the axillary vessels. Large axillary tumors may displace the axillary sheath anteriorly and adjacent to the pectoralis major muscle.
- Divide the short head of the biceps and coracobrachialis (conjoined tendon) and pectoralis minor muscles at their insertion on the coracoid process. Perform the reflection with caution to prevent traction injury to the musculocutaneous nerve, which pierces the substance of the coracobrachialis muscle.
- Detach the pectoralis minor and conjoined tendon. Expose the axillary cavity after detachment and reflection of the second layer of muscles. The coracoid insertion of the pectoralis minor and the coracobrachialis are detached and retracted. Reflect the pectoralis minor muscle medially and the conjoined tendon (coracobrachialis and biceps muscles) caudally. Tag all edges of reflected muscles with a suture for later identification and use in reconstruction.
- The deep axillary fascia, neurovascular bundle, and content of the axillary cavity are now fully exposed. The anatomic relation of the tumor to the neurovascular bundle can be determined and the decision regarding tumor resectability made. At this anatomic site, the artery, vein, and brachial plexus are in close relation, and tumor extension to the neurovascular bundle usually affects all its components and negates resection. Benign tumors and soft tissue sarcomas usually push the adjacent neurovascular bundle; only at a later stage do soft tissue sarcomas break into it. Metastatic carcinomas directly invade the surrounding tissues, irrespective of compartmental borders. For these reasons, resection of large metastases with preservation of the neurovascular bundle is occasionally not feasible. If resection is not feasible, dissect the neurovascular bundle off of the tumor mass, ligate the subscapular and thoracodorsal vessels, and perform hemostasis.
- Resect the tumor with wide margins.
- Reattach the conjoined tendon and pectoralis minor muscles.
- Reattach the pectoralis minor and conjoined tendons to the coracoid process with a nonabsorbable suture. Reattach the pectoralis major to its insertion site on the proximal humerus in the same manner.
- Close the wound over suction drains.

Following surgery, the upper extremity is kept in an arm sling. Continuous suction is required for 4 to 7 days. Perioperative intravenous antibiotics are continued until the drainage tubes are removed. Postoperative mobilization with gradual range of motion of the shoulder joint is then introduced.

CLAVICLE TUMORS

Sarcomas arising from the clavicle are exceedingly rare. ABCs are also rare but are one of the more common neoplasms to arise from the clavicle. Metastatic carcinomas

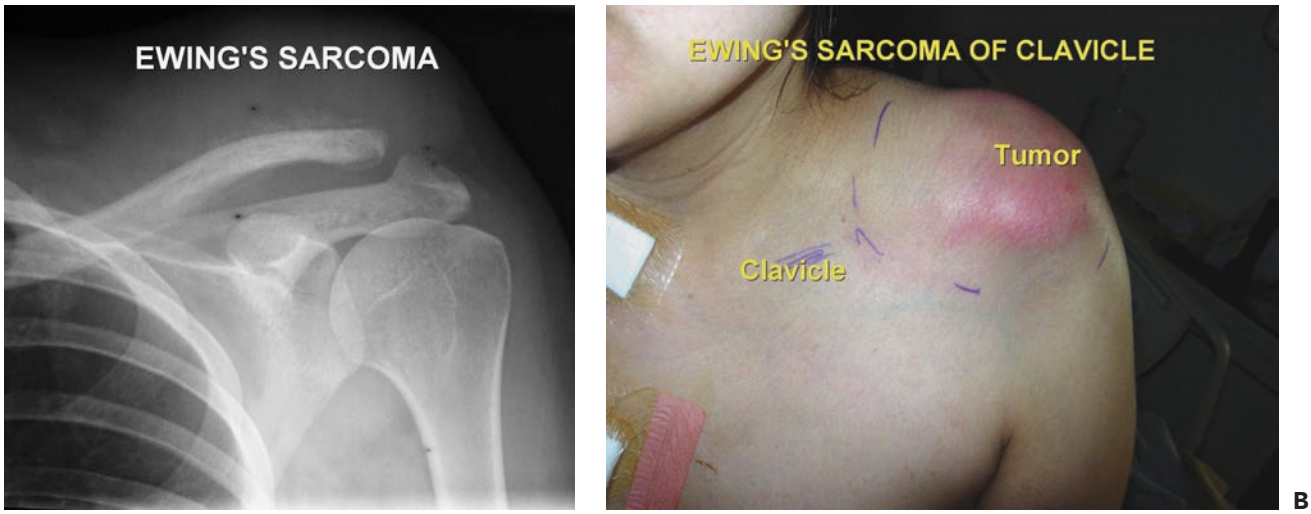


Figure 37-39 **(A)** Anteroposterior radiograph showing sclerosis and destruction of the distal clavicle as well as a large soft tissue mass with no evidence of matrix formation. **(B)** Clinical view of a large Ewing's sarcoma of the clavicle showing a large extraosseous soft tissue mass.

can affect the clavicle. Most can be treated with radiation, although surgical resection may occasionally be indicated for metastatic carcinomas that present with a large soft tissue component that encroaches on the brachial plexus. There is no replacement following radical resection of the clavicle. Most patients will be pain free and have full shoulder motion. The most common postoperative complaint is fatigue of the trapezius muscle (Fig. 37-39).

The key to safe resection of the clavicle is proper dissection and mobilization of the subclavian, axillary vessels and brachial plexus away from the tumor. Proximal exploration and mobilization occur at the base of the neck; distally, the axillary vessels and brachial plexus are explored deep to the pectoralis major muscle and medial to the coracoid process (similar to the axillary approach). Once the pertinent neurovascular structures have been separated from the neoplasm, the clavicle can be resected. A modification of the utilitarian shoulder-girdle incision is used.

The resection proceeds as follows:

1. Extend the incision from the midsternocleidomastoid muscle past the sternoclavicular joint. It extends trans-

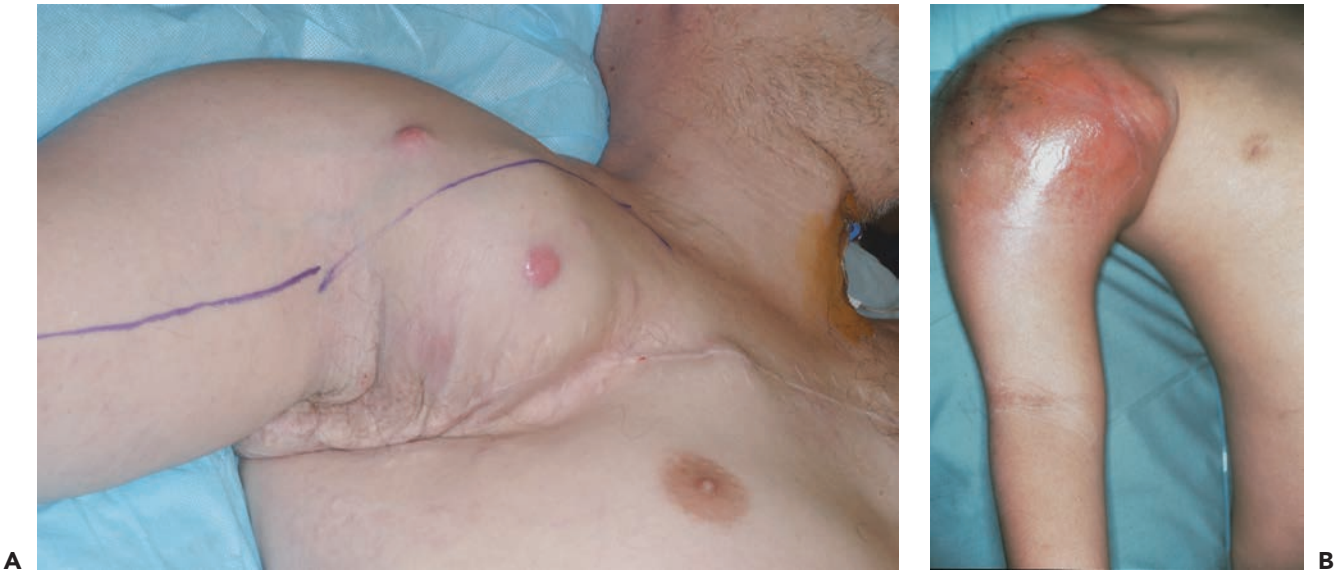


Figure 37-40 **(A)** Large recurrent chondrosarcoma of the shoulder girdle. **(B)** Large telangiectatic osteosarcoma of the proximal humerus. Both patients required forequarter amputation. Although amputation is less common today, approximately 5% to 10% of sarcomas of the proximal humerus or axilla are not candidates for limb-sparing surgery and require amputation.

versely across the chest at approximately the third rib level to the deltopectoral groove.
2. Raise a large subcutaneous flap to expose the clavicle, the anterior third of the deltoid, the sternoclavicular joint, and the posterior triangle of the neck.
3. Release the pectoralis major proximally from the clavicle, leaving a margin on the tumor. Transect the pectoralis minor tendon from its insertion on the coracoid. This exposes the axillary sheath. Open the sheath to expose the axillary vessels and brachial plexus.
4. Release the sternocleidomastoid muscle from its insertion and retract it proximally. The fascia overlying the posterior triangle of the neck is opened to expose the brachial plexus. Identify the internal jugular vein (which may be ligated) and carotid artery and dissect distally to the base of the neck, where the subclavian vessels are identified. The subclavian vein, which is located anterior to the scalenus anticus muscle, is often compressed by tumor in this region and is difficult to visualize. The subclavian artery is posterior to the scalenus anticus muscle. Proximal and distal dissection ensures proper identification and a safe resection.
5. Once the subclavian vessels and brachial plexus have been mobilized, osteotomize the clavicle. It may be necessary to resect the sternoclavicular or acromioclavicular joint. Transect the subclavius and deltoid muscles and then remove the tumor.
6. Rotate the pectoralis major muscle proximally and suture it to the trapezius. Rotate the sternocleidomastoid muscle and attach it to the proximal border of the pectoralis muscle to cover the neurovascular structures with soft tissue.
7. Place drains and suture the skin flap in place.

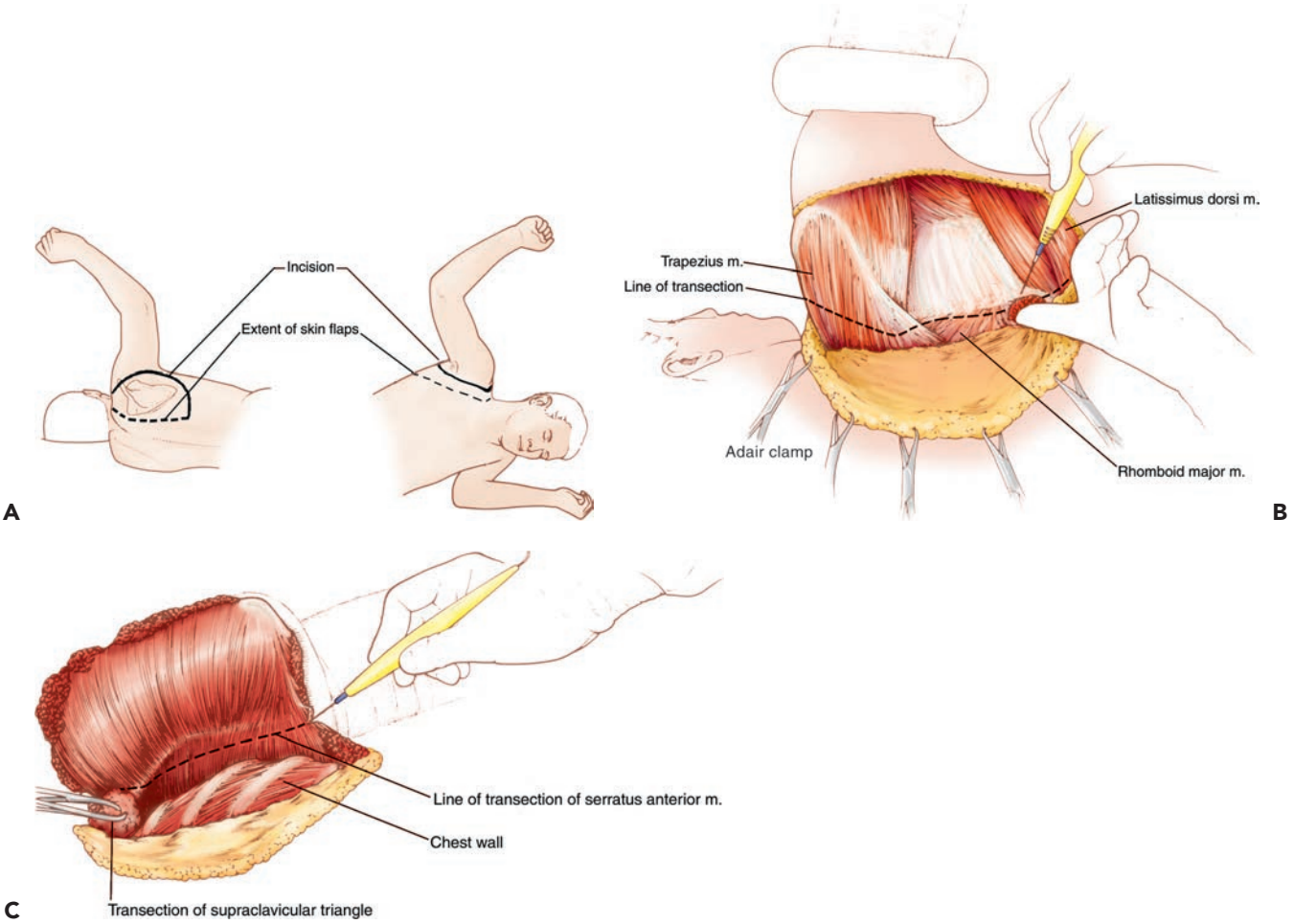


Figure 37-41 Technique of forequarter amputation. **(A)** Incision. **(B)** Release of periscapular muscles. **(C)** Mobilization of scapula and release of the anterior axillary muscles. **(D)** Isolation and ligation of the axillary vessels and brachial plexus. **(E)** Completion of amputation with posterior flap closure. Note that the surgical approach to a forequarter amputation may include initial exploration and ligation of axillary vessels from an anterior incision prior to the posterior approach (see text). (From Malawer M, Sugarbaker PH. Forequarter amputation. In: Malawer MM, Sugarbaker PH, eds. *Musculoskeletal cancer surgery: treatment of sarcomas and allied diseases.* Dordrecht, Netherlands: Kluwer Academic Publishers, 2001:294–296.)

AMPUTATION

As a result of advances in chemotherapy regimens and limb-sparing surgical techniques, forequarter amputations are rare. Only 5% to 10% of patients with primary bone sarcomas and fewer than 5% of patients with soft tissue sarcomas of the shoulder compartment require amputation. In rare instances, however, forequarter amputation may be indicated for palliation of patients with locally advanced, unresectable metastatic carcinomas of the shoulder girdle. These tumors commonly arise from metastatic spread to regional lymph nodes, the proximal humerus, or the scapula. A large soft tissue mass may encase the neurovascular bundle or invade the chest wall. At this point, the tumor becomes unresectable. Patients typically present with the following symptoms: severe intractable pain, a useless extremity, varying degrees of paralysis or sensory impairment, and chronic lymphedema. Continued tumor growth may lead to tumor fungation, sepsis, hemorrhage, and venous gangrene (Fig. 37-40).

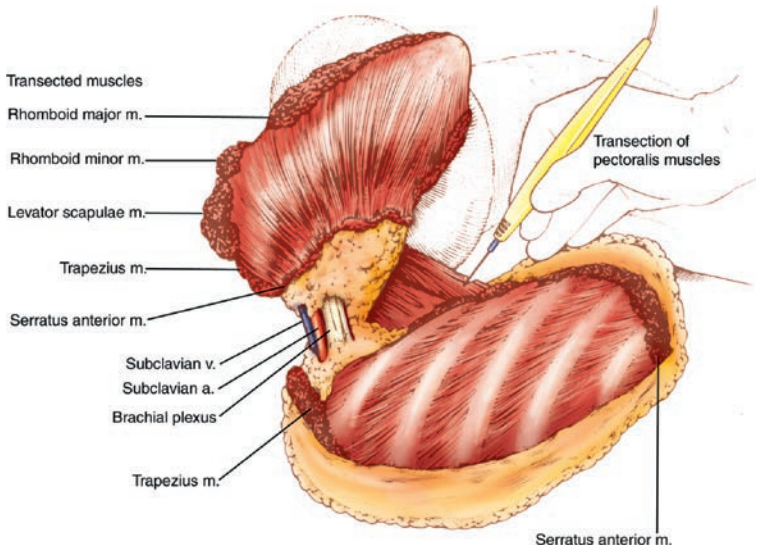
The indications for amputation include extremely large tumors that are associated with pathologic fracture, tumor hemorrhage, fungation, infection, or brachial plexus or axillary neurovasculature involvement. Forequarter amputation is contraindicated when tumor extends to the chest wall or extends to the paraspinal and posterior triangles of the neck structures. The surgeon must not proceed with amputation until after ascertaining that all surgical margins will be free of tumor or for palliation of uncontrolled pain, as this radical procedure is both debilitating and disfiguring and should be avoided in a patient where curative intervention is not anticipated.

Staging studies for an anticipated forequarter amputation are necessary to map out the local anatomy. It is recommended to perform CT, MRI, angiography, and venography prior to surgery. The final decision about proceeding with the amputation is made intraoperatively, after exploration of the tumor and neurovasculature structures.

If a preoperative biopsy is required, the biopsy site should follow the incision to ensure that it can be easily removed during the procedure. Care should be taken to avoid contamination of the large posterior flap, the deltopectoral interval, the suprascapular area near the neck, and the pectoralis muscles.

When forequarter amputation is required, the following surgical guidelines are followed (Fig. 37-41):

- Place the patient in a lateral position to facilitate a semilateral approach.
- Expose the brachial plexus and axillary vessels via the anterior portion of the utilitarian incision; at this point, the tumor is determined unresectable.



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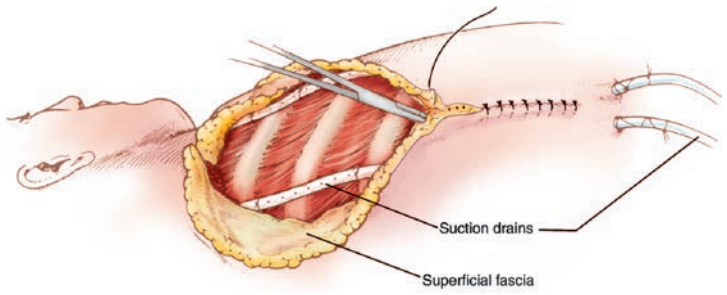

E

Figure 37-41 (*continued*)

- Prepare to explore the anterior vascular vessels by detaching the pectoralis major muscle from the clavicle, using a clavicle osteotomy at the proximal (one-third) junction.
- Clamp the vessels inferior to the clavicle.
- Use the posterior approach to detach the scapula from the trapezius, rhomboid muscles, levator scapulae, and latissimus dorsi.
- Elevate the scapula from the chest wall by detaching the latissimus dorsi to expose the chest wall.
- If no chest wall involvement is noted, the amputation proceeds with extension and connection of the anterior and posterior incisions.
- Remove the forequarter following ligation and transection of the brachial plexus and subclavian vessels.
- Insert perineural catheters bolused with 10 mL of 0.25% bupivacaine into the retained ligated nerves for regional postoperative pain relief.

Close a large posterior flap over the remaining defect and place a chest tube for drainage. A flap may need to be mobilized to accommodate heavily irradiated skin in patients who were previously treated with external-beam radiation therapy.

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38 Sepsis of the Shoulder Girdle

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This chapter will address the issues of diagnosis and management of infections involving the shoulder. The major focus is on primary pyarthrosis of the glenohumeral joint. We will also discuss shoulder sepsis associated with osteomyelitis, septic subacromial bursitis, soft tissue infection, infections involving the sternoclavicular and acromioclavicular joints, and postsurgical complications. No attempt has been made to specifically include Lyme disease, brachial plexus neuritis, or nonsuppurative (viral, fungal, or mycobacterial) infections, which may be part of the differential diagnoses of an

infected shoulder joint. For the purpose of presentation, the topic is subdivided into pathophysiology, specific clinical entities, evaluation techniques, treatment, authors' preferred treatment, prognosis, and directions for further study.

PATHOPHYSIOLOGY

Septic arthritis of the shoulder is an inflammation of the glenohumeral joint involving one or more foreign pathogens that cause, or are suspected of causing, the inflammation. These pathogens can be bacteria, viruses, fungi, or parasites, and can gain access to the joint through a number of different means. The defense mechanisms of the host and the properties of the invading organism play an important part in the pathophysiology of septic arthritis, as does the premorbid condition of the joint. Chronic arthritis and trauma resulting in soft tissue damage can predispose a joint to infection.

Joint sepsis may be classified according to pathogenesis. There are three basic mechanisms: direct inoculation, contiguous spread from adjacent osteomyelitis, and hematogenous dissemination.

Hematogenous Septic Arthritis

Hematogenous dissemination from another organ system, such as skin breakdown, urinary system infections, or pneumonia, is the most common. In over 50% of patients with intraarticular sepsis, there is a positive blood culture.[57] Goldenberg and Cohen isolated the pathogen from a distant focus in 50% of the cases.

In the shoulder, branches of the suprascapular and subscapular arteries, along with the anterior and posterior circumflex humeral arteries, form an extracapsular arterial ring, which supplies the proximal humerus (Fig. 38-1A,B).[51] This anastomosis gives off branches that penetrate the capsule and form an intraarticular synovial ring.[69] This has been termed the "transition zone" and is located between the synovium and the articular surface.[126] It is in this area that the arterioles loop acutely toward the periphery, creating a low-flow state, making the area more susceptible to receptor-specific interaction of the pathogen and the cell surface.

Spontaneous shoulder sepsis is the result of joint invasion during bacteremia. Septic arthritis has been shown to occur in experimental animals when bacteremia is created.[92] The abundance of the synovial vasculature and the absence of a basement membrane between the endothelial cells make synovial joints vulnerable to seeding by bacteria. Furthermore, most patients with hematogenous nongonococcal bacterial arthritis have at least one underlying chronic medical risk factor. These factors may be local, such as prosthetic and metallic implants, or they may be systemic, such as

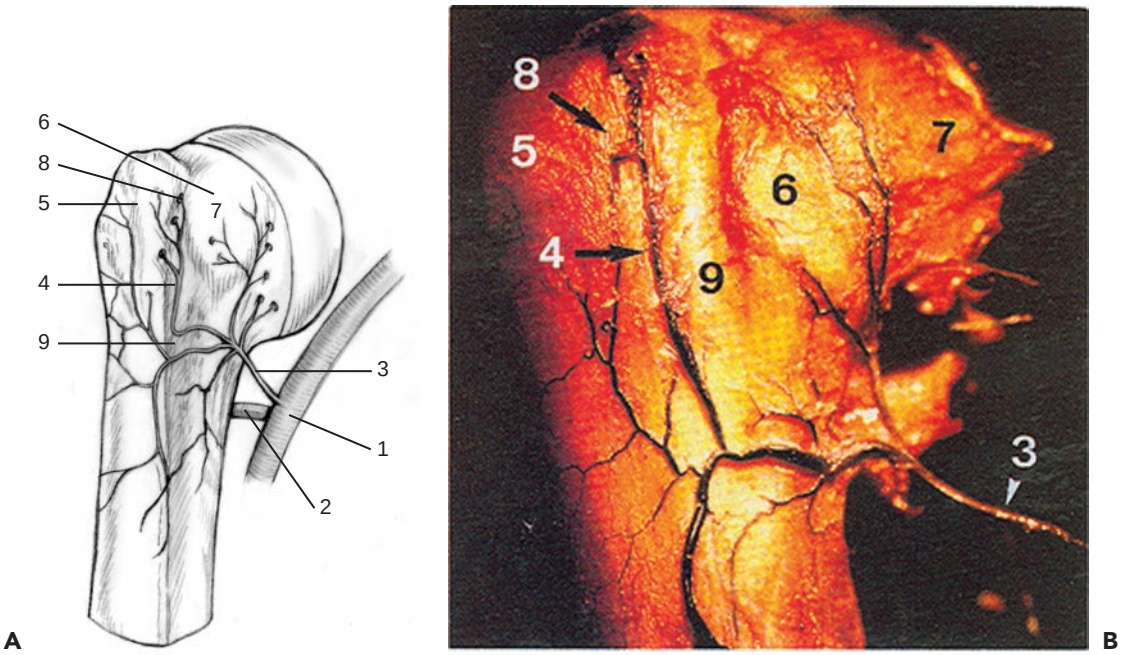


Figure 38-1 **(A)** Graphic representation and **(B)** photograph of the anterior aspect of the humeral head. 1 = axillary artery, 2 = posterior circumflex artery, 3 = anterior circumflex artery, 4 = anterolateral branch of the anterior circumflex artery, 5 = greater tuberosity, 6 = lesser tuberosity, 7 = insertion of the subscapularis tendon, 8 = constant site of entry of the anterolateral branch into the bone, and 9 = intertubercular groove. (Courtesy of Gerber C, Schneeberger AG, Vinh TS. The arterial vascularization of the humeral head. An anatomical study. *J Bone Joint Surg* 1990;72A: 1486–1494.)

TABLE 38-1
RISK FACTORS IN BACTERIAL ARTHRITIS

Host phagocytic defects
Complement deficiencies
Inherited disorders of chemotaxis or intracellular killing
Impaired host defense mechanism
Immunosuppressive drugs and glucocorticoids
Cancer
Chronic debilitating illness
Hypogammaglobulinemia
Direct penetration
Puncture wounds
IVDA
Joint damage
Chronic arthritis
Total or hemiarthroplasty
Other prior surgery

IVDA = intravenous drug abuse.

cancer, cirrhosis, rheumatoid arthritis, and intermittent bacteremic episodes from intravenous drug abuse or indwelling catheters[58] (Table 38-1).

Direct Inoculation

Direct inoculation may be traumatic or iatrogenic. Repeated corticosteroid injections[3] (Fig. 38-2), arthroscopy,[8] and open surgical procedures, such as rotator cuff repair and arthroplasty, have been shown to be associated with pyarthrosis of the shoulder. The incidence of infection following intraarticular steroid injection is extremely small. Hollander reported 18 infections in 250,000 injections,[76] and Gray et al. found only two cases complicating 100,000 injections.[61] The advent of sterile disposable needles and syringes and adherence to meticulous aseptic technique has helped lessen the risk. The existence of foreign bodies in or around the joint, such as nonabsorbable suture, stainless steel, cobalt chrome alloys, methylmethacrylate, and polyethylene, or devitalized bone from trauma can provide a nidus for adhesion and colonization by bacteria.[63,67,69] This nidus allows a glycocalyx biofilm to be expressed by the bacteria, which contributes to antibiotic resistance and limits the effectiveness of the immune response of the host.

Septic Arthritis from Contiguous Osteomyelitis

Osteomyelitis is most often hematogenous in origin and, in particular, is a disease of young children and the elderly. Hematogenous osteomyelitis commonly involves the metaphyseal area of rapidly growing long bones, usually occurring in the hip and knee.[106] When septic arthritis results from a contiguous infection such as osteomyelitis, it spreads from the bone to synovium, then to the joint space. This happens most often in infancy, when there is a vascular anastomosis between the epiphysis and the metaphysis. Studies conducted on the proximal femur by Trueta[145] showed a direct vascular communication between metaphyseal arterioles and the epiphyseal ossicle before 8 months of age. This allows a direct hematogenous communication between an osteomyelitis of the metaphysis and the adjacent joint synovium.[37,109] Between the ages of 8 and 18 months, the last vestiges of the nutrient artery system close down at the growth plate. The open physis at this point provides an effective barrier to the spread of infection to the joint by obliterating this vascular anastomosis.[137] This situation is analogous in the proximal humerus.

At skeletal maturity, there is once again a direct osseous connection between the metaphysis and epiphysis, secondary to closing of the growth plate and reestablishment of anastomoses between the metaphyseal and epiphyseal arterioles.[4] Therefore, infection of the proximal humeral metaphysis may extend to the epiphysis and the joint through the haversian system and Volkmann canals. In addition, the proximal 10 to 12 mm of the metaphysis of the proximal humerus is intraarticular, giving the pathogens of metaphyseal osteomyelitis direct access to the synovium.

Synovial Tissue and Infection

The anatomy of the shoulder joint is intricately involved in the pathogenesis of sepsis. All synovial joints contain synovial fluid, which can act as an excellent growth medium for bacteria and have a relative lack of immunologic resistance.[44] Type B synoviocytes are weakly phagocytic but in most cases are able to limit and clear a blood-borne bacterial infection.[7] Therefore, there must be an imbalance between normal synovial cell function and the invading bacteria for an intraarticular infection to develop.

Synovial tissue is relatively resistant to infection.[7] Examination of joints in which experimental septic arthritis has been produced reveals infrequent colonization of the synovium.[149] Receptors for collagen have been found on several strains of *Staphylococcus aureus*. It may be a lack of ligands or a functional host resistance mechanism that helps to prevent synovial colonization.[69]

Microscopic examination of the synovium shows that it is relatively thin in the area of the transition zone, rarely being more than three or four cell layers thick. The synovial capillaries are superficial, making them more susceptible to trauma. The lack of epithelial tissue in the synovium, and thus the lack of a basement membrane, means that

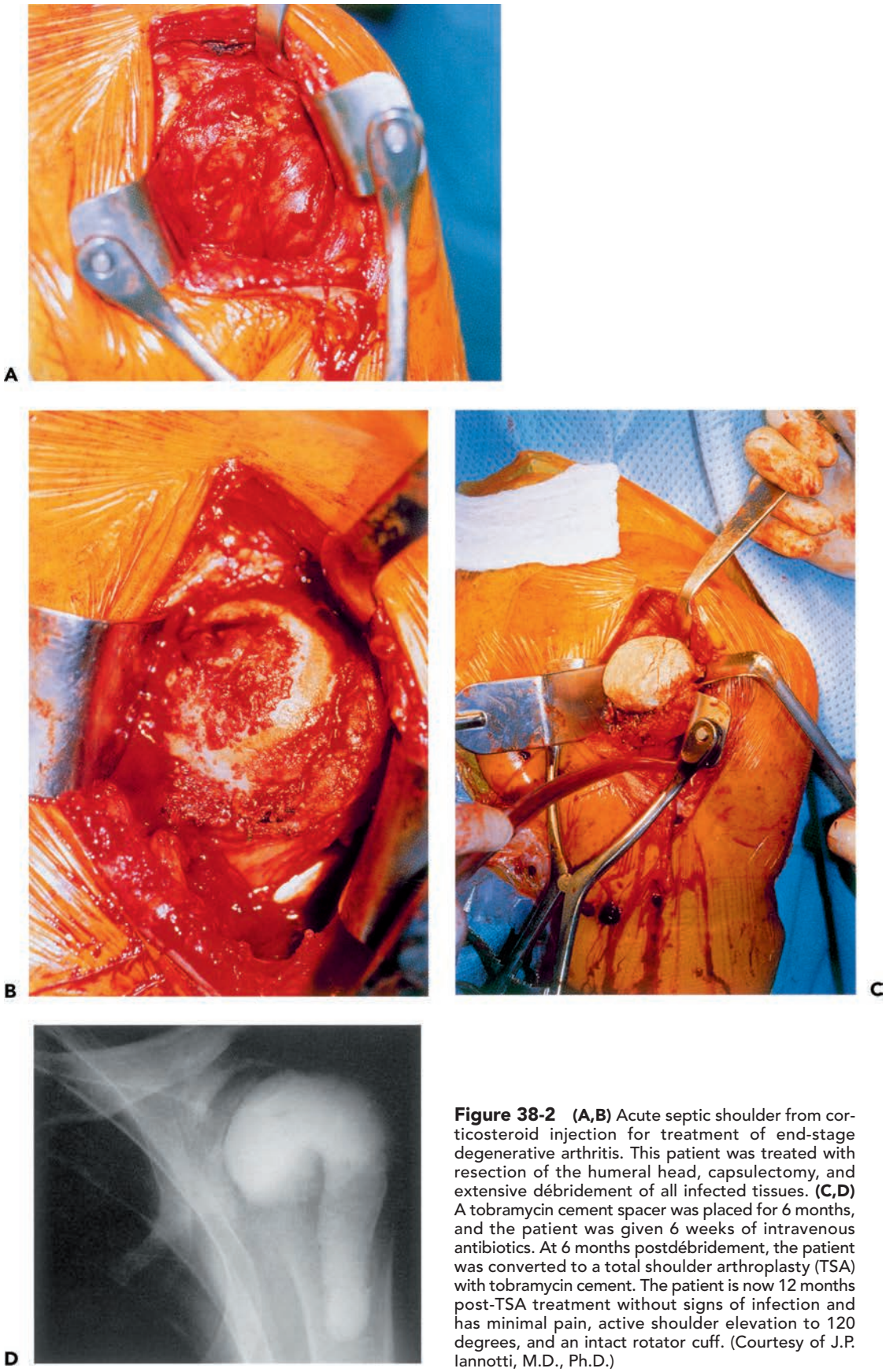


Figure 38-2 **(A,B)** Acute septic shoulder from corticosteroid injection for treatment of end-stage degenerative arthritis. This patient was treated with resection of the humeral head, capsulectomy, and extensive débridement of all infected tissues. **(C,D)** A tobramycin cement spacer was placed for 6 months, and the patient was given 6 weeks of intravenous antibiotics. At 6 months postdébridement, the patient was converted to a total shoulder arthroplasty (TSA) with tobramycin cement. The patient is now 12 months post-TSA treatment without signs of infection and has minimal pain, active shoulder elevation to 120 degrees, and an intact rotator cuff. (Courtesy of J.P. Iannotti, M.D., Ph.D.)

there is no structural barrier to the spread of bacteria from the synovium to the joint. Thus, transient bacteremia and trauma causing intraarticular hemorrhage can play a role in the pathogenesis of septic arthritis.[69]

Bacterial Adhesion

Integral to the pathogenesis of infectious arthritis is the preferential colonization of bacteria to articular cartilage, traumatized bone, or biomaterials that are not integrated with healthy tissues composed of living cells and extracellular matrix proteins.[12,67,69,130,138] Bacterial adhesion involves either very specific receptor–ligand or receptor–lectin– ligand chemical interaction or nonspecific interaction based on charge-related, hydrophobic, and extracellular polysaccharide-based interactions.[66] *S. aureus* has receptors for types I and II collagen,[136] fibrinogen, laminin, fibronectin, thrombospondin, bone sialoprotein, and heparin sulfate.[106] Many factors influence the adherence properties of bacteria, including (a) the surface energy and surface-free energy of the bacteria and biomaterial, (b) the extracellular components of the bacteria, (c) the bacterial interaction in mixed infections, (d) the host immune system, and (e) the extracellular matrix.[44]

All natural, biologic surfaces, with the exception of teeth and articular cartilage, are protected by epithelium, endothelium, or periosteum, which decreases bacterial adhesion by desquamation or by the presence of host extracellular polysaccharide molecules. *S. aureus* is the natural colonizer of cartilage and collagen, because it has specific surface-associated adhesins[33] for sites on collagen, not for enamel. Although the colonization of teeth by *Streptococcus mutans* is a natural symbiotic process that can be slowly destructive, the bacterial colonization of articular cartilage is unnatural and is rapidly destructive.[52,69]

Bacterial adherence is characterized by the production of an extracellular exopolysaccharide, within which the bacteria aggregate and multiply. Bacteria in aquatic environments grow predominantly in these biofilm-enclosed microcolonies adherent to surfaces.[64] Following initial colonization, the microcolonies develop coherent and continuous biofilms[46] that contain more than 99.9% of the bacteria in thick layers, within which they are protected from antibacterial agents[31,64,65] and the host immune defenses. This glycocalyx allows the bacteria to modify their local environment, limiting both the specific and the nonspecific arms of the immune response. Bacteria adherent to bone, methylmethacrylate, orthopedic devices, and surrounding tissue are harder to completely eradicate until the infected tissue and biomaterial are removed. Not infrequently, the infections are polymicrobial and difficult to culture adequately, unless special techniques are used.[54,57,63,65,66]

Microbiology

Patient age and host states help predict the bacterial cause of septic arthritis. Those organisms that frequently cause bacteremia in certain age groups are usually the infecting organisms, since joint sepsis is most commonly caused by hematogenous seeding. However, certain organisms, such as *Neisseria gonorrhoeae* and *S. aureus*, seem to have an avidity for the synovium, causing septic arthritis out of proportion to their incidence of bacteremia.[137] *S. aureus* is the most common cause of adult, nongonococcal bacterial arthritis, occurring in up to 50% of patients.[90] Ward and Goldner[151] noted 77% of infecting organisms to be Gram positive, of which 46% were *S. aureus*. *Propionibacterium acnes* is an anaerobic, Gram-positive bacilli that is found in lipid-rich areas, such as hair follicles and sebaceous glands, and in moist areas, such as the axilla. Although it is rarely the causal organism in other large joint infections, *P. acnes* has frequently been reported as the offending pathogen in shoulder sepsis and should not be dismissed as a skin contaminant.[74] Joint infections with Gram-negative bacilli have been increasing in incidence, ranging from 5% to 30% of all shoulder infections.[43,50,90,143] They most often occur in patients with intravenous drug abuse, malignancy, diabetes, immunosuppression, or hemoglobinopathy.[137] *Escherichia coli* and *Proteus* species are common infecting Gram-negative organisms from the urinary tract, and occur in patients who are not intravenous drug abusers. Although *Pseudomonas* and *Serratia* are the common organisms in intravenous drug addicts, the incidence of *S. aureus* in this population has been increasing.[6,13,43] *Streptococcus pneumoniae* is the most common organism in patients with chronic alcoholism and hypogammaglobulinemia.[107] Polymicrobial infections of the shoulder occur in 5% to 15% of patients, often associated with an extraarticular polymicrobial infection or penetrating trauma, especially in immunocompromised patients.[119]

Incidence

The incidence of shoulder sepsis is increasing as the population ages and the prevalence of chronic, debilitating disease increases.[90,143] Currently, primary shoulder sepsis accounts for 10% to 15% of all joint infections, whereas the hip and the knee account for 20% to 25% and 40% to 50%, respectively.[43] Septic arthritis of the shoulder is a rare occurrence in the young, immunocompetent person. More frequently, it is a disease of the elderly. Most patients have chronic, systemic, immunocompromising conditions such as diabetes mellitus, blood dyscrasias, renal failure, malignancy, and malnutrition.[44,57,90,151] Local factors also play a role in some patients, such as indwelling catheters, intravenous drug use, prior joint disease (rheumatoid or osteoarthritis), trauma, bursitis, or radiation therapy.[44,58,137]

Ward and Goldner reported that 74% of 27 adults with septic arthritis of the shoulder had some systemic condition causing immunocompromise or some type of local tissue abnormality. In 52% of the patients, both were present.[151]

CLINICAL ENTITIES

Natural History of Septic Arthritis

Studies conducted in animals have demonstrated that direct joint inoculation with bacteria is followed by synovial, bone, and cartilage changes within a matter of hours. Experiments have been performed on mice, rats, rabbits, chickens, and hamsters, and most have utilized direct joint inoculation. Septic arthritis caused by *S. aureus* in a rabbit model displays two processes acting simultaneously. The synovium becomes inflamed and hypertrophies within minutes of infection, with an influx of polymorphonuclear cells. This develops into an invading pannus, eroding and undermining the articular cartilage. Bacteria can be identified in and extruded by the pannus, thus maintaining the inflammatory reaction and lysosomal discharge.[118] Within 3 hours, a purulent exudate is observed, and within 24 hours multiple abscesses are seen. By day 5, synovial inflammation is so aggressive that there is extension below the cartilage interface, causing erosion and loosening in this area.

Simultaneously, by day 2, progressive loss of glycosaminoglycan occurs, as observed by a loss of safranin staining. This is most pronounced in the marginal areas near the leading edge of the pannus. The degradation of cartilage occurs through bacterial endotoxin, prostaglandins, and cytokine-mediated events that invoke a host inflammatory response and a release of destructive enzymes by synoviocytes and leukocytes.[44] Total glycosaminoglycan depletion occurs by 14 days, and the protein-polysaccharide–depleted cartilage is susceptible to degradation by collagenases released by the lysosomes.[118,152] The predominant cytokine is interleukin-1 (IL-1), which is released by synovial macrophages and circulating monocytes. IL-1 has been shown to inhibit chondrocyte proliferation and decrease expression of type II and X cartilage, making the articular cartilage more friable and susceptible to bacterial adhesion. Bremell et al., in their studies on septic arthritis in rats, noted the importance of $CD4^+$ T lymphocytes expressing IL-2 receptors, indicating activation. Deletion of T lymphocytes downgraded the intensity of infection, indicating a pathogenic role.[14]

If infection remains untreated for 7 to 10 days, cartilage fissuring and a decrease in height occur, most commonly involving the weight-bearing areas. Continued infection results in joint capsule and ligament dissolution, ending in fibrous ankylosis in 5 weeks in the rabbit model.[118] Antibiotics, administered in this animal model before or at the time of inoculation, significantly reduced joint destruction.[132] Irreversible changes occurred if the joint was not sterilized within 5 days of infection.[106]

Subacromial Septic Bursitis

Pyarthrosis of the glenohumeral joint may extend into the subacromial bursa. Most commonly, the infection occurs by direct erosion through the rotator cuff (Fig. 38-3). However, 10% of the patients may have intact cuffs.[151]

Rarely, subacromial septic bursitis may occur in isolation, as the primary infection,[142] or as a result of hematogenous seeding from a distant source of infection.[29] The diagnosis is made by aspiration of the bursa for Gram stain and culture. Aspiration is performed in an area that will likely have the highest yield, usually where there is maximal tenderness and fluctuation (Fig. 38-4).

Septic Arthritis of the Sternoclavicular Joint

The sternoclavicular joint is an unusual site for infection, comprising 1% of all cases of septic arthritis.[120] Sternoclavicular septic arthritis usually develops in patients with an underlying medical condition or predisposing factor, such as intravenous drug abuse,[13,53,59] diabetes mellitus, rheumatoid arthritis, liver disease, alcohol abuse, renal disease, malignancy, steroid use, or infection at another site.[19,120,155] However, infection may occur via a hematogenous route or by direct inoculation from trauma or subclavian vein catheterization in healthy patients. Ross and Shamsuddin[120] recently reviewed the published reports of sternoclavicular septic arthritis and found 170 cases, 33 of which were associated with intravenous drug abuse. Serious complications such as osteomyelitis (55%), chest wall abscess or phlegmon (25%), and mediastinitis (13%) were common. *S. aureus* was the most common pathogen, responsible for infections in 49% of the cases. *Pseudomonas aeruginosa* was responsible for only 10% of the cases; this represents a dramatic decline in incidence compared to older reports, presumably due to the end of an epidemic of pentazocine abuse among intravenous drug users in the 1980s. Before 1981, *Pseudomonas* was responsible for 9 of 11 cases of sternoclavicular septic arthritis among intravenous drug users. After 1981, 17 of 22 cases in intravenous drug users were caused by *S. aureus.* An earlier review by Wohlgethan et al.[155] found *S. aureus* to be responsible for infections in 8 of 10 patients with rheumatoid arthritis, and three of four patients with renal failure. Of seven patients with a history of alcohol abuse, six were infected with streptococci.

The diagnosis of sternoclavicular septic arthritis is often difficult, and there is usually a delay between the onset of

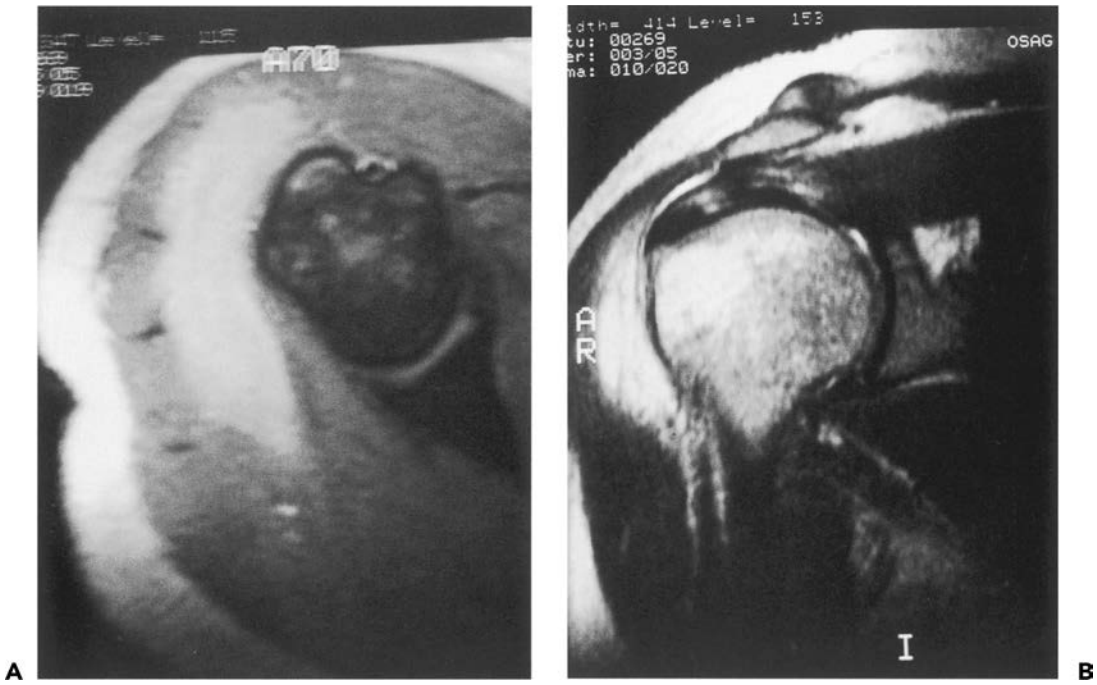


Figure 38-3 **(A,B)** Magnetic resonance imaging of acute subacromial infection with an abscess, following open rotator cuff surgery. (Courtesy of J.P. Iannotti, M.D., Ph.D.)

symptoms and diagnosis. Ross and Shamsuddin's review found that the median duration of symptoms before diagnosis was 14 days (mean 29 days).[120] In those 170 cases, symptoms most commonly involved pain in the anterior chest (78%), shoulder (24%), or neck (2%) long before other signs and symptoms occurred. Fever (more than 38°C) and bacteremia were present in 65% and 62% of patients, respectively. The sternoclavicular joint was tender in 90%, and limited shoulder motion was noted in 17% of patients.[19,48,59,120] Joint aspiration was not feasible in most patients, but when performed, cultures were positive in 50 of 65 patients (77%).[120] Computed tomography or magnetic resonance imaging should be obtained routinely to assess for the presence of chest wall phlegmon, retrosternal abscess, or mediastinitis.

Infection Complicating Hemi- and Total Joint Arthroplasty

Infection of the glenohumeral joint following shoulder arthroplasty is a relatively rare, although potentially devastating, occurrence. Susceptibility is dependent on a number of host factors, such as diabetes mellitus, rheumatoid arthritis, advanced age, remote sites of infection, malnutrition, and immunosuppressive chemotherapy.[154] The reported incidence of postoperative infection from total

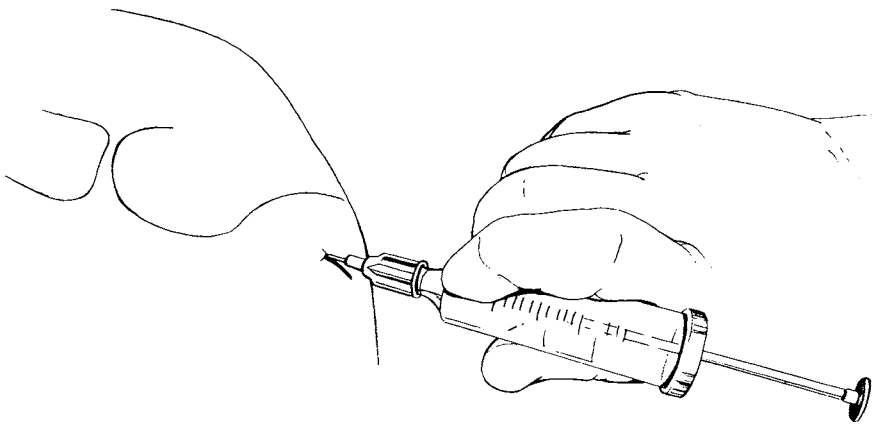


Figure 38-4 Aspiration of the subacromial bursa is usually performed in an area where there is maximal tenderness and fluctuation. The needle is inserted under the acromial edge (laterally in this case) and directed slightly cephalad. This illustration also shows the outline of the acromion and acromioclavicular joint.

shoulder arthroplasty ranges from 0.34% to 2.9%.[26,101] These infections can be divided into acute, subacute, or late. Acute infections are usually the result of intraoperative contamination and usually present within 3 months. Subacute infections occur when there is not a routine postoperative course. An example is a case that is complicated by prolonged wound drainage or fever that spontaneously resolves. This is usually followed by evidence of infection within 12 months, with component loosening. Late infections usually represent hematogenous spread from a distant focus, but could represent a chronic infection acquired intraoperatively.[95] *S. aureus, S. epidermidis,* and *P. acnes* are the most common organisms involved, although *Pseudomonas* and *Candida* species have been reported in the literature.[26,93]

Antibiotic prophylaxis for the prevention of hematogenous seeding of a total joint during the transient bacteremia induced by dental or other procedures is a controversial issue. Approximately 50% of all late prosthetic joint infections are due to staphylococci. This raises the question of overemphasis being placed on infections caused by dental procedures, because *S. aureus, S. epidermidis,* and *P. acnes* are unlikely oral pathogens.[153] Nonetheless, the consequences of prosthetic infection are so grave that bacteremic events should be avoided. In our opinion, prophylactic antibiotics should be administered in anticipation of procedures that cause bacteremia in certain instances. Animal studies have shown that the tendency toward prosthetic infection is greatest in the early postoperative period.[133] Clinical reports of infected prostheses support this finding. Over 50% of infections occur within the first 2 postoperative years.

The current recommendation is to provide prophylaxis for any patient undergoing an invasive procedure that gives the possibility of bacteremia within the first 2 years of prosthetic replacement. Regardless of the timing of the arthroplasty, a procedure performed at a distant site for an acute or chronic infectious process demands prophylaxis. Deacon et al. also believe that any patient with an immunocompromising condition, such as rheumatoid arthritis or hemophilia, should always be treated with prophylactic antibiotics.[38]

There are multiple regimens outlined in the literature, but the choice of prophylactic antibiotic depends on the normal flora of the suspected body region source.[139] The recommendations for prophylaxis during dental, head and neck, chest, and upper gastrointestinal procedures are for an oral cephalosporin, 1 g 1 hour prior to the procedure and 500 mg 4 hours after the procedure is completed.[95,139] Alternately, clindamycin and erythromycin can be used in cases of documented penicillin allergy. With high-risk biliary tract or colorectal surgery, parenteral cephalosporins should be used with clindamycin and an aminoglycoside as an alternative. Obviously, the risk of potential anaphylaxis has to be weighed against the risk of total joint infection during these procedures. Maderazo et al.[95] feel that the morbidity and possible mortality associated with a prosthetic infection warrants the small risk from the antibiotics.

Shoulder Sepsis Following Arthroscopy

Because arthroscopy of the shoulder is a sterile surgical procedure, the infection rates should be very low. They range in the literature from 0.04% to 3.4%.[8,34,84] The use of perioperative antibiotics has reduced the incidence of infection fourfold. Armstrong and Bolding, in a review of seven cases of septic arthritis following arthroscopy, noted a correlation with the use of intraarticular steroids.[3] Four infections occurred in 101 arthroscopies during a 3-month period, and three of these involved the use of intraarticular methylprednisolone. Two of the seven infections involved the glenohumeral joint and were believed to be secondary to unsterile electrocardiogram cables contaminating the operative field. The investigators postulated that the arthroscope was inadequately disinfected between cases in the remainder of the infections. It was noted that on several occasions, the equipment was soaked for less than the 20-minute time period required for disinfection with 2% glutaraldehyde. The review by Armstrong and Bolding demonstrated the importance of adequate patient, equipment, and operating room preparation. For those cases in which an arthroscopic procedure is followed by an open surgical procedure (e.g. mini-open cuff repair), Herrera et al. recommend a second skin preparation with Betadine after the arthroscopy to counteract the decreased efficacy of the initial preoperative skin preparation due to the constant arthroscopic fluid extravasation.[74]

Infection Following Rotator Cuff Surgery, Instability Surgery, and Open Reduction and Internal Fixation

The prevalence of infection after open rotator cuff repair has ranged from 0.27% to 1.7%, while the incidence of infection after mini-open rotator cuff repair has been reported to be 1.9%.[74,96,102] The most common offending bacteria are *S. aureus, S. epidermis,* and *P. acnes.* Infection following rotator cuff repair must be diagnosed quickly and addressed expeditiously to avoid damage to the cuff or the joint itself (Fig. 38-5A,B). Unfortunately, the cuff repair itself is at risk during postoperative pyarthrosis. Residual function depends on the amount and continuity of the remaining rotator cuff[113] (Fig. 38-5C,D). The presentation of deep shoulder infection following rotator cuff repair is often subtle, leading to a delay in diagnosis. Patients usually report pain and restricted motion, but often lack systemic symptoms and fever. White blood cell and neutrophil counts are often normal, but the erythrocyte sedimentation rate is usually elevated.[96,140] Timely diagnosis

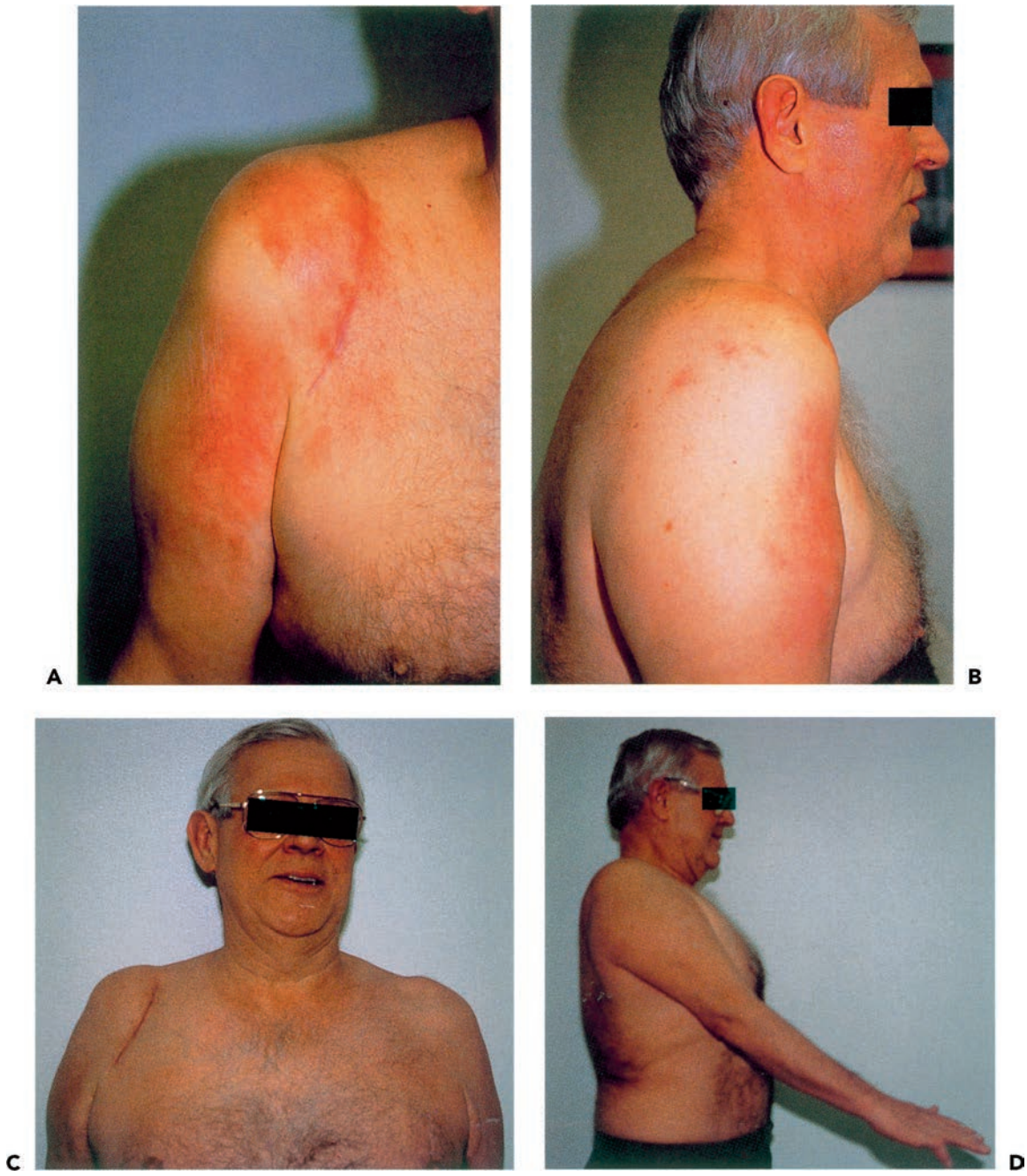


Figure 38-5 **(A,B)** Acute infection after open rotator cuff repair. At the time of débridement of the shoulder, the cuff repair was disrupted, and the deltoid origin was detached. The shoulder was débrided and all sutures were removed. The cuff was not repaired, but the deltoid origin was repaired with absorbable suture. The patient was treated for 6 weeks with intravenous antibiotics. **(C,D)** The infection resolved with the one surgical débridement and the deltoid remained intact. Clinically, the shoulder was painless but with poor function. (Courtesy of J.P. Iannotti, M.D., Ph.D.)

depends on attention to risk factors such as age greater than 60 and chronic lymphedema as well as a high index of suspicion.[96] Sperling et al. examined a short series of patients with early and late postoperative infections following surgery for shoulder instability including Bankart, Putti-Platt, and Bristow procedures.[134] All three cases of late infection had a sinus tract leading to a retained nonabsorbable suture. None of the six patients in the series had recurrent shoulder instability following eradication of their infection. Internal fixation of fractures about the shoulder provides an environment for bacterial adhesion and glycocalyx formation, making infection more difficult to eradicate.

One must also beware of the patient with postoperative drainage, slow incisional healing, and drainage around sutures that are extruded from the skin, because occult infection following open reduction and internal fixation is one of the most common causes of infection following total shoulder arthroplasty.[70]

Septic Arthritis Superimposed on Rheumatoid Arthritis

Patients with rheumatoid arthritis are more susceptible to joint sepsis, compared with those without the disease. Their underlying chronic joint symptoms may delay the diagnosis of infection. Furthermore, the acromioclavicular joint may be involved, adding to the complexity of presentation. Gristina et al. reviewed 13 cases of septic arthritis in patients with rheumatoid arthritis and found that most presented with a sudden exacerbation of the usual arthritic pain, abrupt onset of swelling, and increased joint temperature. Only 9 of 13 were febrile. The infecting organism was *S. aureus* in 12 cases and *E. coli* in one case.[68] Several factors may predispose a patient with rheumatoid arthritis to infection, including a poor overall health status with coexisting morbidities (such as diabetes), the chronic systemic administration of corticosteroids and cytotoxic drugs, and the intraarticular use of corticosteroids.[100] It also has been suggested that the synovial leukocytes of rheumatoid patients may have less phagocytic activity than normal, making their joints increasingly susceptible to sepsis.[10] The grave complication of septic arthritis should be suspected in any patient with rheumatoid arthritis when the clinical course worsens acutely, and synovial fluid should be aspirated immediately for examination. Clinical signs and symptoms are variable and inconstant, and the sedimentation rate and roentgenograms are unreliable. Upon diagnosis, prompt surgical therapy and parenteral antibiotics must be instituted, because this complication can carry a high mortality rate.

Disseminated Gonococcal Arthritis

Unlike patients with nongonococcal shoulder sepsis, those with joint infections secondary to *Neisseria gonorrhoeae* are generally young, healthy adults. Disseminated gonococcal infection is the most common cause of hematogenous septic arthritis of all joints. The most common clinical manifestation is a migratory polyarthralgia (70%). However, fever, tenosynovitis (67%), and dermatitis (67%) are commonly discovered on initial examination.[108] Joint aspirate yields a positive Gram stain result in only 25% of the cases, and 50% of cultures test negative.[109] Synovial fluid white cell counts are less than those for nongonococcal septic arthritis, but are still greater than 50,000 white blood cells (WBCs)/mm^3. Urethral, cervical, rectal, and pharyngeal cultures have a much higher yield and should be obtained from any young, sexually active patient suspected of having gonococcal arthritis. The infection shows a rapid response to ceftriaxone, and the arthritis generally resolves in 48 to 72 hours. Surgical decompression is not needed in most cases, because joint destruction is rare.

EVALUATION

Clinical Characteristics

A general workup scheme for pyarthrosis of the shoulder is outlined in Fig. 38-6. The typical clinical presentation of shoulder sepsis consists of complaints of pain, warmth, and swelling of the involved joint. A patient may exhibit a prodromal phase of malaise, low-grade fever, lethargy, and anorexia before the acute onset.[109] The acute phase usually consists of fevers and chills, with severe, incapacitating shoulder pain as the cardinal clinical manifestation. Physical examination reveals local signs of infection such as erythema, edema, tenderness, increased warmth, and limitation in range of motion (ROM). Previous reports have shown that fever is variably present (40% to 90% of patients), and when present may be low grade or transient.[105,119] Rosenthal noted pain in only 48 of 71 patients with septic arthritis, with limitation in ROM being the most consistent clinical sign,[119] helping to differentiate a superficial soft tissue infection from a joint infection. Atypical presentations occur when there is chronic arthritis, immunocompromised states, extreme age, intravenous drug use, or low-grade prosthetic joint infection. Previous use of antibiotics as well as corticosteroids or nonsteroidal antiinflammatory medication may mask symptoms. These factors, plus the low index of suspicion for shoulder sepsis, often lead to a delay in diagnosis. Ward and Goldner, in a review of 30 patients with shoulder pyarthrosis, noted mild symptoms in the 27 adults and a mean delay to diagnosis of 46 days.[151]

Laboratory Studies

Laboratory evaluation should consist of a peripheral WBC count with differential, erythrocyte sedimentation rate, and/or C-reactive protein. The WBC count may be normal to slightly elevated and may not show a peripheral blood leukocytosis, as was demonstrated to be the case in one-third of the patients with shoulder pyarthrosis described by Leslie et al.[90] Erythrocyte sedimentation rate is consistently elevated in patients with septic arthritis, but this may be elevated baseline secondary to chronic inflammatory arthritis. The C-reactive protein is an acute-phase protein produced by the liver in response to bacterial infection. It has been shown to increase more rapidly than the erythrocyte sedimentation rate, and may be of great value in the second, third, or fourth day of treatment to evaluate

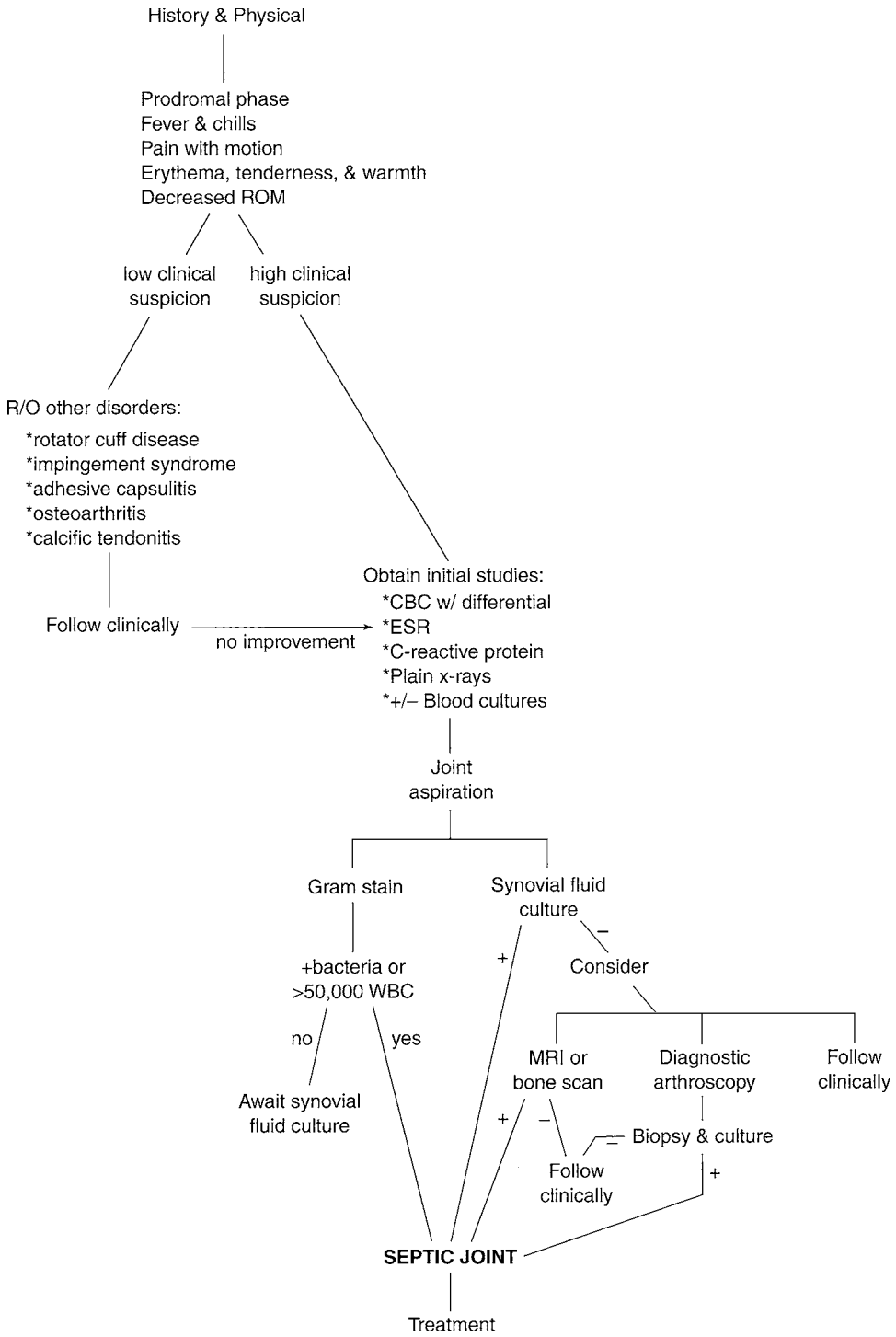


Figure 38-6 A general workup algorithm for infection involving the shoulder girdle.

improvement when the sedimentation rate is still increasing.[147] Blood cultures should be obtained from all patients with clinical signs and symptoms of systemic sepsis. In addition, cultures also should be obtained from any other possible sources of infection before antibiotics are administered.

Given the grave consequences of joint sepsis and the nonspecific clinical and laboratory findings, joint aspiration and culture are essential for making the diagnosis. The procedure must be performed with meticulous aseptic technique, preparing the skin with a povidone solution. Care must be used to enter the joint through intact, uninfected skin. An 18-gauge or larger needle should be used, so that viscous fluid can be aspirated if encountered. If difficulty accessing the joint arises, fluoroscopic guidance or contrast dye can be of assistance. Occasionally, aspirating

fluid from a joint is difficult, and nonbacteriostatic saline may be injected and reaspirated to obtain a sample. Joint aspiration can be painful, and appropriate analgesia and sedation (and, if necessary, general anesthesia) should be used.

Synovial fluid analysis is the key to correctly diagnosing septic arthritis. The fluid should be evaluated for Gram stain, culture, cell count and differential, and polarized microscopy. Cultures should include aerobes, anaerobes, fungi, and mycobacterium. In cases where *N. gonorrhoeae* is suspected, chocolate agar or Thayer-Martin plates should be used. Ideally, the specimen is plated at the bedside; however, this will vary according to each hospital's laboratory procedures. *N. gonorrhoeae* is an extremely fastidious organism, and cultures can be negative in up to 50% of the cases. Polymerase chain reaction is a technique that amplifies small amounts of bacterial DNA and provides a much higher sensitivity than culture alone. If available, it may be helpful when infection is suspected but cultures persistently remain negative.[55]

Examination of synovial fluid is an important step in the diagnosis of shoulder sepsis. Certain fluid characteristics suggest pyarthrosis, but none is absolutely specific. On gross examination, the fluid is often thick, yellow, and cloudy. Leukocyte counts are of limited use even at extreme values, because of the overlap with other types of arthritis. Values greater than 50,000 WBC/mm^3 are suggestive of bacterial arthritis, and values greater than 100,000 WBC/mm^3 are rarely from other causes. Polymorpholeukocytes are predominant, usually comprising greater than 90% of the leukocytes. Synovial fluid glucose levels may be decreased later in the process but are of limited value, unless serum levels are obtained at the same time and after at least 6 hours of fasting. Levels greater than 40 mg/dL below the serum level are consistent with pyarthrosis.[128] The presence of crystals in synovial fluid analysis does not rule out infection, because the two processes may coexist. Joint infection lowers the pH, occasionally causing the precipitation of urate or calcium pyrophosphate.[44] Table 38-2 summarizes synovial fluid findings.

Aspiration of the glenohumeral joint may be performed in one of several ways. The posterior approach (Fig. 38-7) may be the most commonly used route, owing to the ease of locating anatomic landmarks and the patient not being able to watch the procedure. It is performed under aseptic technique, with wide preparation of the skin with Betadine. After local anesthetic is injected into the skin, muscle, and capsule, an 18-gauge spinal needle attached to a syringe is inserted 2 cm inferior and 2 cm medial to the posterolateral edge of the acromion. The needle is advanced in the direction of the coracoid process until the joint is entered. Care must be taken not to contaminate the specimen once it is obtained.

Imaging Studies

Plain radiographs should be the first study obtained when imaging septic arthritis. These are only occasionally helpful in diagnosing primary sepsis of the shoulder within the first 7 to 10 days after infection. They may show joint subluxation or soft tissue swelling, due to either joint effusion or synovial hypertrophy.[16] Later in the infectious process, 40% of patients show abnormalities.[151] As the invading pannus erodes the articular cartilage and adjacent bone within the confines of the capsule, joint space narrowing and marginal erosions appear radiographically.[71] As the septic arthritis spreads to adjacent bone, features of

TABLE 38-2
SYNOVIAL FLUID FINDINGS

Synovial Fluid	Normal	Noninflammatory	Inflammatory	Septic
Appearance	Clear	Clear	Opaque/translucent	Opaque, yellow/green
Viscosity	High	High	Low	Variable
WBC/mm^3	<1,000	<1,000	5,000–75,000	>50,000
Polymorphonuclear cells	<25%	<25%	>50%	>75%
Culture	Neg	Neg	Neg	Pos
Associated conditions		DJD	Rheumatoid arthritis	Bacterial infections
		Trauma	Crystal-induced arthritis	Immunocompromised[a]
		PVNS	Seronegative arthropathy	
		Neuropathic	SLE	
		SLE	Acute rheumatic fever	
		Acute rheumatic fever		

[a] Immunocompromised patients may well not manifest an elevated synovial WBC. A normal or noninflammatory WBC does not preclude an active pyarthrosis in this patient population. DJD = degenerative joint disease; PVNS = pigmented villonodular synovitis; SLE = systemic lupus erythematosus; WBC = white blood cell.

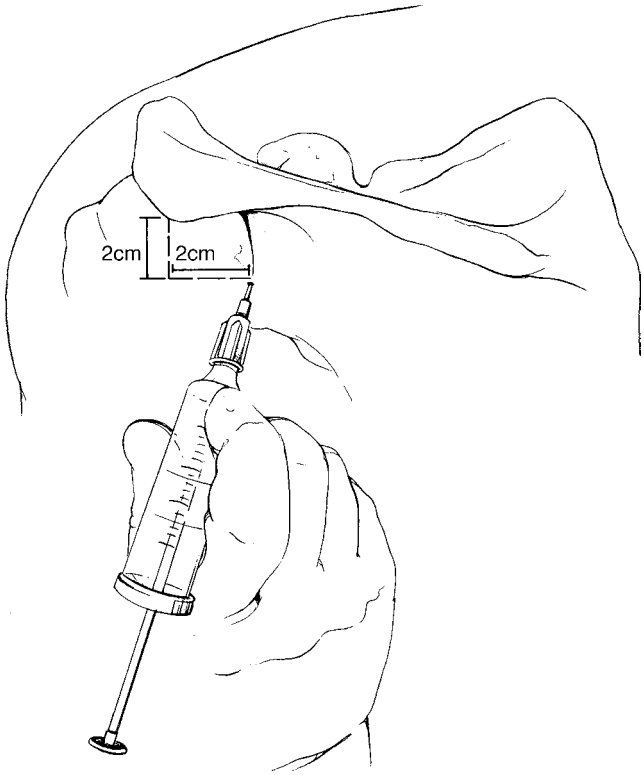


Figure 38-7 Posterior approach to shoulder aspiration. After preparing the skin with Betadine and injecting local anesthetic, an 18-gauge spinal needle attached to a syringe is inserted 2 cm inferior and 2 cm medial to the posterolateral edge of the acromion. The needle is advanced in the direction of the coracoid process until the joint is entered.

osteomyelitis, such as periostitis and bone destruction, are seen.

Plain radiographs become more important in postsurgical infections. They are helpful to assess the presence, condition, and location of hardware that may need removal. This is especially important for suture anchors, which may not be seen at the time of débridement surgery.

Magnetic resonance imaging (MRI) is recommended by some as the next imaging modality utilized if the diagnosis is still in question. MRI gives excellent resolution of soft tissues and fluid collections with an extremely high sensitivity, demonstrating abnormalities within 24 hours. However, it continues to lack specificity. It cannot distinguish infected joint fluid from inflammatory, nonseptic joint fluid.[16] MRI can clearly demonstrate cartilage destruction and small joint effusions, as well as intramedullary bone destruction and marrow edema. Computed tomography (CT) can give better bony resolution than either plain radiographs or MRI, clearly depicting subtle bone destruction.

CT scans also offer an advantage over plain x-rays in joints with complex anatomy or superimposed skeletal structures. In most cases of sternoclavicular pyarthrosis, results of plain radiography are negative, unless late bony destruction has occurred. CT is an excellent modality for diagnosis of sternoclavicular joint septic arthritis and the complication of abscess formation,[40] as well as for preoperative planning if conservative treatment has failed.[19]

Ultrasound is a useful, noninvasive imaging modality in screening for septic arthritis. Sonographic evaluation can reliably determine the presence of either a glenohumeral or an acromioclavicular joint effusion; absence of fluid within a joint makes the diagnosis of septic arthritis extremely unlikely.[32] If an effusion exists, laboratory analysis of the fluid is necessary to determine if the fluid is infectious. Ultrasound may be most useful, therefore, as a guide for joint aspiration in patients with difficult anatomy or large body habitus.

Bone scintigraphy is a physiologic imaging modality and can demonstrate the presence of disease before it appears on radiography. Technetium 99m (^{99m}Tc) is the radionucleotide of choice, although new ones are being developed. A triple-phase scanning sequence should be used. The first phase is the arteriographic phase, where images are obtained every 2 seconds for 1 minute after the ^{99m}Tc bolus. The blood pool phase is obtained at 10 minutes. The third, or bone phase, is obtained 2 to 4 hours after the initial injection.

Septic arthritis manifests as an increased uptake during the first two phases, and during the third phase, there is an increased uptake at the articular ends of the affected bones.[16] This is distinguished from osteomyelitis, in that actual osteomyelitis has a more focal area of uptake. This is a very sensitive, cost-effective study, and a negative scan rules out a septic shoulder. It also may reveal additional sites of joint sepsis in a patient unable to communicate or may diagnose a septic joint in a patient with fever of unknown origin. However, it is not specific, and the findings are the same as in any inflammatory arthropathy. The specificity is increased with labeling of autogenous leukocytes with either indium 111 or ^{99m}Tc. The degree of localization depends on the mechanics of polymorpholeukocyte accumulation. This technique is more sensitive for acute inflammatory lesions. Labeling of immunoglobulin G also has been performed with variable success in detecting joint sepsis.[146] A relatively new technique in orthopedic infection is single-photon emission computed tomography (SPECT). It is reported to have a higher sensitivity and resolution, and can differentiate the radioactivity in inflamed joints or bone from overlying normal soft tissue activity.[146]

Role and Indications for Diagnostic Arthroscopy

Septic arthritis can be a diagnostic challenge. Patients can have negative cultures, because of prior use of antibiotics (i.e., initiation of treatment before specimen is obtained, or treatment for other infections) or because of the presence of fastidious organisms. In such cases, arthroscopy can be a helpful diagnostic, as well as therapeutic, modality.

Arthroscopic examination of a septic joint usually reveals inflamed and friable synovium with fibrinous exudate. Adhesions and loculations of pus also may be present. The articular cartilage may appear normal but more often will have lost its lustrous appearance. In addition to visualization of the joint, direct synovial biopsy and culture of multiple sites can be obtained through the arthroscope.[17] The histology usually will show inflammatory cells with abundant neutrophils. Cultures of synovial tissue biopsy may have a higher yield for the fastidious organisms than the joint fluid cultures.

Classification System

Part of the difficulty in reporting outcomes in infected shoulder patients in the past has been the lack of a uniform classification system for septic joints. A number of systems exist to describe osteomyelitis[23,85,150] or infection around a total joint, but none is universally accepted. In the previous edition we introduced the University of Pennsylvania classification system for septic joints based on (a) the site and extent of tissue involvement; (b) the host's status, systemically and locally; and (c) the duration of symptoms and virulence of the organism (Table 38-3). In this system, the infectious process is staged using four anatomic types, three host physiologic classes, and two clinical settings.

The anatomic types include infection isolated to the periarticular soft tissue only or to the joint only, involvement of the joint and soft tissue, and involvement of the joint and bone. Anatomic type I is periarticular soft tissue infection without pyarthrosis. Such a case may occur in a postsurgical deep wound infection. Isolated glenohumeral sepsis (type II) occurs when the purulent material is confined within the capsule. Anatomic type III exists when there is involvement of the joint and surrounding soft tissue, such as deep wound infection or septic bursitis, along with the joint sepsis. There is no bony involvement in type III. When there is osteomyelitis contiguous with a joint infection, it is classified as type IV. In the shoulder girdle, this usually involves the proximal humerus but may occasionally develop in the acromion, distal clavicle, or glenoid.

The host is classified into either an A, B, or C physiologic group, according to the system of Cierny et al.[23] An A host represents a patient with normal metabolic and immune status. The B host is compromised either locally (BL) or systemically (Bs). Local issues include retained nonabsorbable suture or other biomaterial, local irradiation, scarring from multiple procedures, and lymphedema. Systemic compromise includes extreme age, chronic disease, or any condition causing suppression of the immune system. The C host status is reserved for those patients in whom the risks associated with aggressive treatment would outweigh the negative aspects of the infection. An example of a type C host would be an octogenarian with multiple medical comorbidities, an infected shoulder arthroplasty, and a draining sinus. In this patient, function may be superior with retention of the components, dressing changes, and chronic antibiotic suppression as compared to explantation of the prosthesis and long revision surgery.

The clinical setting takes into account the duration of symptoms and aggressiveness of the organism. We have grouped patients with less than 5 days of symptoms and infection with a less virulent bacterial strain into group 1.

TABLE 38-3
UNIVERSITY OF PENNSYLVANIA CLASSIFICATION SYSTEM FOR SEPTIC ARTHRITIS (MODIFIED FROM CIERNY-MADER OSTEOMYELITIS CLASSIFICATION)

Joint name (glenohumeral, elbow, hip, knee, etc.)
Anatomic type
- I: Periarticular soft tissue infection without pyarthrosis
- II: Isolated septic arthritis
- III: Septic arthritis with soft tissue extension, but no osteomyelitis
- IV: Septic arthritis with contiguous osteomyelitis

Host class
- A: Normal immune system
- B: Compromised host
- B_L: Local tissue compromise
- B_S: Systemic immune compromise
- C: Risk associated with aggressive treatment unwarranted

Clinical setting
- 1: Less than 5 days of symptoms and nonvirulent organism
- 2: Symptoms for 5 days or more, or a virulent organism

Clinical stage for the specific joint
Anatomic type + host class + clinical setting = stage

TABLE 38-4
RECOMMENDATIONS FOR ARTHROSCOPIC OR OPEN SURGICAL DRAINAGE AS OPPOSED TO REPEATED ARTHROCENTESES

Duration of symptoms for 5 days or longer before the initiation of treatment
Aggressive organisms
Methicillin-resistant *Staphylococcus aureus* (MRSA)
Gram-negative bacilli
Enterococcus
Clostridia
Glycocalyx-producing organisms
Elderly[138]
Immunocompromised host
Immunosuppressive therapy
Chronic debilitating illness
Malignancy
AIDS
Malnutrition
Diagnostic dilemma requiring tissue biopsy
Concomitant processes
Rheumatoid arthritis
Osteoarthritis
Periarticular osteomyelitis
Postsurgical infection
Failed repeated arthrocenteses
No or little clinical improvement
Persistent effusions after 5–7 days of treatment

Those patients who are infected with a virulent organism or with symptoms for 5 days or greater fall into group 2. The cut-off was chosen at 5 days because animal studies have shown that irreversible joint damage occurs if septic arthritis persists beyond this time. The virulent organisms may vary between hospitals and geographic locations but generally include methicillin-resistant *S. aureus,* Gram-negative bacilli, vancomycin-resistant enterococcal species, and clostridia (Table 38-4).

The University of Pennsylvania classification system for septic joints provides us a framework with which to approach those patients with septic arthritis, reminding us to consider multiple aspects of the patient including anatomic location, host status, duration of symptoms, and virulence of the offending organism. We believe it is useful for risk stratification, allowing us to make more informed treatment decisions. While we have introduced it in the previous edition of this shoulder text, it can be adapted to other joints.

TREATMENT

Antibiotics

Prompt recognition, correct diagnosis, joint decompression, and an organism-specific antibiotic regimen are essential, if disabling sequelae are to be avoided.[86] Similar to infection of other parts of the body, selection of an antibiotic is ideally based on the identification of the pathogen and its susceptibility profile. However, in some patients, the organism is not isolated; therefore, an empiric treatment must be started for the most likely infecting organism in that clinical setting. This will vary with the patient's age and underlying medical conditions. Furthermore, the choice of drugs must be linked with surgical options and other supportive measures in the overall management of the disease.

Initial empiric antibiotic selection is based on information such as patient age and risk factors, including history of intravenous drug abuse, rheumatoid arthritis, chronic illness, remote sites of infection, and immune status. The treatment may be instituted after a specimen is obtained for the appropriate studies, including Gram stain, culture, and sensitivity. Usually, a penicillinase-resistant antistaphylococcal drug is the initial choice for the typical Gram-positive cocci infection. In the United States, first-generation cephalosporins have been favored, because they are relatively nontoxic and inexpensive.[45] When there is a high likelihood of Gram-negative bacilli or methicillin-resistant staphylococci, the initial antibiotic choice is modified to third-generation cephalosporin or vancomycin, respectively. For patients receiving immunosuppressive agents and those who have developed nosocomial infections, an aminoglycoside needs to be included for additional coverage of *P. aeruginosa.* Initial antibiotics for intravenous

TABLE 38-5
SUGGESTED GUIDELINES FOR EMPIRIC ANTIBIOTICS FOR SEPTIC ARTHRITIS IN ADULTS

Clinical Setting/Gram Stain	Likely Organisms	Drug of Choice	Alternative Drug
Gram-positive cocci	*Staphylococcus aureus*, streptococci	Nafcillin or cefazolin	Clindamycin TMP/SMX Vancomycin
Healthy, sexually active individual with Gram-negative cocci (or negative stain)	*Neisseria gonorrhoeae*	Ceftriaxone	Doxycycline
Gram-negative bacilli	*Pseudomonas aeruginosa*, Enterobacteriaceae	Piperacillin ± gentamicin	Third-generation cephalosporins
Gram-positive bacilli	*Propionibacterium acnes*	Penicillin G	Nafcillin Vancomycin
Intravenous drug abusers	*S. aureus, P. aeruginosa, Serratia*	Cefazolin + gentamicin	Third-generation cephalosporins
Patients with major underlying disease, immunocompromised, or nosocomial infection	*S. aureus, Enterobacteriaceae, P. aeruginosa*, streptococci	Cefazolin + gentamicin	Third-generation cephalosporins
Patients with infected prosthesis	*S. epidermis, S. aureus, Enterobacteriaceae, P. aeruginosa*	Vancomycin + gentamicin	Imipenem

TMP/SMX = trimethoprim–sulfamethoxazole.

drug abusers should be for both *S. aureus* and *P. aeruginosa.* In the young, sexually active individual in whom the Gram stain test is negative for bacteria, gonococcal arthritis should be suspected and ceftriaxone started. Table 38-5 summarizes the guidelines for the initial empiric antibiotic therapy.

After the microbiologic data are available, the spectrum of coverage can be narrowed to maximize efficacy and decrease the risk of systemic toxicity. In light of the differing pathogens, their resistance profile at each hospital, and the advent of newer antimicrobial regimens, we generally obtain consultation with an infectious disease specialist.

The route of antibiotic administration is a subject of some debate. In the past, direct injection of antimicrobials into an infected joint to achieve high local levels was performed on a routine basis.[125] However, animal and human studies of intraarticular antibiotic concentrations have demonstrated that more than adequate levels can be achieved with clinically relevant parenteral doses.[91,124] For example, Frimodt-Moller[49] and Riegels-Nielsen et al.[118] investigated the diffusion (after intramuscular injection) of penicillin G, cloxacillin, clindamycin, and netilmicin into synovial fluid of infected rabbit knees and found sufficient local concentrations of all four drugs. Likewise in humans, similar studies were conducted with penicillin,[41,112] ampicillin,[5,77,104] cephalothin,[104] methicillin,[104,116] cloxacillin,[77] tetracycline,[83,112] erythromycin,[116] chloramphenicol,[83,116] lincomycin,[112] gentamicin,[98] and kanamycin.[5] All the antibiotics evaluated, with the exception of erythromycin, achieved articular concentrations in excess of the level required for bacteriostatic or bactericidal effect.[111] With the presently available agents having excellent synovial fluid penetration and the fact that there are associated risks, intraarticular antibiotic injection has fallen out of favor. Several investigators have reported chemical synovitis secondary to local tissue toxicity[2,44] and sterile abscesses[69] with the administration of intraarticular antibiotic injections. Most authorities now agree that the antibiotic regimen should almost always be given intravenously.[58]

A more controversial issue is the use of oral versus parenteral antibiotics. A number of investigators have shown good efficacy with the use of oral antibiotics to treat osteomyelitis and septic arthritis in children.[28,87,114,141] In all cases, however, the oral regimen was started only after an initial period of intravenous administration, usually 3 to 7 days. Black et al.[9] retrospectively studied this matter in 21 adults. Even though the failure rate was 14.3%, these investigators concluded that oral antibiotics are a "reasonable alternative to inpatient or outpatient parenteral therapy for treating adult patients who have bone and joint infections caused by susceptible organisms." To date, there has not been a well-designed, randomized, blinded, controlled study to determine whether oral antibiotics are as effective as parenterals in treating septic arthritis. Thus, at this time, oral antimicrobials should only be used in acute infections, after an initial period of intravenous administration. Furthermore, there should be ongoing clinical improvement as manifested by improved ROM of the joint, decreased pain, resolution of fever, and normalization of WBC and erythrocyte sedimentation rate.

Oral antibiotic administration has certain advantages, including cost savings, convenience, comfort, and decreased length of hospital stay. Prior to discharge, patients should be evaluated to ensure that adequate serum concentrations are

being achieved. Most clinicians[103,107] have arbitrarily maintained a serum bactericidal titer (SBT) of at least 1:8. However, Prober and Yeager[114] recommend SBT peak levels of 1:16 and trough levels of 1:2. The potential risk of an oral antibiotic regimen is that patients may not take the medication as required, due to lack of compliance or difficulty obtaining the medication. Thus, the importance of a full course of therapy must be stressed, and the patients should be carefully instructed on how to take the medication(s) with regard to proper dosage and timing.

The duration of antibiotic therapy is another issue that is a subject of discussion. There are no controlled studies to document the optimal length of treatment. However, certain guidelines have been established. The total duration of antibiotic regimen varies with the pathogen isolated, the patient's underlying condition, and adjuvant medical or surgical procedures. For gonococcal septic arthritis, 7 to 10 days is generally recommended. For streptococci or *Haemophilus* species, a 2- to 3-week duration is usually adequate. In the cases in which more virulent organisms such as *S. aureus* or Gram-negative bacilli are isolated, a 4- to 6-week course of appropriate antibiotic is required. Immunocompromised patients or those with a slow clinical response will need the full 6 weeks of treatment.[129] It is generally agreed that a septic shoulder in the setting of concurrent osteomyelitis will need 6 weeks of antibiotic treatment after the last major débridement surgery.[22]

Nonsteroidal Antiinflammatory Drugs

Sterilization of the joint with antibiotics and irrigation does not completely remove all the bacterial products. These microbial fragments may persist in the joint for prolonged periods and contribute to "postinfectious synovitis."[123] Nonsteroidal antiinflammatory drugs (NSAIDs) may help to reduce this inflammatory process. In an animal model, it was shown that naproxen, when administered in combination with antibiotics, decreased the amount of glycosaminoglycan and collagen loss.[132] These results suggest that use of NSAIDs in addition to the antibiotic regimen may minimize the destruction of articular cartilage. If used, nonsteroidal drugs should not be started too early in the course of infection, because their action may mask a poor clinical response to the antibiotics. We usually start them after 4 to 5 days of antibiotic treatment.

Antibiotic-Impregnated Polymethylmethacrylate

Since described by Buchholz and Engelbrecht[18] in 1970, antibiotic-impregnated polymethylmethacrylate (PMMA) has been used in the treatment of infections of the soft tissue and joint arthroplasty, as well as osteomyelitis. The main advantage of this treatment is that it allows for a high concentration of antibiotics to be delivered locally while minimizing the risk of systemic toxicity. The antibiotic released is not absorbed systemically, therefore resulting in local concentrations five- to 10-fold higher than when administered parenterally.[72] In vitro and in vivo studies have shown that this mode of therapy is safe and effective. In particular, Adams et al.[1] found that clindamycin, vancomycin, and tobramycin exhibited good elution characteristics and had consistently high concentrations in bone and granulation tissue.

Antibiotic-impregnated PMMA may be utilized in one of several ways: (a) as cement for fixation of prostheses in joint arthroplasty,[144] (b) as a spacer block (see Fig. 38-2C,D) to maintain the soft tissue envelope after resection arthroplasty and débridement,[11,115] or (c) as a string of beads embedded in the soft tissue or bone for chronic infection.[62] Powdered antibiotics are used in the admixture, because adding aqueous solutions of antibiotic to the cement interferes with the prepolymerization process, resulting in mechanically weakened cement.[97] When used for fixation, the fatigue strength of the cement is not significantly altered when 1.2 g of the appropriate antibiotic (usually tobramycin) is mixed per 40 g of PMMA.[36] In the form of a spacer or beads, the fatigue strength of the PMMA is less critical; therefore, a higher concentration of antibiotic may be added to the cement powder. Hofmann et al.[75] described mixing 4.8 g of powdered tobramycin in each 40-g batch of Simplex-P cement (Howmedica, Rutherford, NJ) for use as a spacer block, whereas Cierny et al.[23] reported using 4.8 to 9.6 g of tobramycin per 40 g of PMMA for making the beads.

Elution of antibiotics from the cement occurs by diffusion, in which there is a rapid initial release, followed by a sustained release that progressively diminishes over weeks to months.[47,73] Because the spherically shaped beads have a greater total surface area, more antibiotic per unit time is released when the impregnated cement is used in the form of beads than when used as a spacer block or for fixation.[73] In vitro studies by Marks et al.[97] demonstrated that antibiotics diffused from Palacos-R (Richards, Memphis, TN) in larger amounts and greater duration than from Simplex-P. However, these findings were not substantiated by in vivo studies by these same investigators. In addition, Brien et al.[15] found no statistical difference in the elution characteristics of tobramycin from these two cements.

In shoulder sepsis, antibiotic-impregnated PMMA may be a good adjunct, temporary therapy for those patients with concomitant osteomyelitis of the proximal humerus. They are placed after thorough irrigation and débridement, and the wound is closed. The cement is removed at the time of definitive surgery.

Evacuation and Decompression of the Joint

The goals of treatment of septic shoulder include sterilization and decompression of the joint with removal of all inflammatory cells, lysosomal and proteolytic enzymes,

and fibrinous materials. In non–surgery-related infection, the preferred method of joint decompression remains controversial. Two schools of thought exist. One stems from the medical and rheumatologic literature, suggesting that repeated needle aspirations and appropriate antibiotics may be all that is necessary in a pyogenic arthritis of the shoulder.[58,69] The other is based on the principle that surgical débridement is the best treatment for septic arthritis and osteomyelitis of the shoulder. There are studies to support both views. However, when scrutinized closely, it may be borne out that surgery offers distinct advantages.

Those who advocate repeated arthrocenteses cite extended hospitalization, wound management problems, and anesthetic risks as reasons to avoid surgical intervention. A number of retrospective studies from the 1970s and 1980s, comparing infected joints treated with needle aspirations versus surgical drainage, concluded that septic joints can be medically managed with good results.[56,58,99,119]

In 1980, Rosenthal et al.[119] analyzed 71 nongonococcal septic joints and found that medical therapy (parenteral antibiotics and frequent aspirations) led to good result in 74%, as opposed to only 32% in the open irrigation and drainage cases. Only one patient in their series had a septic shoulder, and was managed successfully without open drainage. A separate study in 59 patients by Goldenberg et al.[56] showed full recovery in 67% of those treated by arthrocentesis versus only 42% of those surgically treated. Only nine of these patients had sepsis of the shoulder with the majority having infected knee joints; results were not specifically reported for the shoulder subgroup. Master et al.[99] reported on a small series of eight septic shoulders in which medical management, including closed drainage, achieved good outcome in four patients (five shoulders). Three patients, however, required open drainage to eradicate the infection.

Like other investigators,[69] we question these data. Clearly, there are weaknesses in these studies. They were all uncontrolled; therefore, the indications for the treatment modality were undefined and determined predominantly by the admitting service. Because of the lack of randomization, there were a number of biases. For example, in the Goldenberg series,[56] 47% of the surgical patients were infected with *S. aureus,* whereas only 26% of the medical patients were infected with this virulent organism (this information is not available in the Rosenthal series). Undoubtedly, infection with a more virulent organism such as *S. aureus* will result in a worse outcome.[50,90] The duration of symptoms prior to treatment (i.e., delay in treatment) will also affect the result.[2,50,56,86] In all three series, there was a greater delay in the group of surgically treated patients who had the poor outcome. Another weakness is the outcome data. No assessment of function or follow-up was reported in the study by Master et al. Although the Rosenthal and Goldenberg series included ROM as part of their assessment, it is unclear how they were able to obtain the premorbid ROM because these studies were retrospective, and the patients were evaluated only after the fact. Of interest, Rosenthal pointed out that of the six patients who failed medical management and subsequently required surgical drainage, three did well. Goldenberg had seven patients undergo surgery after medical failure but did not report follow-up. With the exception of the Master study, most of the cases reported were septic knees, and as such, the generalizability of these studies to the shoulder should be questioned.

If aspiration is the choice of therapy, it should be performed under sterile conditions with a large-bore needle. A long spinal needle may be necessary to penetrate the glenohumeral joint in an obese or muscular patient. Attempts to drain the joint completely are essential and should be repeated frequently (once or twice daily[137]) until the effusion ceases to recur.[58]

Although many internal medicine physicians view incision and drainage as an alternative form of treatment, most orthopedic surgeons believe that shoulder sepsis demands surgical treatment. Proponents of surgical drainage describe technical difficulty with shoulder aspiration, inadequate needle evacuation of purulent material secondary to loculations and adhesions, pain associated with multiple arthrocenteses, and potential iatrogenic needle inoculation of subchondral bone as reasons for surgery.[43] Again, there are only retrospective studies to support this view.

Leslie et al.[90] reviewed 18 cases of septic arthritis of the shoulder with a minimum of 1-year follow-up. Ten patients were treated with arthrocenteses and eight with surgical drainage at the outset. Of the 10 who initially had repeated aspirations, one died, one had no motion, one had only passive abduction and flexion to 90 degrees, and seven required an open irrigation and drainage. Of the latter seven, one died, two had no active motion, one had less than 45 degrees of flexion, and three had greater than 90 degrees of flexion. There were no deaths in the eight patients who underwent an operative procedure from the outset. Two had flexion of at least 90 degrees, two had flexion of 45 degrees or less, and four had no active motion. Although the sample size was too small to yield statistical significance, the investigators noted that arthrotomy resulted in a better outcome than did repeated aspirations. At Bowman Gray School of Medicine, Toby et al.[143] came to the same conclusion after reviewing 15 cases of shoulder infection. Further support for open drainage in joint infections is offered by Lane et al.,[89] who found that patients with a 3 or more day history of knee pyarthrosis and those with *S. aureus* or Gram-negative bacillus infections fared better after open irrigation and drainage.

Without prospective randomized studies to provide conclusive evidence either way, the debate over medical versus surgical drainage of shoulder infections continues. However, mounting evidence is accumulating for the use of

surgical arthroscopy in septic arthritis cases.[17,110] The diagnostic advantage of arthroscopy is that it allows for direct visualization of the entire joint. Visualization is essential in determining the extent of the disease and enabling tissue biopsy in atypical or challenging cases. The therapeutic advantage of arthroscopy is that the joint can be adequately drained, thoroughly débrided, and copiously irrigated. Prognostically, arthroscopic irrigation and drainage reduce hospital stay and allow for early ROM, which may be helpful in preserving joint function.[121] Recommendations for arthroscopic or open surgical drainage, as opposed to repeated arthrocenteses, are summarized in Table 38-5.

Early use of arthroscopy in musculoskeletal infection was entertained mostly for pyarthrosis of the knee. In 1981, Jarrett et al.[81] reported the first successful arthroscopic débridement of an infected knee in a patient who failed medical management but was too ill for general anesthesia and arthrotomy. Since then, a number of small series[79,80,131] have shown good to excellent results with minimal operative morbidity. Stutz et al. demonstrated that arthroscopic irrigation and systemic antibiotic therapy was 91% successful in eradicating septic arthritis in a series of 78 joints including 10 shoulders.[140] Later-stage infections, however, often required multiple procedures to achieve clinical healing.

Suppurative arthritis has also been drained with percutaneous catheters placed under fluoroscopy.[117] A handful of these cases involved the shoulder.[122] Although the investigators state that septic arthritis can be successfully treated with drainage of the joint via a percutaneous catheter in combination with antibiotic therapy, the experience is relatively limited at this time. It remains to be determined whether this modality will have a future role in the treatment of joint infection.

Management of Sternoclavicular Joint Infection

The majority of the cases of sternoclavicular septic arthritis respond to parenteral antibiotics and repeated aspiration or simple incision and drainage. In patients with chest wall phlegmon, retrosternal abscess, or mediastinitis, a more radical débridement including excision of the medial head of the clavicle, intraarticular disc, and portions of the manubrium must be performed.[19,120]

Management of Postoperative Infections

The success of treatment for a postsurgical infection is dependent on obliteration of the infectious organism and the restoration of function. If the infection is superficial and limited to the subcutaneous tissue, meticulous observation, local wound care, and antibiotics are usually sufficient for successful outcome. However, for a deep postsurgical infection, a more aggressive approach is required with débridement of the glycocalyx film, avascular bone, infected sinus tract, soft tissue, and foreign material (Fig. 38-8).

For the infected shoulder in which foreign material is present in or about the joint, management must be individualized to be successful (Fig. 38-9). When the hardware is serving to stabilize a fracture, débridement of bone and soft tissue with adequate drainage may be attempted while preserving the internal fixation in situ. If the infection persists, the hardware must be removed to successfully gain control of the process. On the other hand, if the hardware is loose or not contributing to the fixation, it should be removed at the first débridement. Care must be taken to preserve cuff tissue and the tuberosities if function of the shoulder is to be maintained.

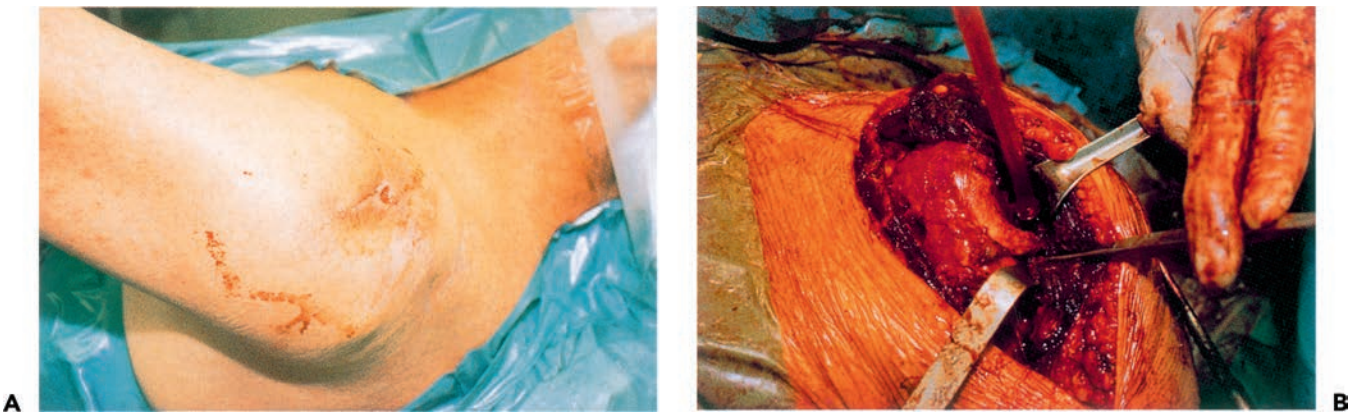


Figure 38-8 **(A,B)** A 2-year chronic shoulder infection occurring after rotator cuff repair treated with multiple limited débridements. The patient presented with a chronic draining sinus and chronic osteomyelitis with a sequestrum. The patient had undergone extensive débridement of all dead bone, soft tissue, sinus tract, and suture material via both an anterior and posterior approach. The wounds were packed open and closed 3 days later, after a second irrigation and débridement. The patient had 6 weeks of antibiotics. The infection resolved without further treatment. The patient was left with a stiff but painless shoulder. (Courtesy of J.P. Iannotti, M.D., Ph.D.)

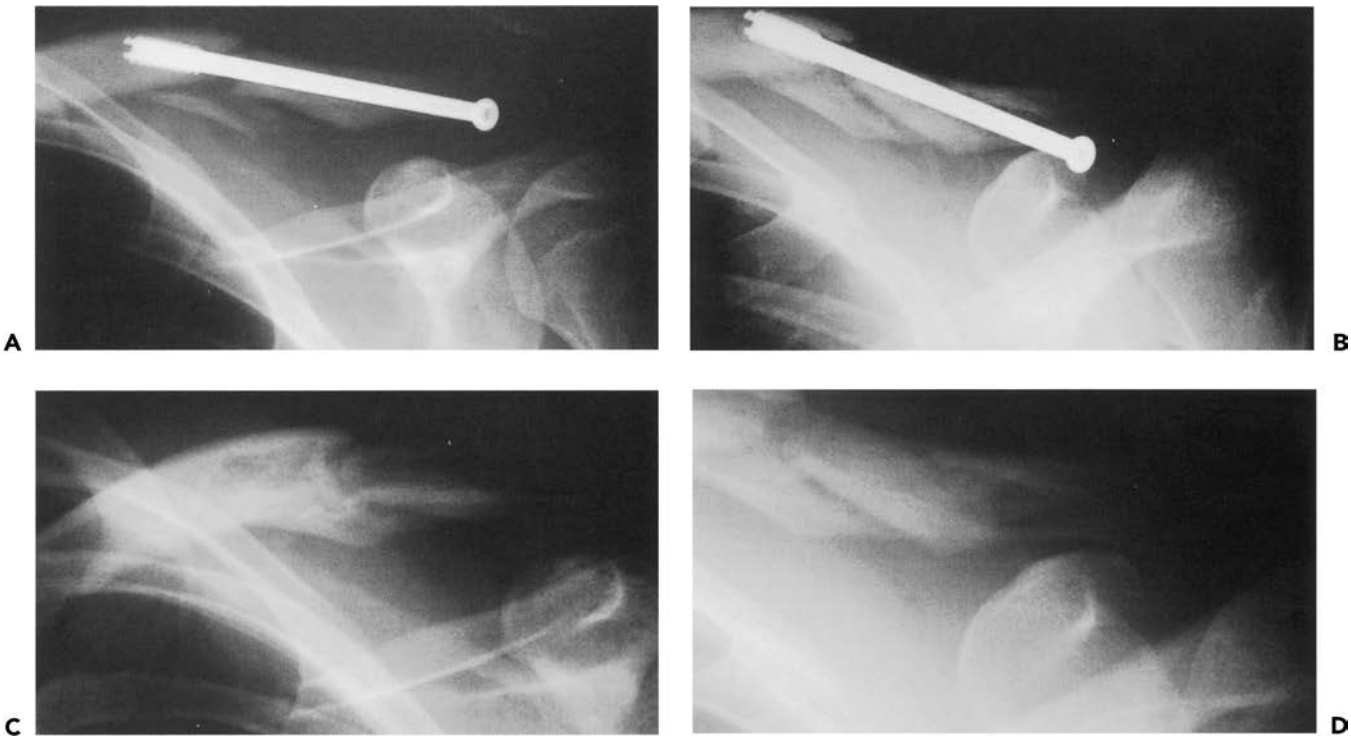


Figure 38-9 **(A)** Immediate postoperative x-rays after open reduction and internal fixation of a comminuted, midshaft, clavicle fracture. **(B)** Four months postoperatively, the osteomyelitis was treated by retention of the hardware and intravenous antibiotics until the fracture healed. **(C,D)** The hardware was removed and the clavicle was débrided, which was followed by another 6 weeks of intravenous antibiotics. The patient has a healed painless clavicle with full function and no signs of infection. (Courtesy of J.P. Iannotti, M.D., Ph.D.)

It has been well established that suture in the wound increases the susceptibility of host tissue to infection. Elek and Conen showed that 7.5×10^6 staphylococci were required to induce an intradermal infection, whereas a bacterial inoculation count of only 300 was needed to produce a similar infection in the presence of silk suture.[42] In 1984, Chu and Williams[20] studied the attachment of bacteria to 10 different suture materials. They concluded that the number of adherent bacteria was dependent on the type of suture material, the specific bacteria, and the duration of contact. Physical configuration (monofilament vs. braid multifilament) and surface area of the suture play a role in bacterial adhesion, but their chemical structure and coating may be even more important factors. These investigators also found that *S. aureus* adhered to sutures more than *E. coli*, and that the adherence is a dynamic process. Thus, in short, suture materials should be treated like any other foreign body. They may be left in place if not grossly contaminated at the time of initial débridement, but may very well require removal to eradicate the infection.

After removal of the foreign material, several additional surgical débridements are usually needed at 2- to 5-day intervals for complete eradication of all compromised tissues. Reconstruction of the defect with bone graft and/or local or microvascular soft tissue transfer may be performed after the wound is culture negative.

Management of Infected Arthroplasty

Fortunately, the incidence of infection after total shoulder arthroplasty is exceedingly low.[101] However, when infected arthroplasty does occur, it represents a potentially devastating complication that is often difficult to manage. Like other joint replacement surgery, septic shoulder arthroplasty can occur early or late. Once the diagnosis is established, the treatment should follow the same principles as the larger joints. Intraoperative frozen sections may be valuable in helping to establish or confirm the diagnosis. As demonstrated by Lonner et al.[94] in revision hip and knee arthroplasties, 10 or more polymorphonuclear cells per high-power field were predictive of infection. Early infections may be managed with wound exploration, irrigation, débridement, wound closure, and antibiotics.[101,154] The implant may be left in place if the components are well fixed.[60] Wirth and Rockwood recommend that early infections with Gram-negative organisms and all late infections be treated with removal of the prosthesis and all cement.[154] Thorough débridement of granulation and scar tissue is also required, combined with 6 weeks of parenteral antibiotics.

Historically, the majority of late infections of shoulder implants have been treated with prosthesis removal and resection arthroplasty. Pain relief usually is achieved in one-half to two-thirds of patients,[25] but resection arthroplasty is often complicated by severely limited ROM as well as loss of strength.[25,135] Recent literature has supported the use of staged exchange arthroplasty with a temporary antibiotic cement spacer as an effective means of treating late infected implants.[24,82,115,127] Jerosch and Schneppenheim[82] and Seitz and Damacen[127] were both able to eradicate infection and achieve good function in each series of eight patients treated with local débridement, intravenous antibiotics, and staged reimplantation with an interim antibiotic cement spacer. Temporary use of an antibiotic spacer offers the advantage of a stable shoulder joint and local therapy with antibiotics while preserving some passive and active motion.[82] Other treatment options include antibiotic suppression, arthrodesis, and amputation.

AUTHORS' PREFERRED METHODS OF TREATMENT

Our generalized treatment algorithm for intraarticular shoulder infections, based on the University of Pennsylvania classification system, is outlined in Fig. 38-10.

Hematogenous, Isolated Subacromial Septic Bursitis

If the infection is limited to the bursa, we prefer needle aspiration of the space, organism-specific antibiotics, and careful observation. Complete evacuation of bacteria and debris is not as critical in this case, because the integrity of the articular cartilage is not in jeopardy.[88] Arthroscopic or open débridement should be performed if the process fails to clear rapidly. Patients who have a protracted course, concomitant osteomyelitis, or glenohumeral sepsis will require surgical drainage and débridement.[17] If the integrity of the rotator cuff or the involvement of the glenohumeral joint is in question, an MRI is obtained for preoperative evaluation.

Hematogenous, Isolated Pyarthrosis of the Glenohumeral Joint

In aspiration-confirmed, nongonococcal hematogenous pyarthrosis of the glenohumeral joint, we initiate appropriate intravenous antibiotic treatment and prefer surgical irrigation and débridement with the arthroscope (Table 38-5). After induction of anesthesia, the patient is placed in the beach-chair position. Preparation and draping are performed in the usual sterile manner for

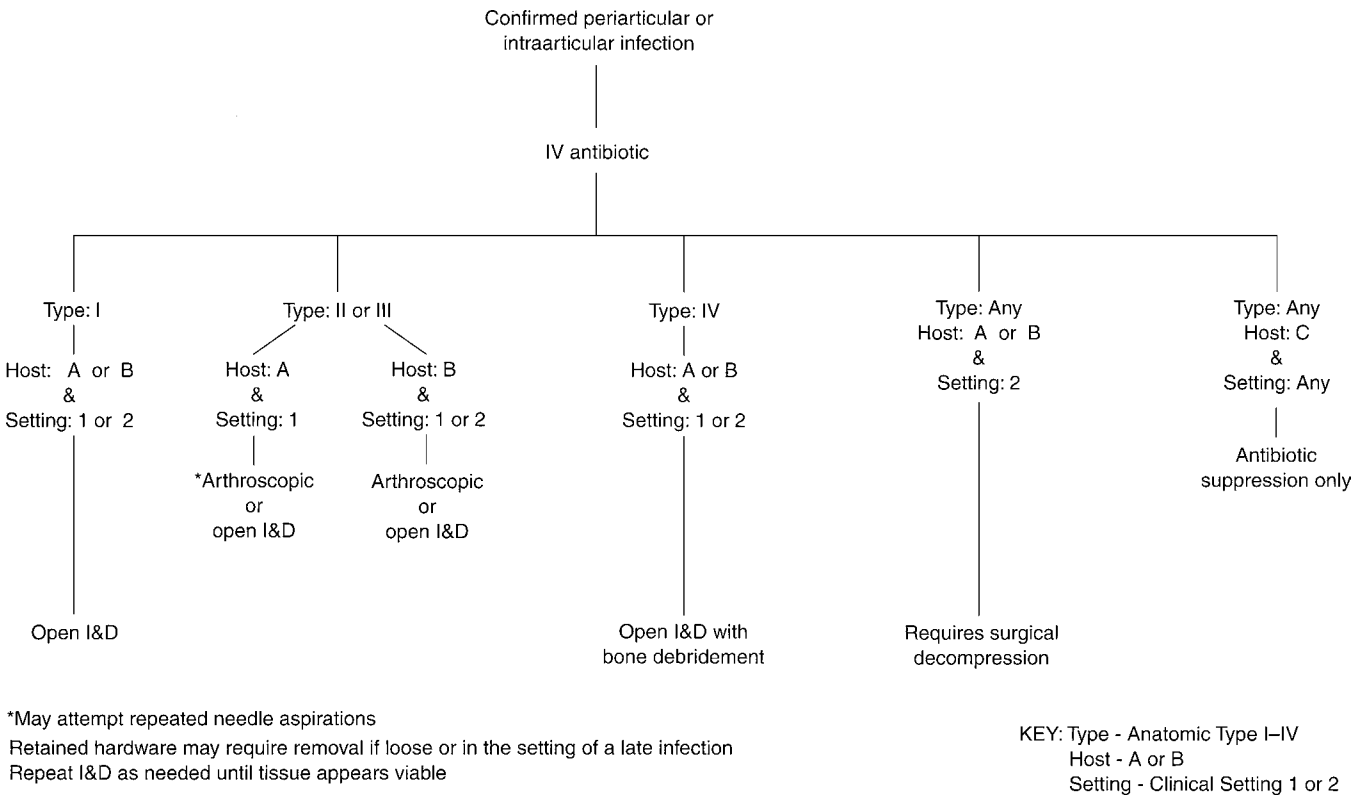


Figure 38-10 The authors' treatment algorithm for pyarthrosis, based on the University of Pennsylvania classification system for septic arthritis (see Table 38-3).

shoulder surgery. The posterior portal is made 2 cm inferior and 2 cm medial to the posterolateral edge of the acromion, in line with the posterior axillary fold. A trocar is then inserted, aiming in the direction of the coracoid. After confirming intraarticular placement by back flow of joint fluid, the arthroscope is inserted, and the joint is inspected. A second (anterior) portal is then made by first advancing a spinal needle from a location halfway between the coracoid process and the anterolateral acromion into the joint under direct arthroscopic visualization. The needle is removed, and a cannula with a trocar is inserted, following the same direction as the needle. Tissue for cultures and histology is obtained. The two portals are used for ingress and egress of irrigation fluid. After irrigation, the entire joint is once again inspected for signs of rotator cuff or articular damage and retained purulent material.

We include a limited open procedure to explore the biceps tendon when there is tenderness along the biceps or if the infection is caused by an aggressive organism. This can also be done by opening the biceps sheath by arthroscopic technique from the subacromial space. When the cuff is intact the biceps tendon and groove should be marked with a spinal needle, which can then be used to locate the sheath from the bursal side of the rotator cuff. Opening the biceps sheath ensures that loculated pockets of purulent material or soft tissue abscesses have not developed from organisms tracking down the bicipital groove. In all cases of sepsis of the glenohumeral joint the subacromial space should be evaluated and irrigated after the glenohumeral joint is thoroughly cleaned.

We recommend débridement via open arthrotomy if the surgeon is less experienced in shoulder arthroscopy. We prefer the deltopectoral approach and opening the rotator

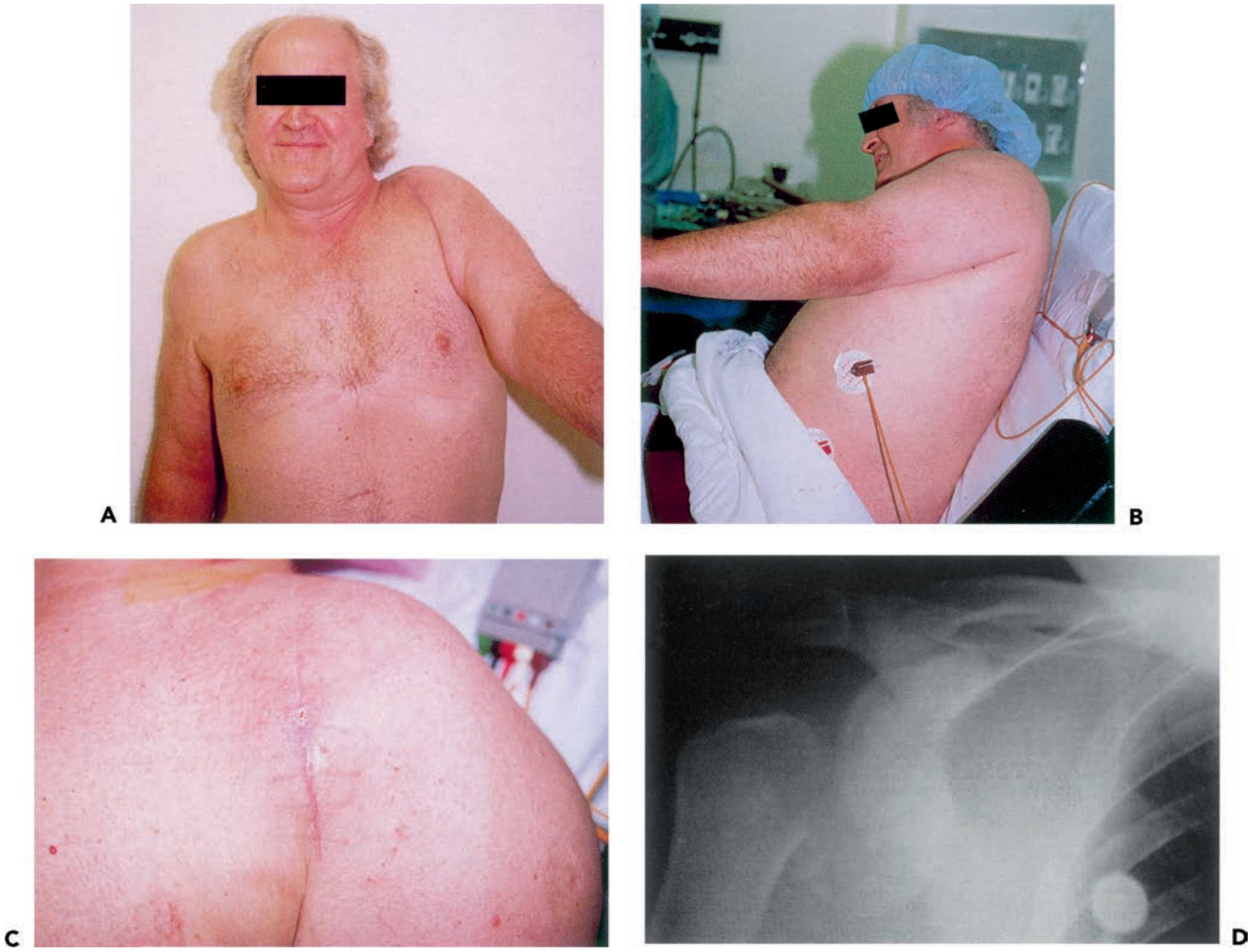
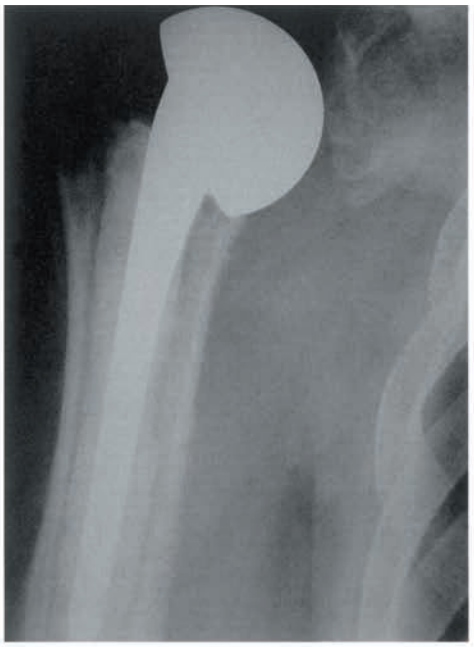


Figure 38-11 **(A–D)** Chronic osteomyelitis after hemiarthroplasty for treatment of an acute four-part fracture. The patient had chronic wound drainage and a painful arm and was initially treated with oral antibiotics. **(E)** The postoperative x-rays showed a detached greater tuberosity and a loose cemented hemiarthroplasty.



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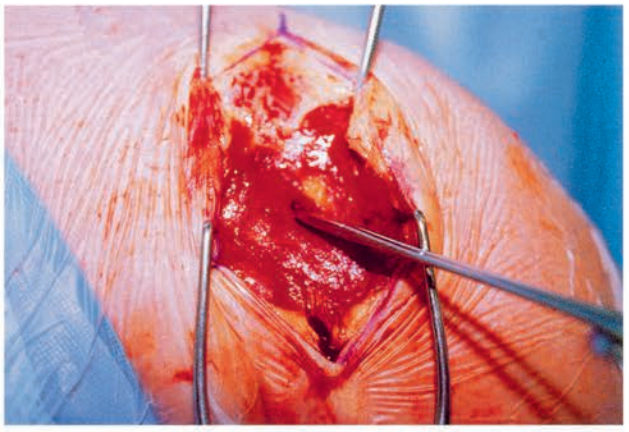

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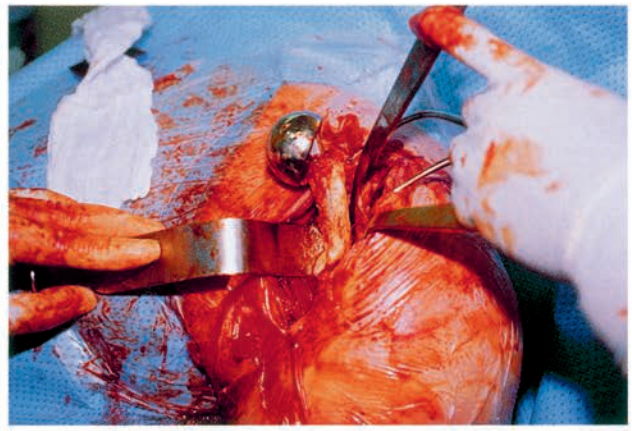

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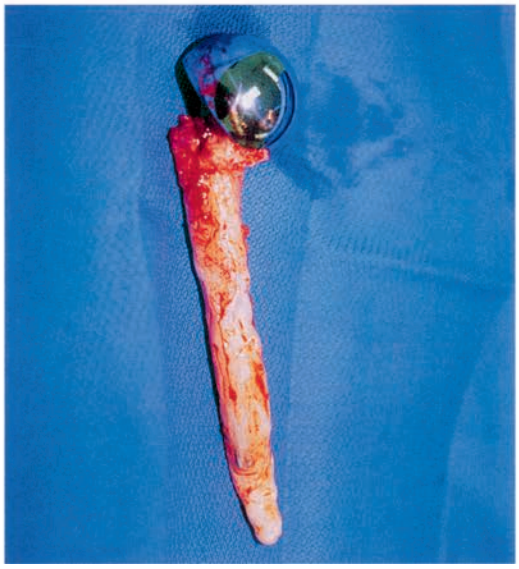

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Figure 38-11 (*continued*) **(F–H)** Intraoperative photographs showing a chronic sinus tract to the prosthetic stem that was loose and easily removed with the entire cement mantle.

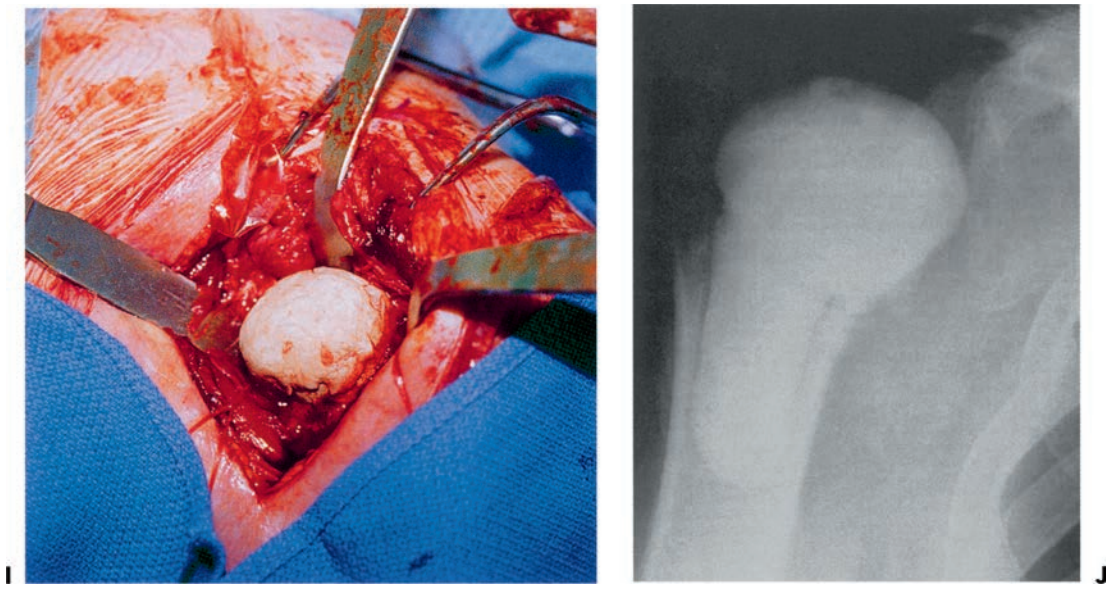


Figure 38-11 (*continued*) **(I,J)** A tobramycin cement spacer was placed for 8 months, during which time the patient was on antibiotics. The erythrocyte sedimentation rate was monitored monthly. It decreased from 92 preoperatively to 20 before revision surgery, 8 months after débridement of the infected prosthesis. At the time of revision, the spacer was removed, a total shoulder arthroplasty was performed with tobramycin cement, and the tuberosity was mobilized and internally fixed to the proximal humeral shaft. (Courtesy of J.P. Iannotti, M.D., Ph.D.)

interval. After irrigation and débridement, a closed suction drain is placed in the joint and the interval is closed with a monofilament, absorbable suture. A second drain can be placed in the soft tissue.

Medical management with antibiotics and repeated aspirations should be reserved for less virulent infections that are diagnosed early and for patients who are medically unable to tolerate surgery. At the time of this writing, we believe that percutaneous catheter drainage is yet to be proven to offer any additional benefits.

Postoperatively, analgesics are adjusted so that the patient can participate in pendulum or passive ROM exercises within 24 hours of surgery. A sling is used for support, as needed, during ambulation. An NSAID is started after a positive clinical response to the antibiotic regimen is established, usually after 4 or 5 days.

Postoperative Wound Infection Complicating Fracture Stabilization or Rotator Cuff Repair

Although some investigators believe there is a role for arthroscopy in postoperative wound infections, we prefer an open wound débridement, including arthrotomy, as needed. The extent of débridement must be individualized for each patient. However, if the infection is caught early, at our first débridement we attempt to irrigate the wound thoroughly, débride any granulation tissue and glycocalyx, and allow the internal fixation device or cuff repair suture to remain. The patient is returned to the operating room at 2- to 3-day intervals for redébridement as needed. Closed suction drainage is used. Every attempt is made to avoid leaving the wound open or changing packing at the bedside because of the risk of introducing a hospital-based resistant organism. Antibiotic beads are inserted if the wound size will allow. Removal of compromised suture and fracture fixation hardware is performed as soon as it becomes obvious that the organism is not responding. We emphasize avoidance of electrocautery for dissection and meticulous soft tissue and bone débridement to have adequate host material at the time of reconstruction or definitive wound closure. If a rotator cuff repair is found to be disrupted during the initial débridement, definitive repair is delayed until the final irrigation and débridement to optimize long-term results.

If the deltoid was taken down for exposure, care must be taken to repair it to prevent postoperative dehiscence. At the time of the definitive procedure, we suture the superficial and deep deltoid fascia directly to bone. We prefer to use monofilament, absorbable sutures in an interrupted manner for this closure.

Postoperative mobilization is restricted to pendulum exercises, depending on the stability of the cuff or fracture repair. In the case of a fracture involving the proximal humerus, fracture healing takes precedence over joint motion.

In extreme circumstances of patient noncompliance or if the soft tissue envelope is severely damaged by the infection or multiple procedures, a shoulder spica cast is used for wound protection. Soft tissue immobilization is maintained in the position of least tension to the repair. In our

opinion, a spica cast is better immobilization than an abduction pillow, because there is less tendency for the arm to shift.

If the nonabsorbable rotator cuff suture has been allowed to remain in situ, parenteral, organism-specific antibiotics are delivered for 6 weeks. In the case of retained fracture fixation hardware, oral antibiotics are continued after the 6 weeks of parenteral antibiotics until the fracture is clinically and roentgenographically healed. After confirmation that healing has indeed occurred, the hardware is removed if the infection persists.

Infection Complicating Hemi- and Total Joint Arthroplasty

Surgical wound infections that develop during or immediately after the index hospitalization are treated aggressively with débridement and irrigation, rather than expectantly, because of the underlying biomaterial.

For late deep infection, our preferred treatment is staged débridement and reimplantation, if possible. An antibiotic-impregnated cement spacer is utilized to facilitate the definitive reconstruction (Figs. 38-11 and 38-12).

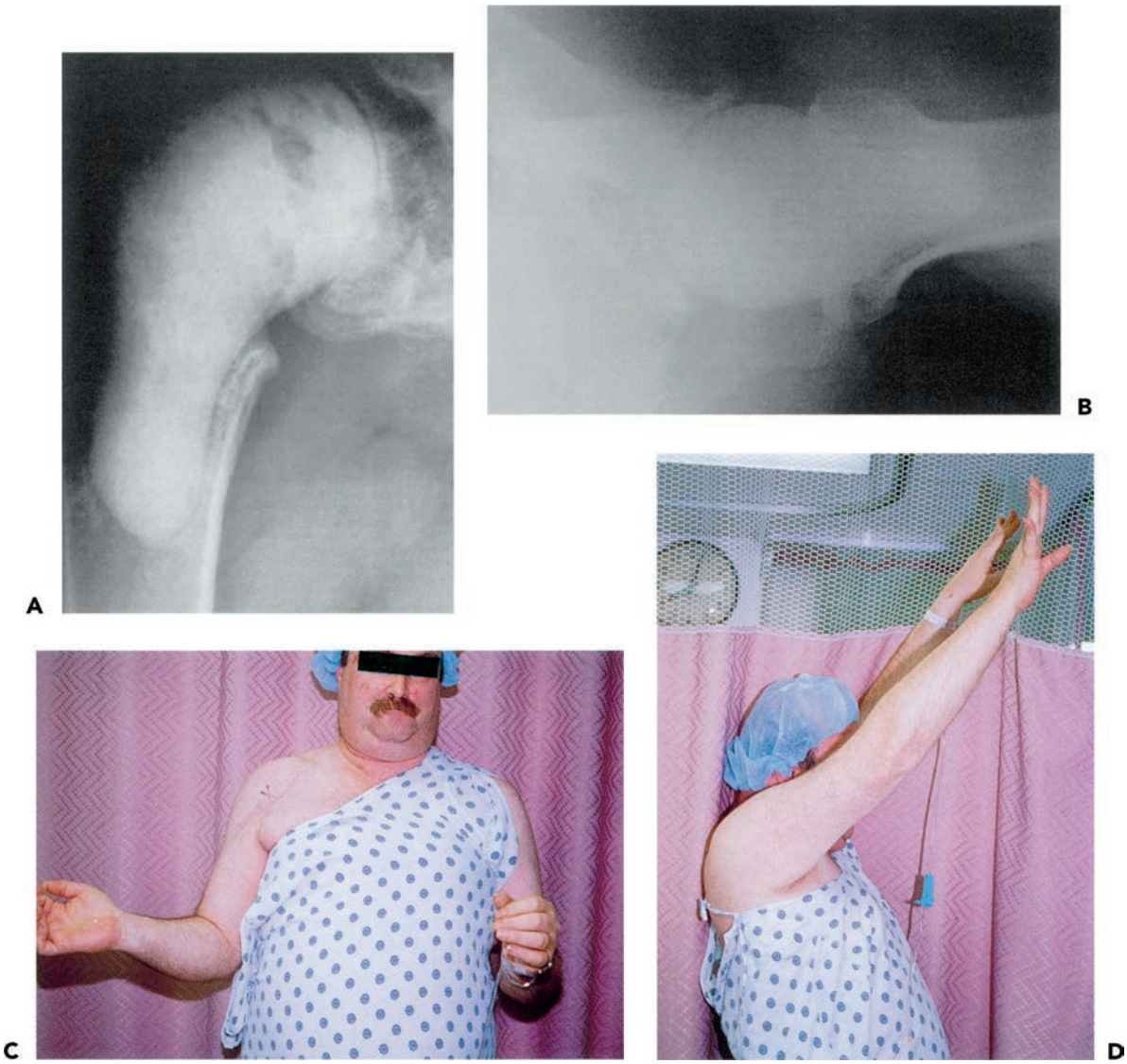


Figure 38-12 **(A,B)** Cement spacer used on the right shoulder for treatment of a late hematogenous seeding of a hemiarthroplasty, placed 18 months prior to infection. The hemiarthroplasty was placed for arthropathy secondary to an excessively tight repair for recurrent instability of the right shoulder, 18 years before the index arthroplasty. The cement spacer maintains the soft tissue envelope for later prosthetic conversion. In some patients, remarkable shoulder function can be maintained with a spacer in place. **(C,D)** This patient had to be called back to have the spacer replaced with a hemiarthroplasty because of progressive glenoid erosion. He maintained excellent function and minimal pain with the spacer 1 year after débridement of the infected joint. His left, uninfected shoulder had less function, due to prior shoulder surgery for treatment of recurrent dislocation 18 years previously. (Courtesy of J.P. Iannotti, M.D, Ph.D.)

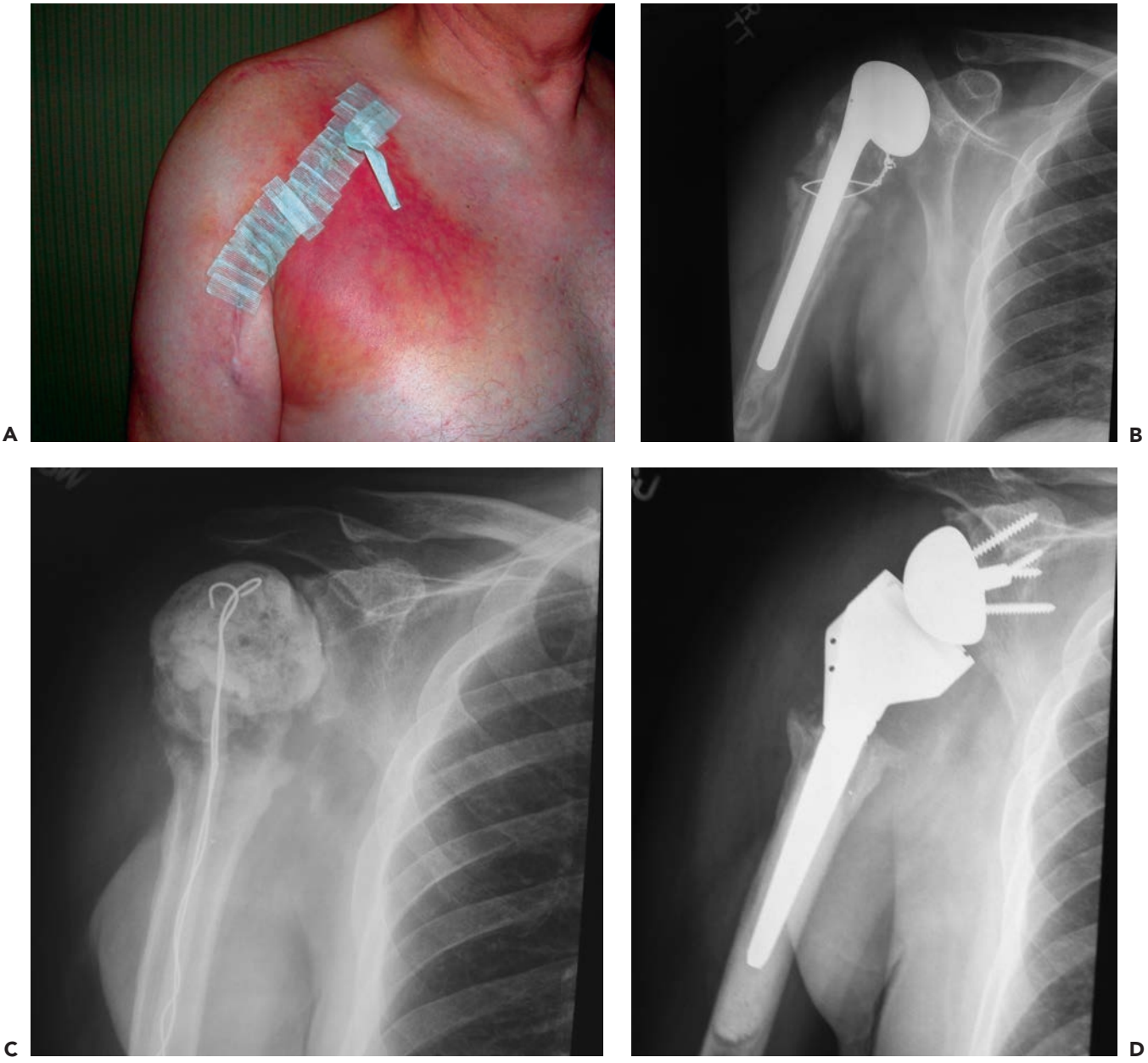
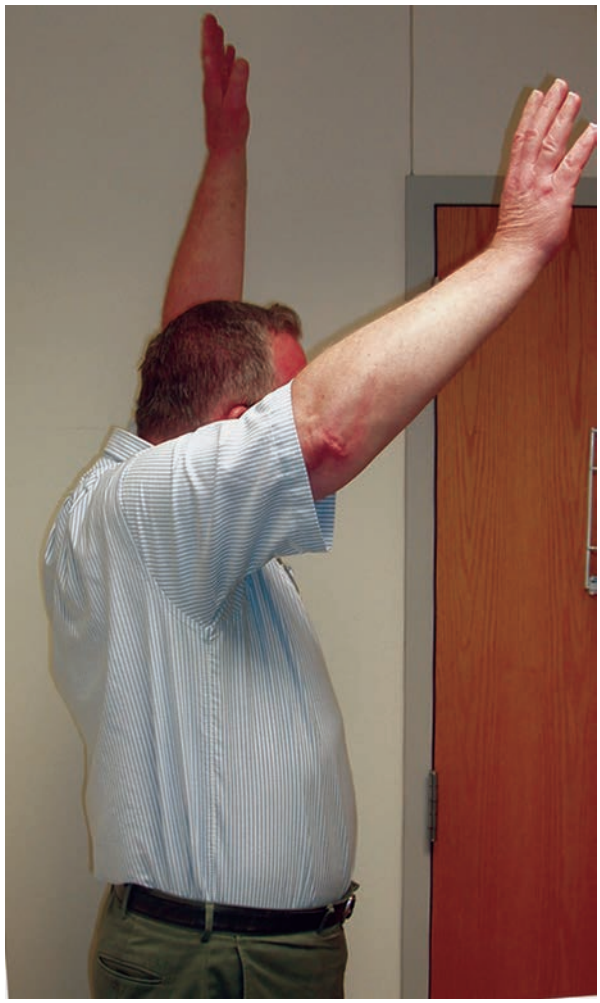


Figure 38-13 **(A)** Clinical infection with Gram-positive cocci (*Staphylococcus aureus*) with **(B)** severe loosening and osteolysis of the humeral component. Patient had hemiarthroplasty performed for proximal humeral fracture with severe rotator cuff and bone loss (tuberosities). **(C)** This patient was treated with a two-stage revision with a vancomycin spacer for 4 months (6 weeks of IV antibiotics). **(D)** Second-stage revision was with a Delta (DePuy, Johnson and Johnson) reverse total shoulder arthroplasty.

The spacer is usually hand formed to make a smooth spherical shape approximately the size of the humeral head and is made stable by placing at least several centimeters of the cement within the proximal humeral shaft. The cement is allowed to cure in situ and is continuously irrigated to minimize tissue damage from the heat generated from the large amount of cement. We make all attempts to repair the rotator cuff to bone, and if there is a displaced tuberosity with an attached rotator cuff, fix the bone to the humeral shaft or cement mantel. We use a #2 monofilament absorbable suture. The goal of this repair of fixation is to maintain as much length of the soft tissues or tuberosity for later reconstruction. After placement of the spacer we encourage the patient to use the shoulder and upper extremity for all waist- and chest-level activities of daily living. We start them on pendulum exercises during the first week after surgery. Revision arthroplasty is performed at least 3 months after all



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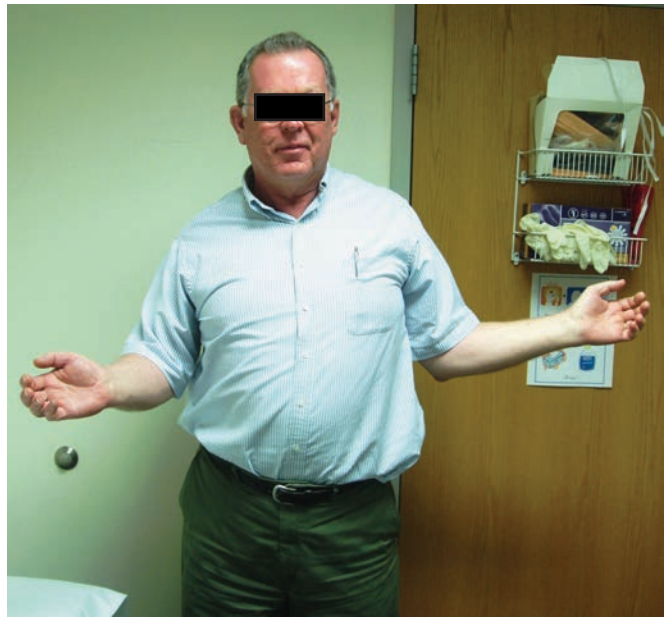

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Figure 38-13 (*continued*) Six months after surgery the patient had good shoulder function **(E–G)**. (Courtesy of Joseph P. Iannotti, M.D., Ph.D.)

antibiotics are stopped and the erythrocyte sedimentation rate, C-reactive protein levels, joint aspirate, and clinical findings do not demonstrate signs of infection. At the time of surgery intraoperative cultures and frozen sections are obtained and intraoperative antibiotics are then given. When all signs of infection are absent, the prosthetic is implanted with antibiotic cement that is most suitable to the bacteria originally isolated. In cases where the rotator cuff is intact with good deltoid function, an unconstrained hemi- or total shoulder arthroplasty is performed. In cases with major bone or cuff deficiency, a reverse shoulder prosthetic is preferred so long as there is sufficient bone to allow for secure placement of the metaglene (Fig. 38-13). In some cases of severe glenoid deficiency where the infection is cleared, the antibiotic spacer is removed, the glenoid is grafted, and hemiarthroplasty is performed. If the patient is dissatisfied with the function, the infection remains cleared 1 year after being off all antibiotics, and there is incorporation of the bone graft material, the hemiarthroplasty can be converted to a reverse shoulder replacement. The editors have used this protocol for two-stage reconstruction for infected arthroplasty in over 40 patients with over a 90% success in the resolution of active signs of infection. Results of surgery have been quite variable with both unconstrained and constrained arthroplasty but are more reliable for active shoulder-level elevation with the reverse prosthetic.

If a reimplantation cannot be performed, arthrodesis is the next option, if possible, because it yields better function than resection arthroplasty. This is indicated when the functional deficits of a resection arthroplasty are unacceptable to the patient. In these cases it is almost always necessary to use a vascularized fibula to achieve an arthrodesis and compensate for the massive bone loss present in most cases on both the humeral and glenoid sides. However, because there is usually extensive bone loss and this patient population is usually elderly, resection arthroplasty is performed more commonly than

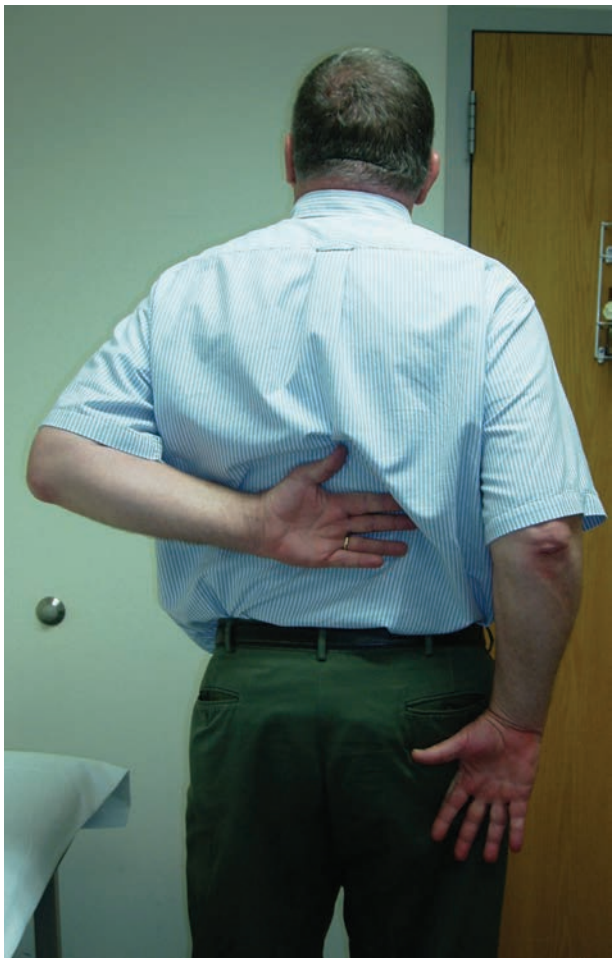

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Figure 38-13 (*continued*)

fusion. Deep late infection with a nonaggressive organism in an elderly patient is suppressed with antibiotics, if possible.

PROGNOSIS

The prognosis for septic arthritis of the shoulder joint is highly dependent on prompt diagnosis, the nature of the infecting organism, and the patient's immune status. Most patients will not have the typical signs and symptoms of a septic joint, and the laboratory studies may be equivocal. For these reasons, many are initially diagnosed as having bursitis or tendonitis, and the correct diagnosis may not be made for up to 6 months.[90] It is therefore imperative that the evaluating physician have a high index of suspicion and that, if the diagnosis is entertained, the glenohumeral joint be aspirated to rule out pyarthrosis.

Gelberman et al. reported satisfactory results in 8 of 10 patients when treatment was initiated within 4 weeks of the onset of symptoms.[50] Master et al.[99] found that treatment delays of greater than 1 month resulted in therapeutic failures, with persistent infection and poor function. However, other investigators had worse outcomes in their series. Leslie et al.,[90] who defined a poor result as the death of the patient or the absence of active motion of the glenohumeral joint, found that 9 of the 10 poor results were in patients who had been diagnosed with septic shoulder within 4 weeks. Ward and Goldner[151] found that all unsuccessfully treated patients had symptoms for more than 1 week. The discrepancy between the results cited in the above series may be due to a number of factors, such as the aggressiveness of the infecting organisms or the method of treatment. However, it is clear that delay in treatment results in worse prognosis.

Outcomes following treatment of infected shoulder arthroplasties can be promising.[30,78,127,135] In a series of 42 patients (49 infected prostheses) treated with a variety of methods, 71% were able to clear their infection.[30] Although only 6 of 10 patients treated with two-stage prosthetic revision showed no signs of persistent infection (mean follow-up of 34 months), the authors advocated staged reimplantation as the best compromise between eradication of infection and preservation of function. In another series of eight cases followed for an average of 4.8 years, all patients achieved pain relief with no recurrence of infection following staged reimplantation using an interim antibiotic spacer.[127] All eight patients demonstrated a significant increase in their functional capacity; however, each also reported a decrease in motion and strength compared to their normal side. Equally good outcomes have been reported by Ince et al. following one-stage exchange for infected prostheses.[78] After a mean follow-up of 5.8 years, none of the nine patients in their series showed signs of recurrent infection. In each of these studies, early and aggressive treatment of periprosthetic infections was associated with more favorable outcomes compared to delayed treatment.

FUTURE DIRECTIONS

Exciting work is ongoing with reference to the prevention of infection associated with orthopedic biomaterials by coating fracture fixation hardware with antiseptics prior to implantation. Work in the animal model has shown a significant reduction in infection using chlorhexidine and chloroxylenol.[35]

Evaluation of patients with potential joint sepsis is currently imperfect at best. Emerging diagnostic techniques are focusing on detecting a specific host response to infection. Utilizing microarray technology, it has been shown that the synovial fluid leukocytes have a genomic profile that is specific for infection.[39] This genomic technology may lead to simple tests in the future that identify

infection, and may even distinguish bacterial species involved.

Even after the bacteria have been destroyed, the enzymes released from the damaged tissue and by the bacteria and the host defense mechanisms can further degrade articular cartilage proteoglycan. Preliminary work by Cohen at al. has shown that the use of an adenosine A_{2a} receptor agonist reduces joint inflammation and glycosaminoglycan loss in rabbits with septic arthritis, and may be helpful in preventing the early degradative effects of joint sepsis.[27]

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Rehabilitation and Outcome Measures

IX

39 General Techniques of Shoulder Rehabilitation

Martin J. Kelley *Brian G. Leggin* *Kenneth J. Rogers*

PRINCIPLES 1235

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SUMMARY 1260

Shoulder rehabilitation is critical to the recovery of patients with shoulder symptoms and following trauma or surgery. Controversy exists regarding how much rehabilitation is required relative to frequency and supervision. Prudent rehabilitation is extremely valuable if a team approach is utilized and open communication fostered. The team is composed of the physician/surgeon, the therapist, and the patient. If the physician, therapist, and patient are actively interacting and fulfilling their responsibilities, the team, individually and as a whole, will be successful.

The components of shoulder rehabilitation and the rationale for intervention will be discussed in this chapter.

PRINCIPLES

Principles of rehabilitation remain constant regardless of whether the patient's status is postinjury or postsurgery. First, pain is always respected and rarely encouraged. Increased symptoms following the introduction of a new exercise or technique indicate the need for reevaluation of the recent intervention. Therefore, to assess the efficacy of treatment interventions, modalities or exercises are introduced one or two at a time. Only then can the clinician identify effective or aggravating treatment interventions.

Second, performing a thorough examination to identify pathology, tissue reactivity, impairments, and functional deficits and disability is critical when developing an effective intervention program. However, constant reevaluation of response to treatment and tissue reactivity is equally essential. Manual therapy by stretching and strengthening allows continuous reevaluation while treating. The real-time feedback gained by manual contact allows immediate

modification of techniques and exercise or correction of the patient's movement patterns. Manual contact is essential in discovering subtle and sometimes blatant problems that only arise with repetitive motion or those that manifest with fatigue.

Tissue reactivity is determined by subjective reporting and objective testing and qualifies the irritability of the involved structures (mild, moderate, or severe). The patient's reactivity becomes the guide for program progression. An iatrogenic plateau is sometimes reached, which essentially means that further recovery is hampered by the very same rehabilitation process that initially allowed some degree of recovery.[49] The plateau is characterized by stagnating mild tissue reactivity. A short but full rest from exercise, lasting 4 to 10 days, is recommended, and typically the tissue recovers, as does the patient's progress.

Third, exercise and techniques are advanced in motion, resistance, and movement planes based on the symptoms and functional demands of the patient. Range of motion (ROM) is progressed from passive ROM, to active assisted ROM, to active ROM. Movement may start in gravity reduced positions and progress to antigravity positions. Strengthening is progressed from isometrics, to short arc, to full arc. Submaximal resistance is initially used, advancing to maximal resistance. Progression of shoulder position or motion plane during exercise is from the nonprovocative to the provocative. Slow controlled exercise using elastic bands, free weights, and manual resistance is progressed to higher-velocity uncontrolled exercise using Plyoballs. Stress and safety zones are identified in Fig. 39-1. The plane of the scapula (POS) lies in the center of the safety zone. These nonprovocative and provocative positions and planes vary based on pathology or surgery, but most commonly the least provocative position is somewhere between 20 and 55 degrees of scapular plane abduction.

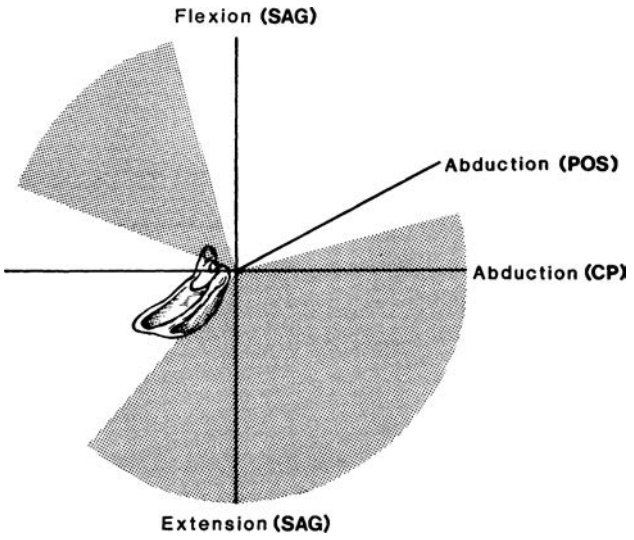


Figure 39-1 Stress (*shaded*) and safety (*white*) zones.

TABLE 39-1
PRINCIPLES OF TREATMENT

No pain—all gain
Constant reevaluation
Utilize the plane of the scapula
Rest when needed
Scapular muscle integration and balance
Nonprovocative → provocative
Submaximal → maximal
Short arc → full arc
Individualize program based on symptoms and functional demands

Keeping the humerus below 55 degrees prevents subacromial impingement, whereas avoiding full adduction minimizes excessive tension across the supraspinatus–coracohumeral complex and/or capsuloligamentous complex (CLC). The POS is advantageous in exercise performance for several reasons. It is an inherently stable position for the glenohumeral joint when working with a patient with instability because tensile and torsional stresses to the rotator cuff and capsuloligamentous–labral complex (CLLC) are minimized during exercise.[45] Additionally, strengthening in the POS utilizes the optimal alignment of the supraspinatus and deltoid.[45] Progression to provocative end-range positions may be required. Consider the throwing athlete who must function at 90 degrees of abduction and full external rotation. This position not only stresses the CLLC and twists the rotator cuff, but it can also cause supraspinatus impingement against the posterior glenoid rim.[18,41,109]

Fourth, balanced scapular muscle function is constantly integrated into all dynamic exercises from simple rotator cuff exercises to sport-specific manual resistance, plyometrics, and possibly isokinetics. We have found this principle to be extremely effective in all patients, especially the high-demand overhead athlete or laborer.

Lastly, the patient's rehabilitation program is individualized based on reactivity, hyperelasticity/hypoelasticity, personality, demand, goals, criteria met, surgical concerns, and complications (Table 39-1).

REHABILITATION GOALS

This section will discuss the goals of rehabilitation, the problems that those goals address, and the rationale for the goals (Table 39-2).

Patient Education

The goal and ultimate success of patient education cannot be underestimated, particularly in light of our current managed care environment. The patient must understand the

TABLE 39-2
GOALS OF REHABILITATION

Goal	Problem	Rationale
Patient education	Patient lacks knowledge	Prevent injury or reinjury
Antiinflammation/ reduce pain	Inflammation exists	Decrease prostaglandin production and reduce swelling
Facilitate collagen healing	Disruption of collagen	Gentle stress assist in collagen orientation and strength
Strengthen muscle tendon unit	Muscle weakness/dysfunction	Improve neuromuscular stabilization and control
Improve ROM	Adhesion/synovitis/contracture	Improve CLC pliability and muscle tendon length
Optimize proprioception	Impaired mechanoreceptor input	Optimize mechanoreceptor input and sensitivity
Endurance	Fatigue	Reduce fatigue effect and vulnerability for injury

CLC, capsuloligamentous complex; ROM, range of motion.

basic concepts of their pathology so instruction regarding modifying irritating activities is understood. Postoperative precautions must be stressed and understood. Patients are instructed in how to promote healing through positioning (Fig. 39-2) and the use of heat and ice. When appropriate, they are instructed in the proper performance of relatively pain-free exercises and, if they are to be discharged, indications for progression of the program. Without effective patient education, the surgical intervention and the rehabilitative process may be jeopardized and slowed, respectively.

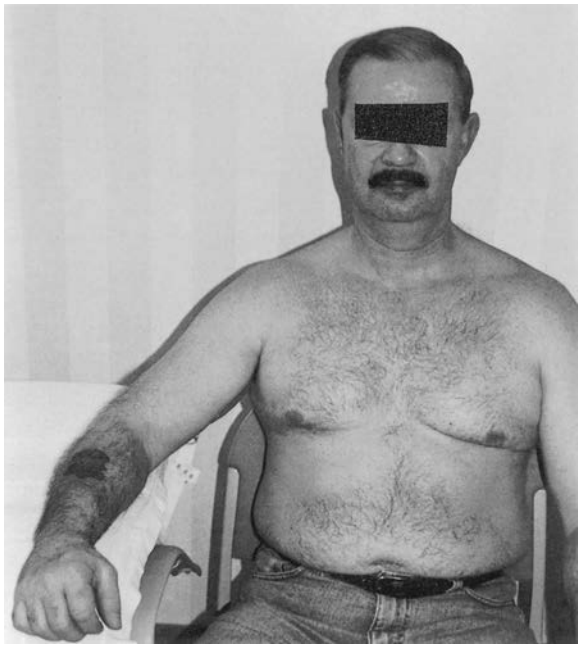


Figure 39-2 Placing the patient in a supported, neutral rotation position reduces pain and discourages contracture.

Antiinflammation and Pain Reduction

Modalities and their antiinflammatory and pain-reducing effects will be discussed later in this chapter. Stopping and modifying aggravating activities is a key to pain reduction. This includes the cessation popular "workout" exercises (i.e., bench press, overhead press, or those prescribed by a health care provider). As rehabilitation specialists, we must recognize that "Mother Nature" is a powerful healer and it is best to work with her.

Strength

Muscular strength refers to the capacity for active tension development by a muscle.[96] This is a simplistic definition of a very complex concept. Strengthening exercises can produce healthy or destructive effects. During exercise, the tension required to produce strength gains must not exceed the tissue/fixation threshold developed in a healing tendon or tendon–bone interface. Strengthening progression is based on time from injury, surgery, and tissue reactivity. In the early phases of strengthening, less activity, and therefore less tension, may protect healing soft tissue. Wise et al.[111] and Gaunt and Uhl[23] investigated what exercises create low rotator cuff activation. More needs to be known about tissue tension overload, particularly due to the reported high retear rate in the literature following large rotator cuff repairs.[8,22,25,27,70]

Multiple factors influence muscle strength. Strength gains occur by two basic types of neuromuscular changes: increased motor unit recruitment and hypertrophy.[56] Significant strength gains have been shown to occur in the absence of hypertrophy due to improved motor unit recruitment and synchronization; in other words, the

muscle learns the task. Appropriately loading a muscle will result in hypertrophy or increased muscle mass because a muscle with a larger cross-sectional area can create more tension. Although improving muscle mass may be a goal in rehabilitation, the concept of enhancing synchronization and the number of motor units recruited—neuromuscular system training—provides the rationale for why most individuals with rotator cuff tendinopathy and glenohumeral instability are placed on a rotator cuff and scapular muscle strengthening program.

The ultimate objective is to teach the glenohumeral and scapulothoracic dynamic stabilizers to work repetitively, in an efficiently integrated manner, throughout the ROM while centering the humeral head on the glenoid.[19,54,84,87]

Proprioception

Proprioception is defined as the sensation of dynamic joint motion and joint position.[66] It is mediated by numerous specialized mechanoreceptors in the muscle, tendons, capsule, and ligaments.[10,66] Stimulation of the mechanoreceptors initiates a neurologic feedback loop, causing protective stabilizing muscle function. When the CLC mechanoreceptors are injured or the CLC mechanism is disrupted, as seen in a traumatic dislocation causing a Bankart lesion, the proprioceptive feedback loop is compromised, resulting in reinjury. Altered proprioception has been demonstrated in individuals with known instability, and was restored following surgical anterior stabilization and rehabilitation.[65] Glousman et al.[30] showed decreased activity of critical muscles during throwing in pitchers with known instability. Rehabilitation must address the effect of a compromised neuromuscular mechanism. When appropriate, strengthening exercises emphasizing muscular "control" must be performed at different degrees of functional elevation, enhancing the neuromuscular system and improving proprioception.

Range of Motion

ROM or stretching exercises are designed to maintain or improve soft tissue pliability and prevent adhesions while protecting the injured or surgically repaired tissues. Exercise categories are typically passive, active assisted, and active. Passive range of motion (PROM) exercise is performed when muscle activity is to be avoided, as in the early postoperative phase of rotator cuff repair. Prophylactic stretching exercises that improve tissue pliability and length, in the absence of pain or pathology, are also considered passive. Active assisted range of motion (AAROM) exercise requires the unaffected arm, therapist, or equipment to assist movement. AAROM allows appropriate muscle activation through a ROM while preventing tissue reinjury or disruption. Active motion is allowed when the affected or repaired tissues at risk have been given adequate healing time or when the patient can independently move the segment without increasing symptoms. The targeted tissue and rationale for ROM exercises may be different, depending on whether the patient's status is postoperative or postinjury, whether the patient has developed a contracture, or whether a pre- and postworkout flexibility program is required.

Facilitate Collagen Healing

The importance of positively influencing connective tissue healing is overshadowed by the other effects of maintaining joint motion and strengthening. Any disruption of connective tissue is followed by collagen healing and remodeling. Gentle ROM exercise and submaximal strengthening in the appropriate time frame following injury or surgery stresses collagen promoting improved tissue alignment and tensile strength.[56]

Endurance

Muscular endurance is the ability of a muscle or muscle group to repeatedly contract and sustain tension over a period of time. Strength and endurance may not always correlate well to one another because an individual may be strong but may have poor endurance. This scenario relates to the worker who must lift a 2-lb drill gun overhead; he may have the strength to do it 1 or 10 times, but can he do it 100 or 200 times in a short period of time? We must not forget that a multitude of muscles from the lower extremities, trunk, and shoulder girdle contract when the arm is lifted. The weak link relative to poor endurance may be the scapular muscles or the trunk extensors, and not the rotator cuff. However, the resulting injury may be manifested at the rotator cuff. Electromyographic (EMG) studies in the industrial population and in athletes have shown that experience, not strength, may have a greater influence on fatigue. Sigholm et al.[97] found that experienced welders were able to avoid fatigue better than their less experienced counterparts. Gowan et al.[31] found that professional pitchers activated all the muscles of the rotator cuff, except the subscapularis, less than amateur pitchers. Endurance and possibly "strength" may relate to the efficient use of the shoulder complex through adaptation or better mechanics. The therapist must work not only on the endurance of individual muscles but also on efficient movement patterns. Another aspect of endurance that cannot be overlooked is cardiovascular endurance. One would expect that the better-conditioned individual is less prone to injury.

INTERVENTION

Modalities

In addition to terminating aggravating activities, modalities have been advocated for antiinflammation and pain

reduction. Much of the evidence for their use is anecdotal or a product of questionable experimental design. A variety of modalities are used during the rehabilitation process. The four main objectives for using modalities are to (a) reduce inflammation, (b) reduce pain, (c) improve motion, and (d) promote muscle reeducation. Most modalities achieve their physiologic effect by the transfer of thermal energy. These modalities include hot packs, cold packs, ultrasonography (ultrasound), diathermy, and infrared. Another mode of achieving a physiologic effect is through the use of electric stimulation. Electric stimulation modalities include transcutaneous electric nerve stimulation (TENS), as well as low-voltage, high-voltage, interferential, and direct current stimulation.

Heat Modalities

Heat therapy may be used to reduce muscle guarding and increase tissue temperature to assist with stretching.[28] Hot packs can be applied before or during ROM exercises. Application of moist heat in conjunction with stretching has shown improvement in muscle elasticity.[40] This may occur by a reduction of muscle viscosity and neuromuscular-mediated relaxation.[95]

Ultrasound

Ultrasound is a deep heating modality used during the subacute and chronic injury stage. Ultrasound is proposed to increase tissue temperature and may be helpful the healing process. The application of ultrasound is typically applied in the 1-MHz and 3-MHz ranges and is dependent on tissue depth and osseous location. Appropriate application of ultrasound is dependent on the type and depth of the soft tissue, injury status, and goal of therapy. Ultrasound has both thermal and mechanical properties that are thought to increase vascularization and elasticity of the tissue.[28] These latter proposed effects of ultrasonography may be the most effective in conditioning the tissue for stretching and strengthening. Muscle regeneration morphology was not established with the application of pulsed ultrasound on rat gastrocnemius muscle.[88] However, this may be dependent on the dosage and mode of the ultrasound application. Ultrasound applied in vitro demonstrated an age-dependent response of a patellar tendon with a correlation between decreased tendon metabolic activity and aging.[2] Recently, Gursel et al.[32] reported that ultrasound, compared with sham ultrasound, brings no further benefit when applied in addition to other physical therapy interventions in the management of soft tissue disorders of the shoulder.

The technique of phonophoresis can be used to drive antiinflammatory medication, usually either hydrocortisone or dexamethasone, through the skin and into the inflamed tissue. Whether this is truly achieved is still controversial.[7] Tissue depth due to adipose and muscle tissue; hydration state of the individual; and application procedures may counteract the effectiveness of phonophoresis.

Cryotherapy

Cryotherapy may be used for pain and swelling control.[1,99,102] Cold is best applied by a conforming pack. Cold has been shown to be beneficial in reducing pain in the postoperative shoulder patient.[99,102] A sterile circulation pad can be applied over the surgical dressing. A pump in the water reservoir circulates the water through the pad at a temperature of 50°. The application time can be from 1 to 3 hours. Singh et al.[99] completed a prospective, randomized investigation on the efficacy of continuous cryotherapy for both open and arthroscopic procedures of the shoulder. Cryotherapy controlled pain by decreasing the severity and frequency while allowing a more normal sleep pattern. In addition, the cryotherapy patients reported less pain during the rehabilitation exercises when compared with the age-matched control group.

Electric Stimulation

Electric stimulation therapies can be employed to reduce pain,[72,100] increase circulation, increase muscle activity for reeducation or strength effects,[100]and reduce spasm (by fatigue).

There are numerous variations of electric stimulation devices based on the type of current delivered (AC or DC), the operating voltage (less than 100 V is called low voltage vs. high voltage, which operates at greater than 500 V), the available frequency (interferential utilizes 4,000 Hz), and wavelength. Different waveforms are utilized dependent on the goals of the treatment program. The following are the different types of electric stimulation that can be utilized: transcutaneous electrical nerve stimulation (TENS), high-voltage pulsed stimulation (HVPS), interferential stimulation (IFS), microcurrent electrical stimulation (MET), and neuromuscular electrical stimulation (NMES).

Shoulder Slings and Braces

Shoulder slings and braces are utilized for protection after injury or surgery and for injury prevention during functional/sports activities. They may vary in design from a simple sling used following an injury to an elaborate multijointed device used to place the shoulder in a specific position to protect soft tissues postsurgically. Use of shoulder slings/braces is specific to acute postinjury/surgery and return to activity/play.

Acute Postinjury

Initial management, regardless of most injuries, requires the patient be placed in a sling for comfort and tissue

protection. The sling needs to support the weight of the arm and prevent motion. Most commonly the arm is placed in adduction and internal rotation providing adequate positioning. Time spent in a sling is variable and dependent upon the injury. Typically, patients are instructed to wean themselves from sling use to prevent a frozen shoulder. The type and length of immobilization is variable, but longer immobilization has not been considered important especially in reducing the recurrence rate of instability.[55,98] However, recent research has shown anterior labrum healing and a lower recurrence rate following traumatic anterior dislocation when an external rotation brace was used.[37] Immobilization in external rotation causes subscapularis tendon tightening across the anterior joint, providing a "counterforce" and promoting the anatomic position of the glenoid labrum.[39,78] Positioning the arm into internal rotation slackens the anterior structures, separating the labrum from the glenoid rim, thereby discouraging healing.[38,39] Itoi et al.[37]compared instability recurrence rates in 80 patients immobilized in external rotation and internal rotation within 3 days of the traumatic anterior dislocation. They found a significant difference between the recurrence rates for those in the external rotation group, 10%, and the internal rotation group, 30%. For individuals under 29 years of age, the recurrence rate for external rotation was 11.5% and internal rotation was 37.5%. Prefabricated braces immobilizing the arm in external rotation are now available (Fig. 39-3).

Fracture

Typically, a sling is used to immobilize a patient following a proximal humeral head fracture. Koval et al.[60] found that those individuals who started motion early, before 14 days, had better results than those who started after 14 days following a minimally or nondisplaced proximal humeral head fracture. Koch et al.[57] demonstrated that 95% of patients obtained good and excellent results and no significant treatment morbidity when Sarmiento bracing was used for humeral shaft fractures. Patients with trauma just to the humerus and no polytrauma had less of a chance for a nonunion and potential need for surgical fixation.

A figure-eight brace has been typically used to stabilize clavicle fractures. The figure-eight brace needs to be applied with proper tension so the shoulder girdle is pulled posteriorly. Overtightening or contact with bony prominences could potentially cause skin irritation and, if left untreated, skin breakdown and infection.

Postoperative

Modifications of the simple sling have been made relative to material used for durability, breathability, and added padding or bolsters. The addition of a small bolster placing the arm in slight abduction may have positive effects not only on comfort but on reducing tissue tension (Fig. 39-4). Several studies have shown that passive tension is increased and can propagate supraspinatus retear when the arm is placed at the side following a rotator cuff repair.[90,113] If a rotator cuff repair is performed "under tension," an abduction pillow may be used to reduce the tension and improve healing. It is not used, however, unless the tendon can be repaired with the arm at the side.

Airplane splints or abduction braces may be used following certain surgical procedures like a latissimus dorsi transfer. The "gunslinger brace" can be used following posterior capsulorraphy to maintain the arm in external rotation and prevent inferior translation of the humeral head.

Return to Activity/Play

Braces used for returning a patient to functional or athletic activity usually address glenohumeral joint instability.

Figure 39-3 Posttraumatic anterior dislocation brace that maintains the arm in slight external rotation.

Figure 39-4 The Ultrasling brace (Donjoy) places the arm in slight abduction.

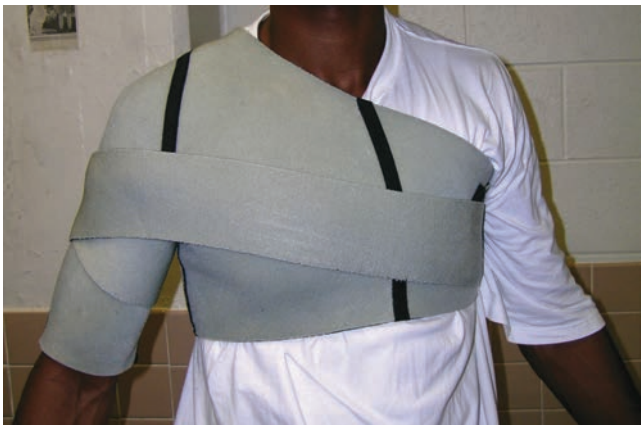


Figure 39-5 The SAWA ( ) brace for use in athletes with anterior instability but who require overhead mobility.

Choosing what brace to use is based upon the activity/sport and anticipated stresses to which the shoulder will be exposed. A person returning to a collision sport or heavy labor may need to wear a shoulder stabilizer brace. These braces are designed prevent instability events by limiting shoulder flexion, abduction, and external rotation. In addition to restricting motion, neoprene shoulder stabilizer braces have been shown to improve external rotation joint repositioning in patients with known instability.[17] The Dennison-Duke Wyre brace (C.D. Denison Orthopaedic Alliance Co., Baltimore, MD) is commonly used for individuals participating in football and hockey. Other braces like the Sulley or SAWA (BRACE International, Scottsdale, AZ) allow greater excursion during overhead sports like basketball (Fig. 39-5). Buss et al. reported on 27 athletes from various sports who returned to play the same season as having an instability event. Nineteen of the athletes (70%) wore either a Dennison-Duke Wyre or Sulley brace, depending on their sport. All participated in a postevent rehabilitation program of ROM exercises and strengthening. They found that 37% of the athletes suffered at least one other instability episode, while 59% were event free.

The stabilizer brace needs to fit snugly around the body so that good support is provided. The straps can be customized to control the range of motion to protect the joint but also to allow a functional range of motion for the activity being performed. The straps can be adjusted to primarily prevent anterior or posterior translation. The stabilizers typically fit under or on the protective gear. In some sports, the stabilizer may need modifications to prevent injury to another (i.e., wrestling).

Taping

Taping has been proposed for numerous effects about the shoulder, including improving stabilization, reducing pain, inhibiting abnormal muscle activity, improving muscle activation, and improving posture. Unfortunately, no

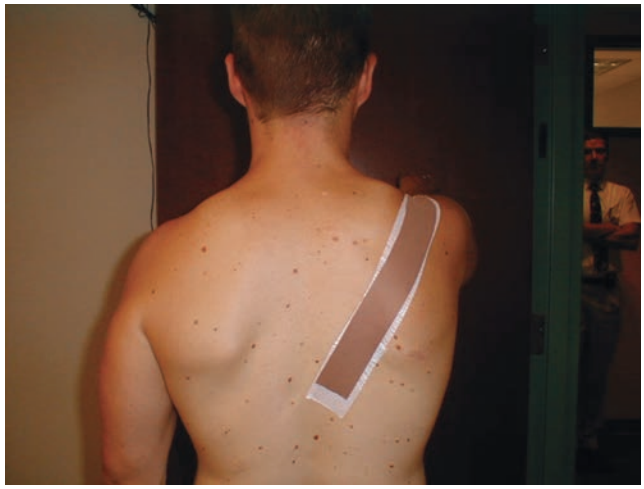


Figure 39-6 Taping across the medial scapular border provides tactile feedback to the patient with motor control dyskinesia.

studies have shown taping to be efficacious. Lewis et al.[68] studied 60 patients with subacromial impingement who were assessed before and after scapular and thoracic taping. They found taping was effective in changing static scapular position, improving patient range of motion, and increasing range of elevation before symptoms were experienced but did not reduce the symptoms associated with subacromial impingement. The authors use taping sparingly and usually for the tactile feedback effect. As Lewis et al. reported, scapular position can be changed with taping techniques, and this provides approximately 24 to 48 hours of appropriate postural feedback. Taping has also been found to be influential when working with patients with scapular motor control dyskinesia. Placing tape from the acromion to approximately the T10 level provides tactile feedback as the medial border displaces into the tape (Fig. 39-6). The negative aspects of taping are skin breakdown with repetitive use and the need to have someone else available to apply the tape.

Frequency and Supervision

The question of supervision level and optimal visit frequency is difficult to determine. Ideally, high-quality care needs to be maintained while containing cost. The level of supervision and frequency decision should be based upon the pathology, patient reactivity level, cognitive status, whether the patient is postoperative, and surgeon/ physician preference.

Rockwood believed in surgeon directed "ortho" therapy. Following a rotator cuff repair, Leffert and Rowe[63] advocated only home exercise instruction, while others advocated supervised therapy for 40 to 60 visits.[21,24] We examined 75 patients 7 to 9 days following arthroscopic rotator cuff repair and determined the supervision need for therapy over the subsequent 5 weeks based on passive ROM,

reactivity level, cognitive status, and ability to correctly perform the exercises. Those placed on a home program could correctly perform the exercise and had an average passive range of motion of 117 degrees of forward elevation and external rotation of 27 degrees. Patients placed in supervised therapy had either a reactive presentation and less motion (PROM of 90 degrees of elevation and 21 degrees of external rotation) or could not correctly perform the exercises.[64]

Patients with primary frozen shoulder treated with a therapist-instructed home exercise program and intraarticular glenohumeral injection versus 12 supervised visits for 4 weeks were found to have no difference in 1-year outcomes.[15] Dierks et al.[20] followed 77 patients with idiopathic frozen shoulder for 24 months and determined that those who had "supervised neglect" (limited supervised visits) actually had better outcomes measured by the Constant score than those who had structured aggressive physical therapy. McClure et al.[75] found that treating patients with rotator cuff impingement one time a week for 6 weeks using a simple ROM and strengthening program with elastic bands significantly improved range of motion, strength and outcomes related to pain, patient satisfaction, and function. Ginn and Cohen [29] performed a randomized controlled trial on 66 patients with various diagnoses including rotator cuff tendinopathy, frozen shoulder, osteoarthritis, biceps tendinitis, and AC joint arthritis. One group was treated with physical therapy including stretching, strengthening, and neuromotor training for 4 to 10 visits in 1 month, while the second group received no treatment. Significant improvement was noted in the physical therapy–treated group in all outcomes. These studies come to no consensus on how often to treat patients with various shoulder pathologies, but recognize the need of therapist intervention.

Roddey et al.[93] compared home exercise instruction using videotape (therapist available for questions) versus therapist instruction in patients following arthroscopic rotator cuff repair. They found no difference in self-reported outcome measures or self-reported compliance when using the two methods of instruction.[92] Anderson et al.[3] reported on 43 subjects following arthroscopic subacromial decompression who were rehabilitated by supervised therapy versus a self-training leaflet. The Constant-Murley outcome score showed no significant difference between the two rehabilitation methods. Reo and Mercer[91] found that live and videotape instruction were significantly better than handouts alone for learning a shoulder exercise program.

We would advocate that frequency be determined by a team decision between the patient, the surgeon/physician, and a knowledgeable therapist. Our general philosophy favors therapist-instructed programs using intermittent visits as determined by the team and the patient presentation. Patients presenting with less pain, expected ROM and strength, and improving function are seen with less supervision. Those with unexpected protracted pain and stiffness resulting in limited function are seen more often in supervised therapy.

Range-of-Motion/Stretching Exercises

Shoulder ROM exercises can primarily isolate the glenohumeral joint structures as in external rotation stretching or can affect all the shoulder joints and spine as in shoulder elevation stretching. Indications and rationale vary for stretching based upon time from injury or surgery, tissue reactivity, contracture development, or prophylaxis. This section will discuss ROM exercises for the shoulder followed by stretches for the spine structures and scapular muscles. Three phases of stretching and indications for each will be discussed.

Phase I

Phase I exercises are indicated early postinjury or postsurgery. Tissues may be reactive or require protection from overload for 4 to 6 weeks. Painful, aggressive stretching should be avoided, recognizing that pain and reflexive muscle guarding are a normal response following trauma and surgery. Passive ROM exercises are initiated in "relatively" pain-free ranges or restricted ranges to improve or maintain ROM, provide gentle stress to healing collagen tissue,[56] and optimize the subacromial gliding mechanism. Neer et al.[80] found that those individuals who performed early passive motion reached their motion goals for discharge much sooner than did those not involved in a physical therapy program. The stretching effect can be enhanced by applying heat to the shoulder while stretching is performed.[58,59]

Phase I PROM exercises include pendulums, supine elevation, and external rotation (Fig. 39-7). Although we generally consider these exercises passive, research has shown muscle activation with PROM exercises.[112] Elevation stretching can be modified by performing the "chair" stretch or table stretch. The patient places the hand of the involved shoulder on the back of a chair, then steps backward and leans forward (Fig. 39-8). Knowledge of positional effect and tissue tension is necessary for safe and effective stretching. For example, stretching into external rotation with the arm in adduction may place the excessive tension on the rotator cuff repair. This position has been shown to significantly increase tensile load to the repair site and rotator cuff interval.[34,89,113] A less stressful position that will achieve the benefits of passive rotational stretching is placing the arm at 45 degrees in the POS (Fig. 39-7C).

Phase II

When appropriate time from injury or surgery (3 to 6 weeks) has passed and tissue reactivity reduces, phase II ROM

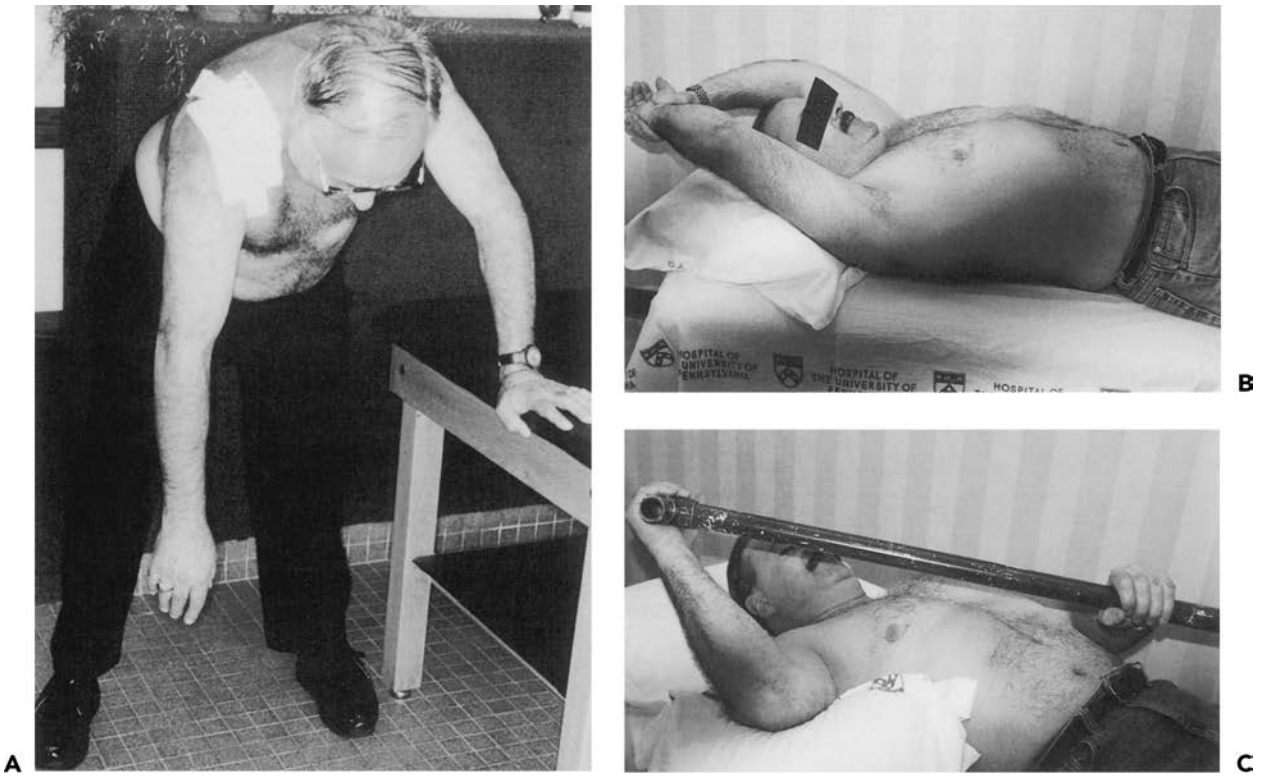


Figure 39-7 Phase I range-of-motion exercises. **(A)** Pendulum. **(B)** Passive elevation. **(C)** Passive external rotation in 45 degrees of elevation in the plane of the scapula.

exercises can be initiated. These include active assisted exercise, therefore muscle activation of the affected shoulder and placing the shoulder in provocative positions, including internal rotation and horizontal adduction, extension, and combination external rotation/elevation (Fig. 39-9). Connective tissue load and muscle activation can be altered based on the provided assistance, lever arm, position relative to gravity, or use of equipment to support the arm. Extension and internal rotation exercises are performed in an active assisted manner while the other two exercises are passive. Patients are instructed to remain "relatively pain free" and hold between 5 to 10 seconds. If the patient presents with greater reactivity the hold time may only be 1 second. Exercises are typically performed 20 times, three to six times per day.

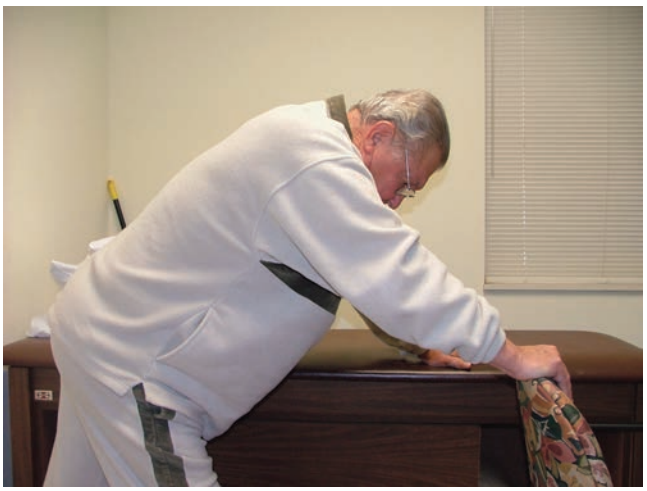


Figure 39-8 Chair stretch is alternative to supine passive elevation stretch.

Pulley use will be discussed separately since this device can be used in different phases and creates different effects. The pulley can be used in phase I for elevation stretching, but the patient must be instructed to relax. Elevation range of motion can be significantly influenced depending on whether shoulder internal versus external rotation is allowed, especially if external rotation is restricted. The patient will achieve greater elevation motion, with less pain, if taught to use the pulley with the affected elbow straight versus flexed (Fig. 39-10). A straight arm allows the arm to elevate with obligatory internal rotation (assuming elevation is occurring toward the sagittal plane). When elevating with the elbow flexed (in external rotation), the CLC is already taught and less motion is available for elevation. Terry et al.[104] suggested that elevation in the sagittal plane, while in external rotation, increased supraspinatus tendon tension. This would be undesirable following a rotator cuff repair.

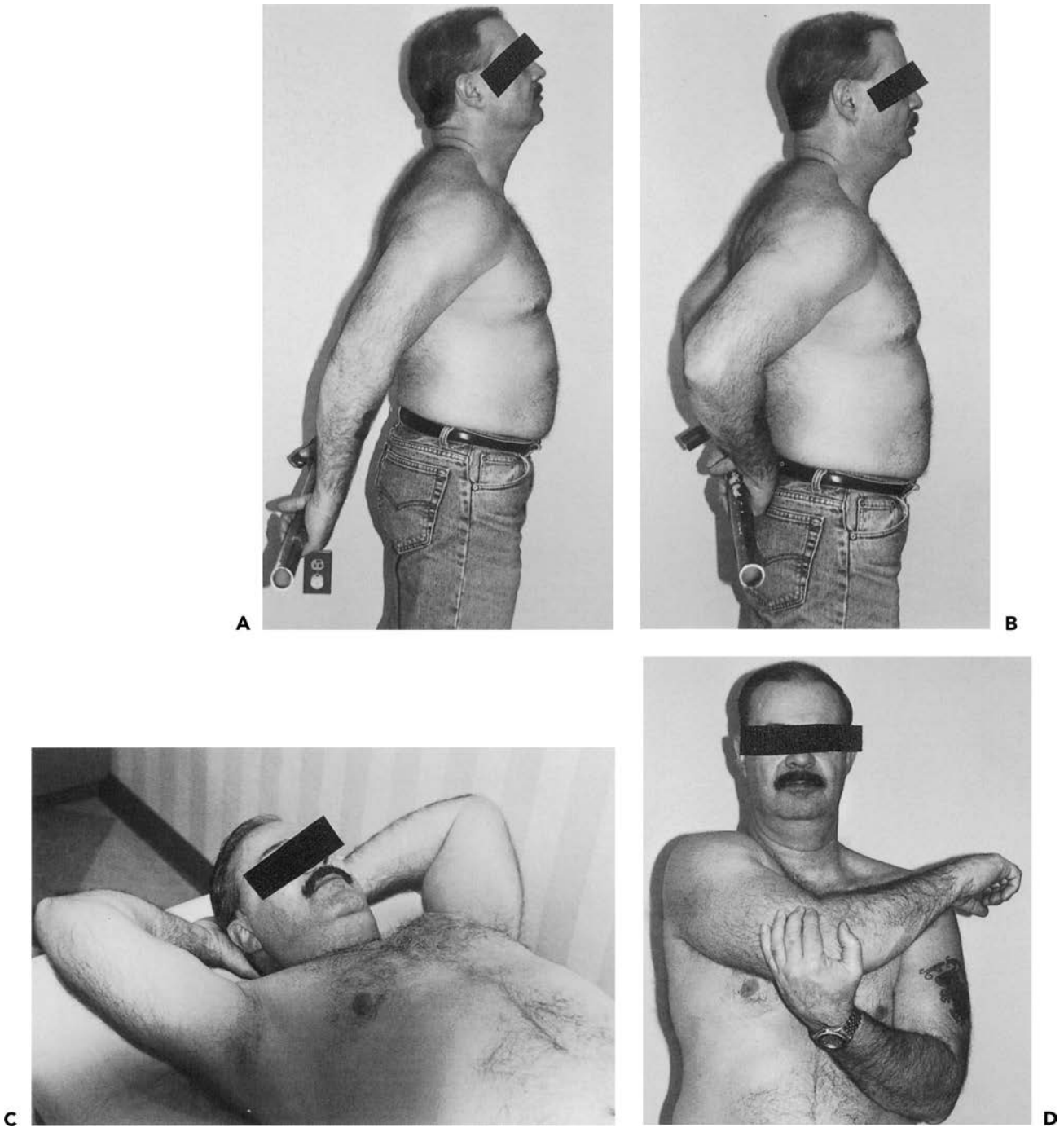


Figure 39-9 Phase II range-of-motion exercises. **(A)** Extension. **(B)** Internal rotation. **(C)** Modified elevation/external rotation. **(D)** Posterior capsule/cuff stretch.

The pulley can also be used in an active assisted manner by "deweighting" the affected extremity by assisting with the unaffected arm or by adding weights to the contralateral pulley handle. Prolonged stretching can be started when the patient can tolerate pain-free end-range positioning.

Although the pulley is an excellent way to gain elevation motion, an active assisted to active progression program is effective between 4 and 8 weeks. Exercises are moved from AAROM to AROM while the tissue integrity is protected from the increasing muscle–tendon tensile forces. McCann showed progressive shoulder muscle activation through Neer's three phases of rehabilitation.[74] Protecting tissue while advancing exercise intensity requires taking advantage of short lever arms, positioning relative to gravity, and

Figure 39-10 Pulley with the elbow straight **(A)** allows greater elevation than with the elbow flexed **(B).**

simple equipment such as a ball or slant board. Wise et al.[111] demonstrated that supported "closed chain" elevation exercises required less supraspinatus activity than an unsupported "open chain" exercise. These authors use the term "closed chain" to describe supported exercise even though the distal segment is moving (i.e., sliding the hand on a table or ball). However, this may be a liberal use of the term "closed chain," which typically refers to having the distal segment fixed while the proximal segment moves (i.e., pushup). We prefer the term "supported active assisted exercise" (SAAE) to describe activity during which the arm is supported by a surface, ball, or device that reduces the extremity weight, provides assistance, or allows use of antagonistic muscles. Fig. 39-11 shows a progression of AAROM and SAAE used to improve elevation ROM. The elevation progression is discussed further in the section on strengthening. These exercises produce low-demand (less than 15%) maximal voluntary contraction (MVC) of the rotator cuff.[23]

Contracture development is a common and sometimes inevitable problem, especially in the frozen shoulder patient and following proximal humeral fractures. Phases I and II stretches are very useful in maintaining or improving motion during contracture development. Two phases of contracture development will be discussed: painful and noninflammatory established contracture. Noninflammatory established contracture exists when the inflammatory stage is extinguished but the tissue is left fibrotic and shortened. However, end-range stretching is relatively pain free (this will be discussed in the next section). Painful contracture is a result of concomitant synovitis/inflammation or angiogenesis and fibrosis as commonly seen in primary and secondary frozen shoulder.[11,81,82] Unfortunately, many individuals with active inflammation cannot tolerate the required time for prolonged stretch to elongate tissue. Short duration hold (less than 10 seconds) ROM exercises performed by a patient having an active synovitis appear to minimize or prevent further loss of motion; however, one must be cautious about being too aggressive with stretching activities because they may aggravate symptoms.[20] Diercks[20] found that patients with frozen shoulder who were treated with "painful stretching" had significantly worse Constant scores at 1-year follow-up. Stretching should be performed near the painful end range and held for short periods of 1 to 10 seconds. Phases I and II stretching exercises form the core program, in addition to the use of a pulley. Static progressive or dynamic splint devices designed for the shoulder tend to be bulky and not tolerated

Figure 39-11 Unsupported active assisted range-of-motion **(A)** and supported active assisted exercise surface **(B),** on ball **(C),** and with Upper Extremity Ranger (Rehab Innovations, Inc., Omaha, NE) **(D).**

well in the painful stage. As tissue reactivity reduces and end-range stretching is tolerated, the length of hold time can be increased from 10 seconds to minutes.

Joint Mobilization

Joint mobilization is a manual stretching technique used to reduce pain and improve joint motion.[47,71] Depending on the degree of tissue reactivity or joint stiffness, grades I and II glenohumeral joint mobilization may assist in pain reduction and motion improvement when the motion is limited by pain. Joint mobilization is a technique in which one joint surface (i.e., the humeral head) is directionally translated relative to the other joint surface (i.e., the glenoid) (Fig. 39-12). Oscillations are performed at the end of the translation. Joint mobilization is usually graded from I to V, although grade V is considered a high-velocity thrust manipulation performed at end range. The proposed effect of joint mobilization is pain relief (grades I to II) and improved ROM by stretching capsuloligamentous tissue (grades III to V). Pain relief is thought to be mediated through a neurophysiologic mechanism beginning in the joint and soft tissue mechanoreceptors and nocioreceptors.[47,71]

By translating the articular surfaces, stimulation and/or accommodation of the receptors are initiated, which results in pain reduction. If the translation force is significant enough, as in grades III and IV, mechanical stretching of the tight soft tissue may result.[47,71]

Phase III

Stretching in the presence of noninflammatory established painless or relatively pain-free contracture is performed to mechanically elongate soft tissue that has lost its normal pliability. Connective tissue must be brought into a plastic range to cause deformation. Prolonged stretch is effective in creating tissue creep opposed to intense short-duration stretching.[58,69] Stretch intensity and time at end range can be increased when the patient presents with relatively painless contracture. Therefore, prolonged end-range positioning is encouraged. Holding a stick or positioning in

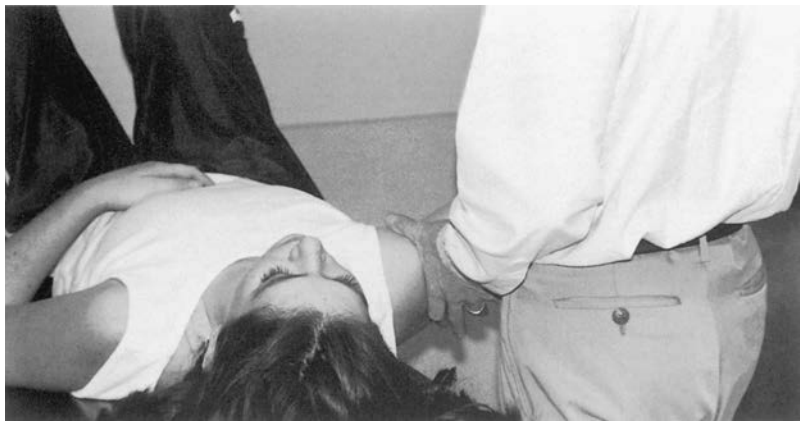


Figure 39-12 Joint mobilization.

elevation and external rotation for 1 to 5 minutes can be effective in assisting the remodeling process (Fig. 39-13). The total end-range time (TERT) concept is utilized. This means that the more time that is spent at end range, the greater the tissue lengthening effect is.[62,69,76] Elevation can be achieved by a door hang stretch (Fig. 39-14) and internal rotation with a towel. Static progressive splints may be indicated with this type of patient since end-range stretching is better tolerated.

Posterior capsule stretching is a recognized necessity in the overhead athlete's shoulder.[12,106,108] Harryman[33] demonstrated increased anterior and slight superior humeral head translation when the posterior capsule was imbricated. Posterior capsule tightness is thought to cause secondary compressive lesions of the rotator cuff.[33,73,77] Burkhart et al. found that excessive tightness of the posterior capsule in throwing athletes resulted in superior–posterior humeral head migration and excessive load on the long head of the biceps, causing the

Figure 39-13 Prolonged stretch into external rotation and abduction.

Figure 39-14 Door hang stretch.

Figure 39-15 Patient performing the "sleeper" stretch for posterior capsule.

superior labrum to "peel back" and tear.[13] A relationship exists between the development of a superior labrum anterior–posterior (SLAP) lesion and limited internal rotation at 90 degrees of abduction of 25 degrees compared to the other side.[14] Excessive loss of internal rotation at 90 degrees of abduction is known as glenohumeral internal rotation deficit (GIRD). The "sleeper" stretch is an effective way of self-stretching the posterior capsule[14] (Fig. 39-15). The patient's posterior capsule can be manually stretched while prone. The patient's hand is placed on their back while the therapist stabilizes the medial scapular border against the thorax and gently moves the elbow anteriorly. This is an aggressive stretch; therefore, the patient's symptoms must be monitored. The posterior capsule and cuff is also stretched in the cross body stretch. Holding a fixed object with the arm across the body can magnify this stretch by gently leaning away as the involved arm is pulled toward the chest and the body rotated toward the involved shoulder (Fig. 39-16).

Figure 39-16 Magnified posterior cuff stretch using body weight.

Prophylactic stretching to maintain or improve pliability of the musculotendinous cuff and CLC is reasonable for recreational or competitive athletes as well as for individuals in the occupational arena. One need only evaluate the external rotation motion of a professional baseball pitcher to recognize that excessive external rotation is required to generate torque to hurl a baseball at 95 miles per hour. The occupational athletes may require a certain degree of glenohumeral/trunk mobility to perform certain tasks. Much attention has been focused on regaining elasticity by stretching the glenohumeral posterior capsule.

Cervical/Thoracic Spine

The cervical and thoracic spine and related structures can negatively impact shoulder function because of poor orientation, dysfunction of any muscle that shares insertion between the spine and scapula, or intrinsic spinal pathology (i.e., radiculopathy).

Poor orientation or posture can influence scapular kinematics.[48,53,67] Keaetse et al.[48] found that a fixed thoracic kyphosis decreased scapular upward rotation and posterior tilt and resulted in increased superior scapular translation. Less shoulder elevation motion was found when the thoracic spine was in excessive kyphosis compared to the erect spine position.[48,67] Improving thoracic extension motion should be considered when treating a patient with shoulder pathology, especially the older individual or the athlete. Postural awareness education by using a lumbar roll when sitting helps to orient the thoracic and cervical spine toward anatomic position. Thoracic extension exercises are encouraged while sitting by performing active thoracic extension and scapular retraction. A passive thoracic stretch is achieved by having the patient lie supine on a towel roll or semi-rigid roll with it oriented perpendicular to the spine at approximately nipple level. The knees are bent and the arms placed over the head to increase the fulcrum effect. The patient breathes deeply to encourage costovertebral joint motion (Fig. 39-17). The patient progresses up to 2- to 5-minute stretches. A similar stretch can be achieved using a therapeutic ball. Trunk lateral bends and rotational stretching are performed to improve both trunk muscular and spinal mobility.

Cervical stretches are performed by side-bend and rotation exercises. Side bending elongates the contralateral trapezius, scalenes, and levator scapula. Direct stretching of the levator scapula is achieved by fixing the scapula (holding the leg of a chair with the involved arm) and combining cervical flexion with contralateral side bend and rotation.

Combination stretches are performed to affect spinal, scapular, or pectoral muscles. An excellent pectoral stretch that influences spinal and scapular position is performed with the patient lying supine on a towel roll or semi-rigid roll positioned inline with the thoracic spine. The patient

Figure 39-17 Thoracic extension stretch using a towel roll.

can lie on the floor or bench and place the arms in 90 to 110 degrees of abduction and relax. The roll enhances the pectoral stretch by "spreading" the chest. A specific pectoralis minor stretch is performed with the roll by actively retracting the scapula or by having the therapist manually place the scapula posteriorly and into elevation. Breathing can further accentuate the stretch by having the patient forcefully exhale as the scapular is displaced posterosuperiorly. Exhaling depresses the ribs and moves the pectoralis minor origin away from the posteriorly displaced coracoid insertion.

Stretching the medial scapular muscles can be difficult because of their oblique orientation. An effective stretch for the rhomboid, middle, and lower trapezius is performed with the patient sitting with the arms folded across the chest. The opposite arm to the side being stretched is placed over the other arm. The "stretching" hand is placed on the posterior shoulder region of the affected shoulder. The patient flexes the lumbar, thoracic, and cervical spine as the stretching hand pulls the shoulder to the opposite knee. Gentle breathing is encouraged (Fig. 39-18).

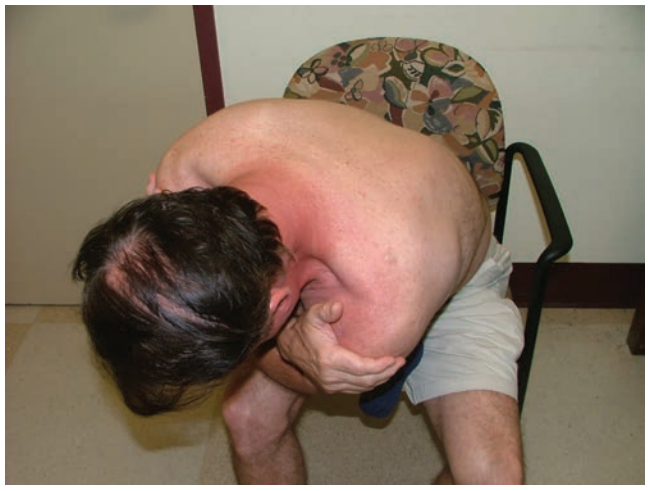


Figure 39-18 Left medial scapular muscle stretch.

Treatment of intrinsic cervical and thoracic pathology is outside of the scope of this chapter. However, when both shoulder and spinal pathology coexist, the authors use a valuable rule of thumb: Treat the spinal pathology first. Effective spinal intervention clarifies the true shoulder structure symptoms and can eliminate prolongation of symptoms.

Strengthening

Many investigators have performed EMG studies to evaluate muscle activity during exercise,[9,23,74,79,105,111] during athletic activity,[30,42,83,85,94] during occupational use,[46,97] and after nerve block and nerve palsy.[16,36,61] Although these studies give us useful insight regarding muscle function, the rehabilitation specialist must be careful about data interpretation and application to a patient with a pathologic condition. Typically these studies are interested in what exercises create the "greatest" activity; however, treating patients in the early postoperative/injury phase with significant pathology, tissue reactivity, or low demands may require exercises producing "less" activity to protect the soft tissues and minimize pain.[23,74,111]

Phase I

Phase I strengthening exercises are used in patients who are weak and/or in the early postoperative/injury period but can tolerate low-resistance exercise in nonprovocative positions. Strengthening may begin with AAROM and AROM exercises. Connective tissue load and muscle activation can be altered based upon lever arm, position relative to gravity, or the use of equipment to support the arm. The therapist and patient must appreciate a simple biomechanical principle when trying to achieve full active elevation: The patient must be able to lift the weight of the arm against gravity. Arm weight can be considerable, since some patients have very heavy upper extremities due to large bulk or excessive adipose tissue. Elevation requires moving the arm through the 90-degree arc where the arm weight and moment arm are greatest. Inability to lift the arm through the available range can be considered an active lag, analogous to the patient with a weak quadriceps who has an active extensor lag. Consider the patient who is 8 weeks post–rotator cuff repair and can only actively lift the arm to 70 degrees of elevation (due to weakness, not pain). Assuming repair and neurologic integrity, one can determine that the cuff/deltoid complex cannot centralize the humeral head and create the force to lift the arm weight. Exercise should emphasize elevation, but in positions that reduce the weight of the extremity and protect the tendon fixation. Gaunt and Uhl[23] and Wise et al.[111] examined EMG activity during supported and unsupported exercise. They provided us with evidence to guide our early strengthening phase when protecting the soft tissue structures in critical. Earlier in this chapter we

Figure 39-19 Progressive elevation strengthening using ball **(A),** wall tap **(B),** and supine elastic resistance **(C).**

discussed supported AAROM progression, but as the exercise difficulty is progressed, strengthening is achieved. Fig. 39-19 shows an elevation progression demonstrating increased demand on the rotator cuff.[23,111] This principle is also used to increase load on other muscle groups (i.e., external rotators).

Another simple but effective manner of increasing muscle–tendon demand during elevation is "gatching" (Fig. 39-20).[6] This technique uses an elevating surface to change the patient's orientation to gravity, thus increasing the weight of the arm. Gatching can be combined with isometric isolation of muscle groups as one elevates. For example, a ball can be squeezed (increasing internal rotator activation) as elevation occurs (Fig. 39-20B). Aquatic therapy is another alternative approach that reduces the extremity weight and muscle activation.[51]

Pain-free submaximal isometrics and isotonic or elastic band exercises may be started either at the shoulder or the elbow. Commonly, isometrics are reserved for the patient whose symptoms are more reactive. However, caution must be practiced when using isometrics following a tendon repair, since tension overload is the suspected

Figure 39-20 Gatching using no weight **(A)** and weighted ball **(B).**

mechanism for tendon repair failure.[26,27,90,101] We do not know how much is too much, but the tension developed during a maximal or possible intense submaximal isometric exercise may be greater than the tension produced during submaximal isotonic or elastic band exercise. Positioning of the glenohumeral joint in a slightly abducted position by using a bolster may result in less passive tension of the contracting soft tissues.[90,113]

Isometrics can be performed in all directions, but many times resistance into abduction and flexion is painful. Therefore, the following movements are usually performed: external rotation, internal rotation, extension, elbow flexion, and elbow extension. Contractions are held for 3 to 6 seconds, and one to three sets of 10 to 15 repetitions are performed.

Phase I strengthening exercises using free weights of 1 to 3 lb or elastic bands are initiated. Elastic bands are easier to use, can be used in the functional erect position, and allow better integration of scapular muscles (versus side-lying).[79] These exercises include external rotation, internal rotation, and extension (Fig. 39-21). A bolster may be used between the arm and body to reduce passive supraspinatus tendon tension. Additionally, restricting external rotation to 20 to 30 degrees past neutral rotation may prevent tension overload due to the lengthening rotator cuff interval and disadvantaged posterior rotator cuff length–tension relationship as the band resistance increases.[34] Scapular muscle integration is encouraged at all times when strengthening the rotator cuff and deltoid muscles since coactivation allows both muscle groups to be strengthened simultaneously, reinforces synergistic firing patterns, and provides a balanced stable base upon which the compromised rotator cuff muscles may anchor, giving them a biomechanical advantage. Therefore, when performing glenohumeral dynamic strengthening exercises, the scapular adductors should be consciously activated or "set" by pulling the scapula slightly up and back, before the glenohumeral muscles are activated (Fig. 39-22). Research has shown that the commonly performed rotational exercises do not allow for scapular muscle integration.[79] Moseley et al.[79] found minimal activity of any scapular muscles when internal and external rotation strengthening exercises were performed in the side-lying position.

In addition to strengthening the scapular muscles through integration, isolated strengthening can be initiated using elastic band/free-weight resistance, manual resistance, or body weight. Our belief is that scapular muscles are not necessarily weak; instead, lack of coordinated control is the problem. Burkhart et al.[12] discussed numerous "control" exercises for the scapula that help overcome scapular dyskinesia. Kibler[54] is also an advocate of integrating scapular muscles and even includes lower extremity and trunk motion. More aggressive scapular muscle strengthening will be discussed in the phase III strengthening section.

Exercises are often started at two sets of 10 with a particular band or weight (can be the weight of the arm) and progressed to three sets of 10 when the patient has no pain or difficulty with two sets of 10. When three sets of 10 are easy to perform, the patient is progressed to three sets of 15. Progression to the next band resistance/weight is made when three sets of 15 become as easy as three sets of 10. This progression cycle is continued until the appropriate resistance level is achieved. When the patient can achieve three sets of 10 with green elastic bands, they are typically progressed to phase II strengthening. Patients are encouraged to perform the exercises at home one to three times a day depending on the patient and problem

Manual Resistance

If the patient is being seen in a supervised manner, manual resistance can be performed as a form of assessment and

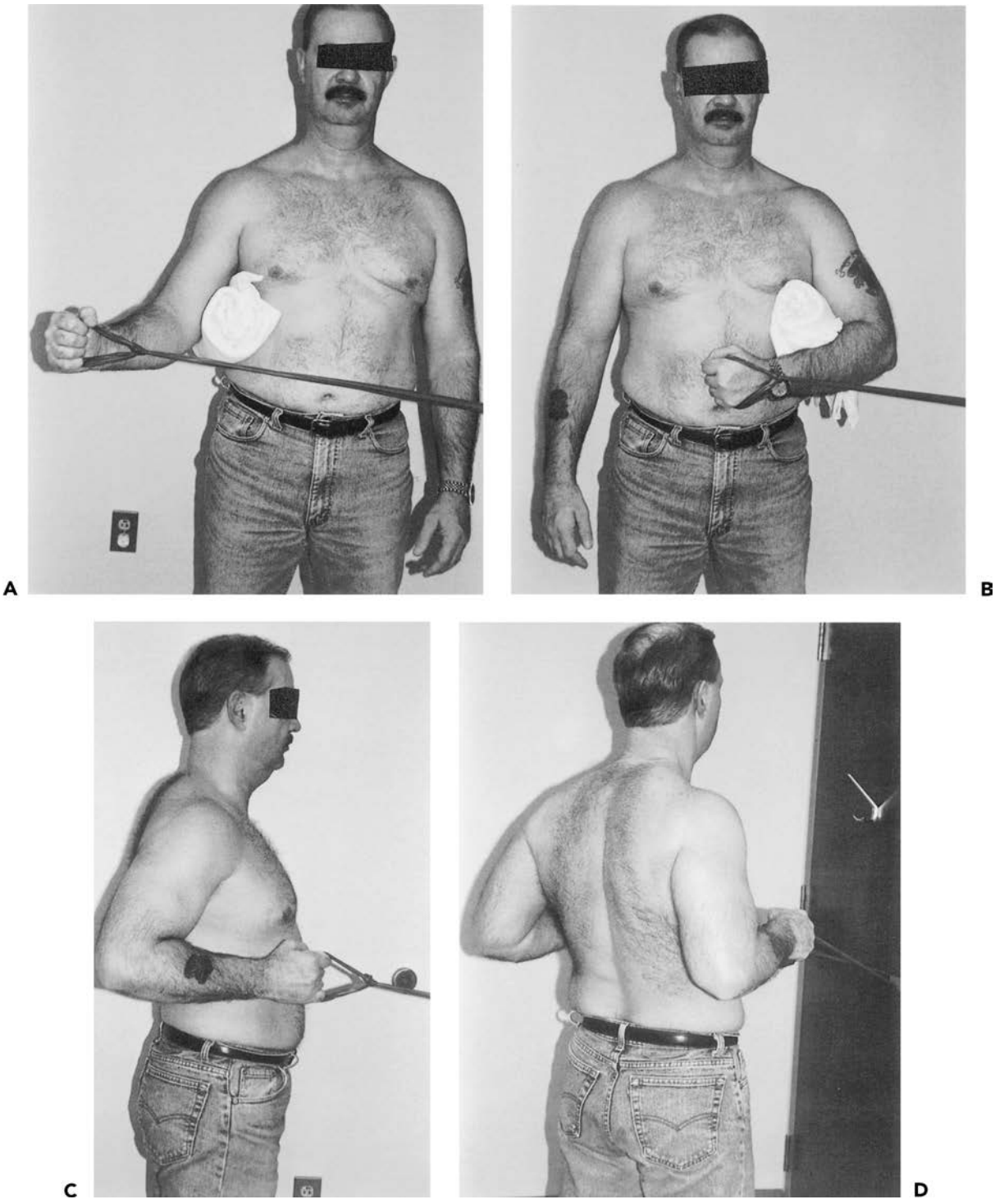


Figure 39-21 Phase I strengthening exercises. **(A)** External rotation to 30 degrees. **(B)** Internal rotation. **(C)** Extension (unilateral). **(D)** Extension (bilateral) to encourage scapular adductor muscle integration.

exercise. Manual resistance can be isometric or accommodating through the range while isolating scapular and glenohumeral muscles or facilitating cocontraction of both. Manual resistance is applied in multiple directions about the shoulder (i.e., internal rotation, external rotation, and elevation) in varying positions of elevation to determine whether a patient is ready for resistive exercises and what positions and/or directions of resistance to choose. The typical start position is in 60 to 70 degrees of elevation in the POS (Fig. 39-23). If pain is felt upon appropriate

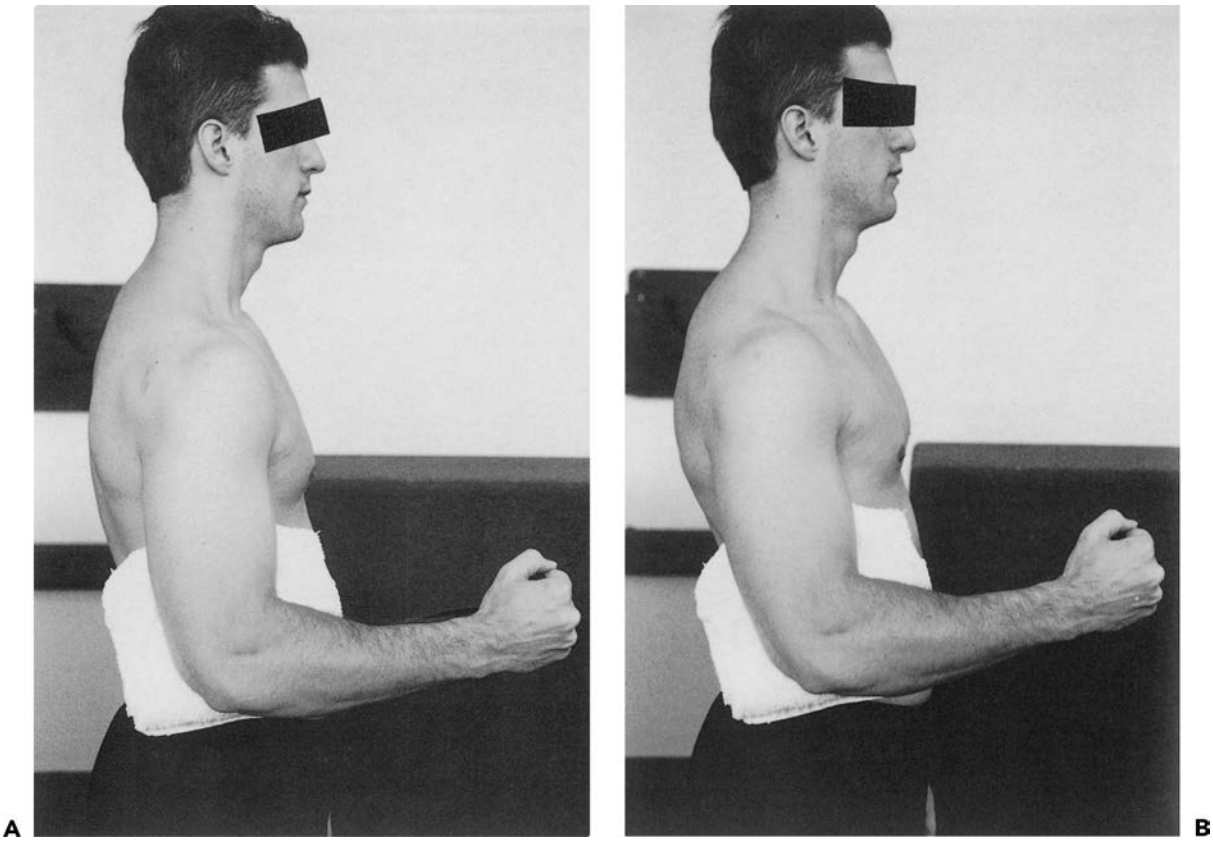


Figure 39-22 External rotation strengthening with bolster. Incorrect scapular position **(A)** and with scapular muscle integration **(B).**

submaximal resistance, the patient will refrain from resistive exercises, or modifications in position are attempted. We advocate the use of manual resistance as a leading form of supervised therapy because it provides immediate feedback from the patient regarding strength, pain, integration of scapular muscles, and appropriate scapulohumeral rhythm. By systematically resisting different muscle groups (i.e., abductors/external rotation or adductors/internal rotation) in varying degrees of elevation or rotation, neuromuscular training can be enhanced and the patient's home resisted-exercise program can be developed and modified.

Figure 39-23 Isometric manual resistance to the glenohumeral abductors and external rotators while encouraging scapular stabilization.

Manual resistance therapy following a rotator cuff repair can be essential to regaining active elevation motion. When a patient cannot actively elevate against gravity, not only does the rotator cuff lack the cross-sectional mass to translate enough force to stabilize and elevate the humerus, but it also "forgets" how. By providing very light resistance or assisting (while resisting) the arm into the elevated positions (90 to 140 degrees of POS abduction), neuromuscular training to facilitate elevation activity is achieved; the elevators (rotator cuff and deltoid) "relearn" how to contract. One advantage of training above 90 degrees, recognizing that the position is in the impingement zone, is that the deltoid's line of pull is improved (relative to below 90 degrees), creating a stabilizing joint compressive force and less shear force.[86] To strengthen in the elevated positions, the patient is placed supine and is first treated with heat, joint mobilization, and stretching to maximize the elevation range. Multiangle isometrics are performed at

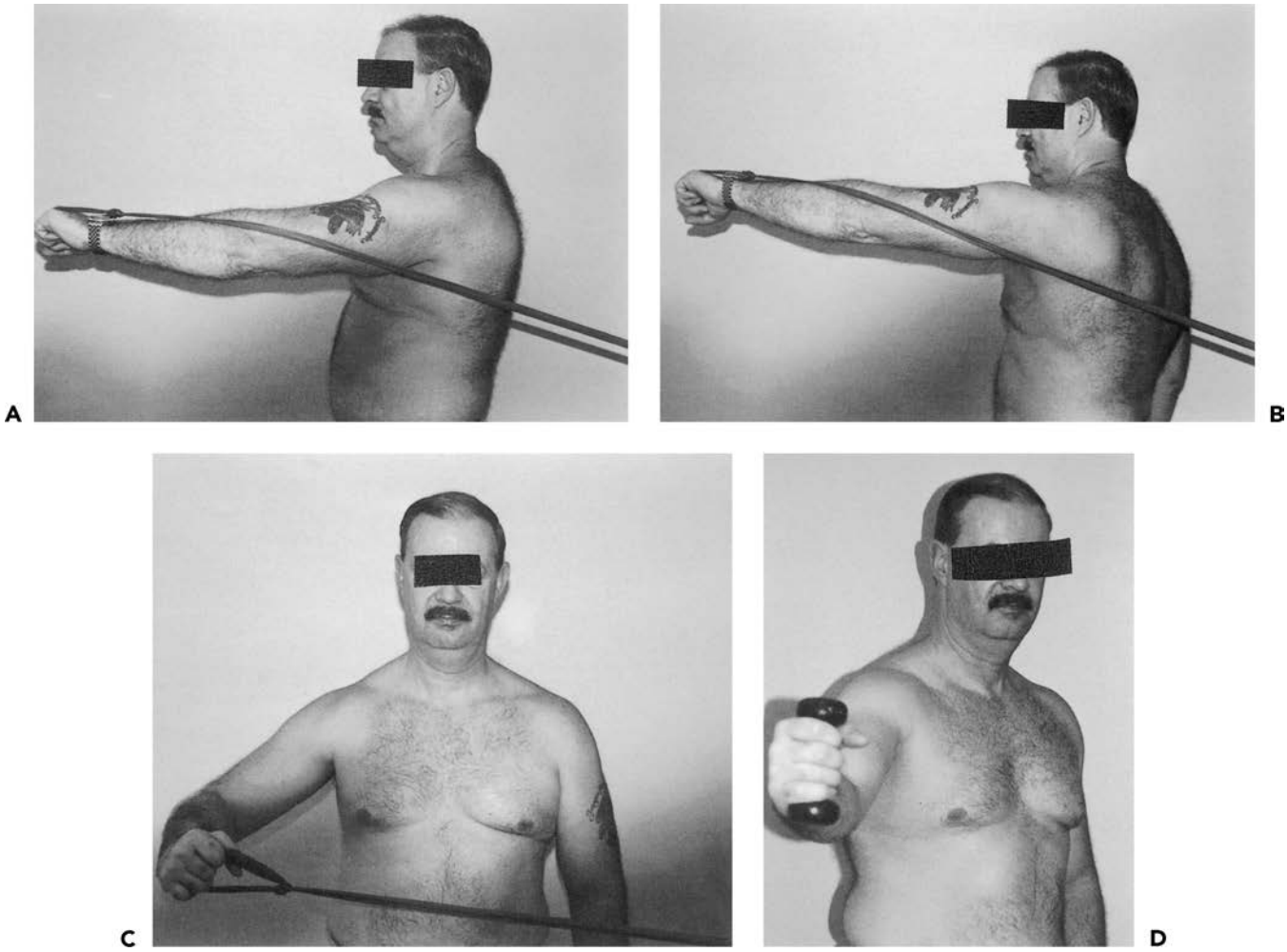


Figure 39-24 Phase II strengthening exercises. **(A)** Flexion. **(B)** Flexion with "plus" to encourage serratus anterior integration. **(C)** Abduction to 45 degrees. **(D)** Abduction to 90 degrees in the plane of the scapula.

different positions of elevation to improve the muscle's ability to contract and to improve the number of motor units recruited. The patient is then placed at 80 to 90 degrees and asked to elevate through the pain-free range while less than the weight of the arm resistance is given. Scapular shrugging is discouraged through verbal and manual cues to reinforce appropriate synergistic scapular muscle activity as the glenohumeral muscles are activated. This sequence has proved very effective in safely strengthening the rotator cuff-deltoid-biceps complex in functional ranges while encouraging scapular muscle integration.

Phase II

Phase II strengthening can be started when the patient can lift the extremity through the full passive range against gravity and when sufficient time has passed postinjury/surgery. Resistive exercise using elastic bands or light free weights is continued, but the resistance is increased and more provocative positions are used. The same set and repetition progression is used as in phase I.

Phase I exercises are continued, but the following phase II exercises are added: abduction, flexion without or with a "plus," supported external rotation at approximately 45 degrees, elbow flexion, and extension (Fig. 39-24). The phase I and phase II exercises form the core of the strengthening program. Some patients may not reach this level or progress beyond phases I and II because of low-demand needs or extensive pathology such as a rotator cuff deficiency. Scapular muscle integration is performed even with resisted elbow flexion and extension. Performing a "plus" maneuver during resisted shoulder flexion enhances serratus anterior activity[79] (Fig. 39-24B). External rotation strengthening is progressed to elevated functional positions but the arm is supported. The patient is progressed to the unsupported position and, if appropriate, to 90 degrees of abduction (Fig. 39-25). Backhand- and forehand-type motions can be initiated to encourage coactivation of

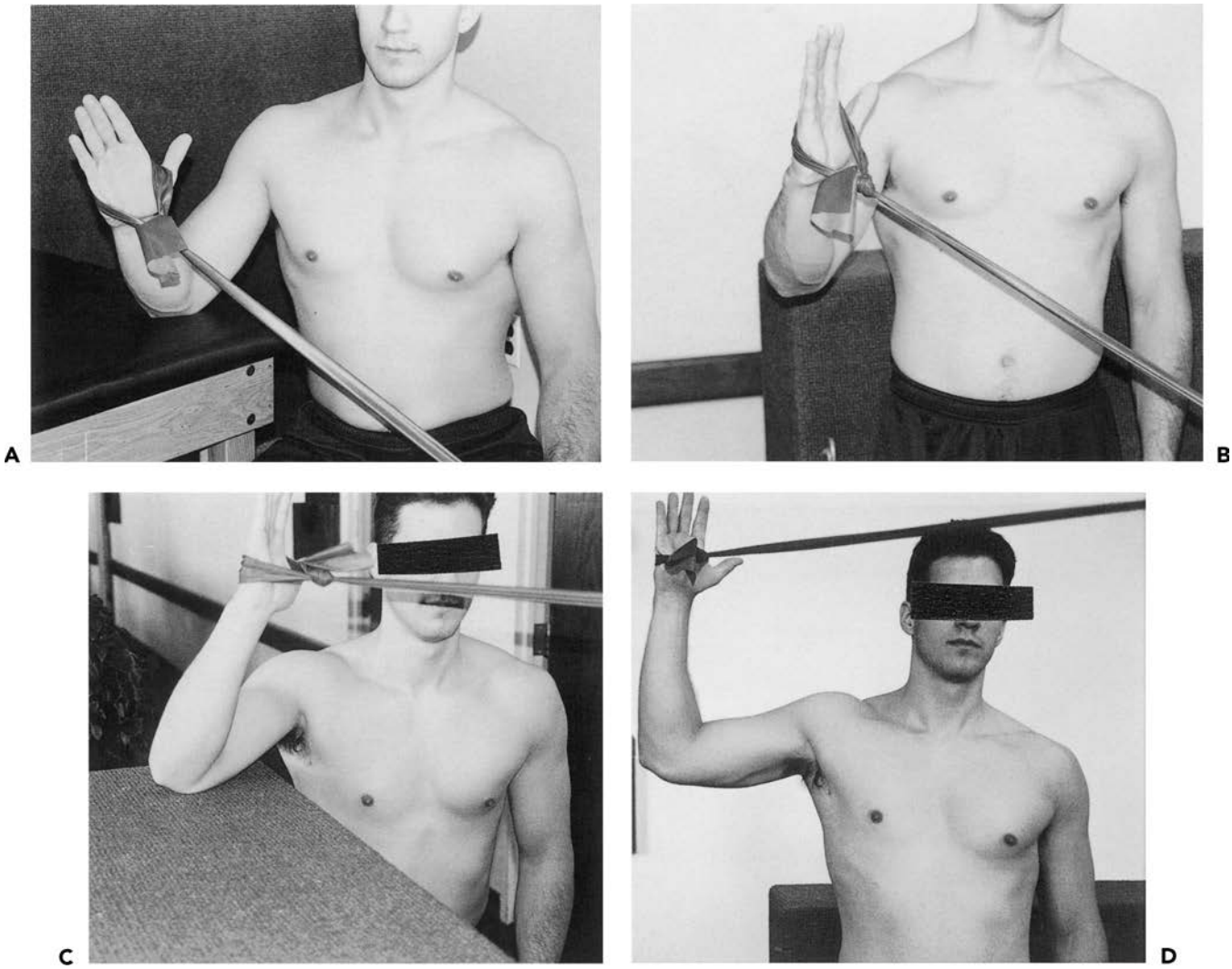


Figure 39-25 Elastic band strengthening progression. **(A)** 45 degrees plane of the scapula (POS), supported. **(B)** 45 degrees POS, unsupported. **(C)** 90 degrees POS, supported. **(D)** 90/90, unsupported. (Reproduced with permission from Kelley MJ. Anatomic and biomechanical rationale for rehabilitation of the athlete's shoulder. *J Sport Rehabil* 1995;4:122–154.)

scapulothoracic and glenohumeral muscles in functional positions. The backhand encourages the scapular retractors while the glenohumeral abductors and external rotators work. The forehand activates the serratus anterior and glenohumeral internal rotators.

Scaption (abduction in the POS) with external rotation or internal rotation using the extremity weight or a free weight can be performed. An elastic band may be used, but because such a long lever arm is created and the elastic can be stretched to a point of excessive resistance, light free weights may produce less potential tendon overload. Scaption with external rotation is preferred since sufficient supraspinatus activity is created and rotator cuff and bursal impingement is discouraged.[52] We have found that many patients have increased pain when performing scaption with internal rotation. Although this exercise was found to isolate the supraspinatus[43] and create the greatest muscle activity for the anterior and middle deltoid, supraspinatus, subscapularis,[105] and upper trapezius,[79] motion *above* 90 degrees was required. Although scaption with internal rotation may be beneficial at some stage of rehabilitation, performing it too early can result in further irritation of the rotator cuff due to increased shear forces and direct cuff compression.[35,105] The reader is encouraged to consult the literature on muscle activity based on EMG findings. Although these studies are valuable, direct application of study positions is not always clinically prudent because their use may be contraindicated relative to the patient's symptoms and pathology.

Commonly, exercises are performed in two to three sets of 10 to 15 repetitions, based on the patient's response and the rationale for exercise; however, in some high-demand

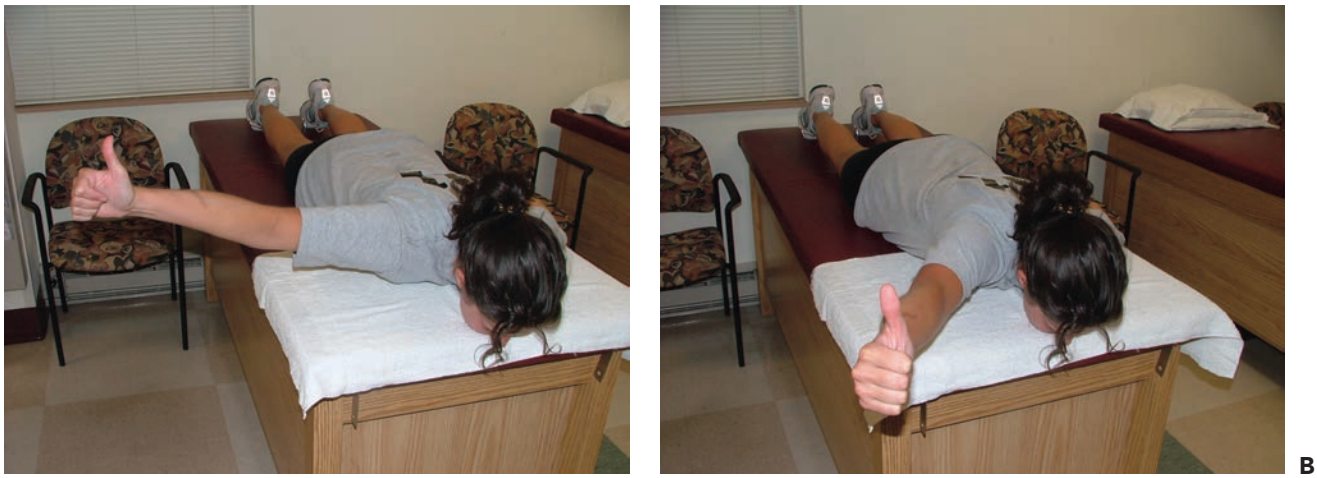


Figure 39-26 Prone horizontal abduction/end-range strengthening at 90 degrees **(A)** and 120 degrees **(B).**

patients, the repetitions are increased to 25 to 30. We feel that increased repetition achieves all the previously stated rationales for resistive exercise in addition to improving shoulder girdle muscle endurance.

Phase III

In this phase the patient should have relatively pain-free active range of motion, be able to perform a full-range stretching program, and be able to perform phases I and II strengthening exercises without symptoms. Individuals who have low demands other than activities of daily living are typically released on a home stretching and strengthening program. Previous rehabilitation literature[5,50,110] and research regarding proprioception and the neuromuscular mechanism[65,66] shows that rehabilitation exercise should be performed in positions of function. We advocate progressing strengthening exercises to functional (and possibly provocative) positions for patients who have high demands or must repetitively function with the arm overhead (i.e., the athlete or laborer). Neuromuscular control, joint stabilization, and strength of the shoulder complex muscles are enhanced as the level of difficulty is advanced. As the patient performs exercises in the elevated positions, appropriate scapulohumeral rhythm should be assessed and facilitated. Many patients who have adequate ROM and strength can still demonstrate abnormal rhythm during elevation. Learned behavior appears to be the reason for the persistent abnormal rhythm and must be identified and then addressed.

Many authors have reported rotator cuff and scapular muscle activation levels during closed chain or provocative position exercises.[9,44,79,105,107] Unfortunately, overenthusiastic and uninformed rehabilitation specialists can injure patients or prolong symptoms with the use of these exercises. The problem is not the research or the position, but the inappropriate use of both for the patient's pathologic condition. These exercises are reserved for the patient meeting phase III strengthening criteria.

Prone horizontal abduction at 90 to 120 degrees in external rotation has been shown to maximally activate the supraspinatus, infraspinatus, middle, and posterior deltoid (Fig. 39-26).[9,105] Caution must be practiced since this exercise is extremely provocative to patients with reactive rotator cuff pathology or anterior glenohumeral instability. Moseley et al.[79] evaluated scapular muscle function using the same exercises as Townsend et al.[105] However, they did not include scaption/internal rotation but only scaption/external rotation. Scaption/external rotation was found to optimally recruit six scapular muscles, but peak activity occurred above 120 degrees. Rowing was advocated due to increased trapezius (all heads), levator scapula, and rhomboid activity. Prone horizontal abduction with external rotation also increased activity of the scapular retractors. The middle and lower fibers of the serratus anterior were significantly recruited during scaption/external rotation as well as during performance of a pushup with a "plus."[79] Again, the patient with rotator cuff tendinopathy or glenohumeral instability must be able to safely tolerate these positions. The rehabilitation specialist must first consider the pathology, tissue reactivity, time from surgery, and target muscle before placing a patient into a provocative position for the potential benefit of maximizing activity of particular muscles. Only patients who have progressed to symptom-free status after adequate healing time has passed should be progressed to the most provocative positions.

Exercise progression continues into more provocative positions based on the patient's response and functional requirements. Later in the rehabilitation process, the exercises are higher demand and may be designed to ready the athlete or laborer. The "throwers ten" program and closed-chain exercises may be utilized when appropriate.[4] Manual

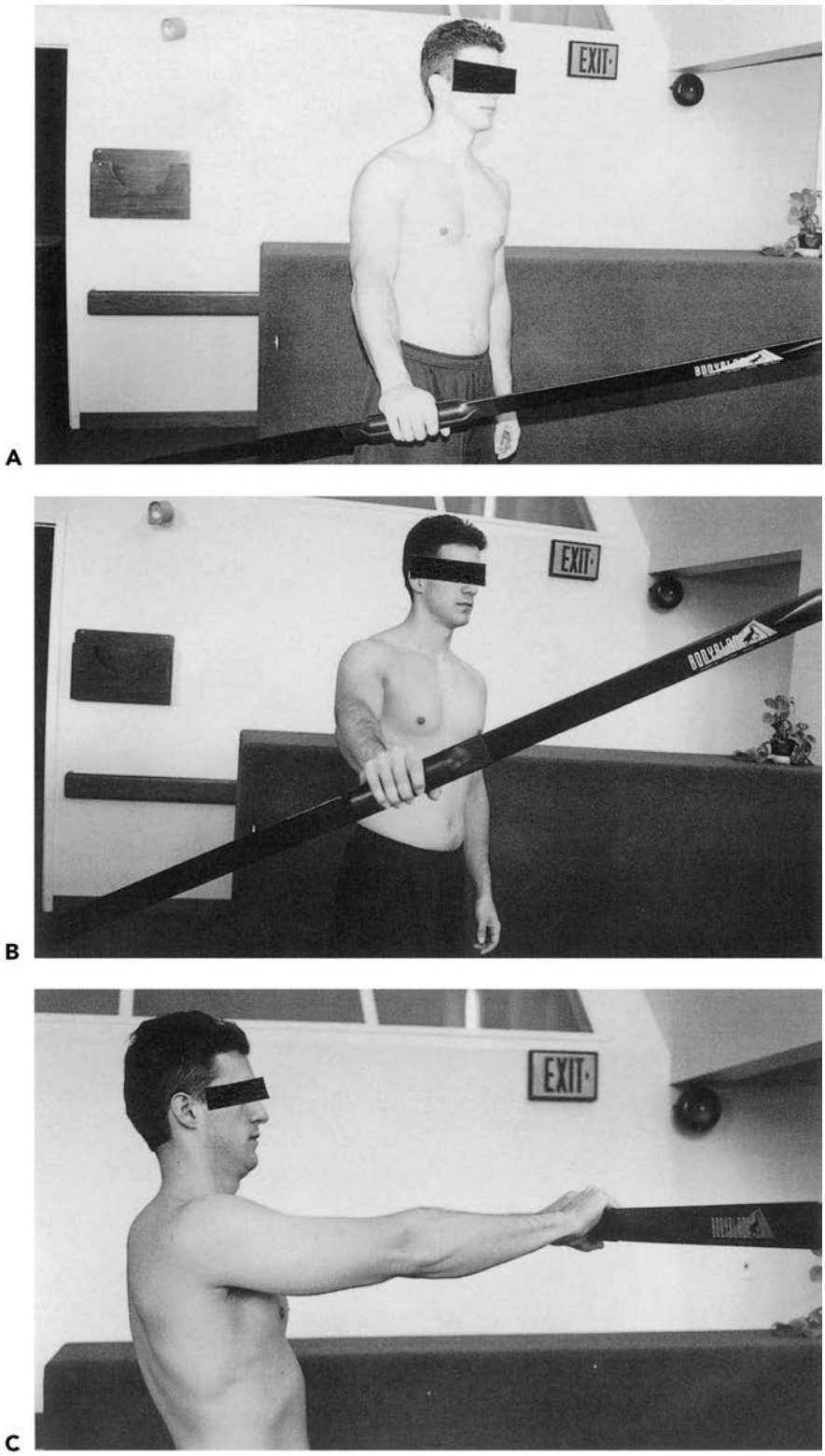
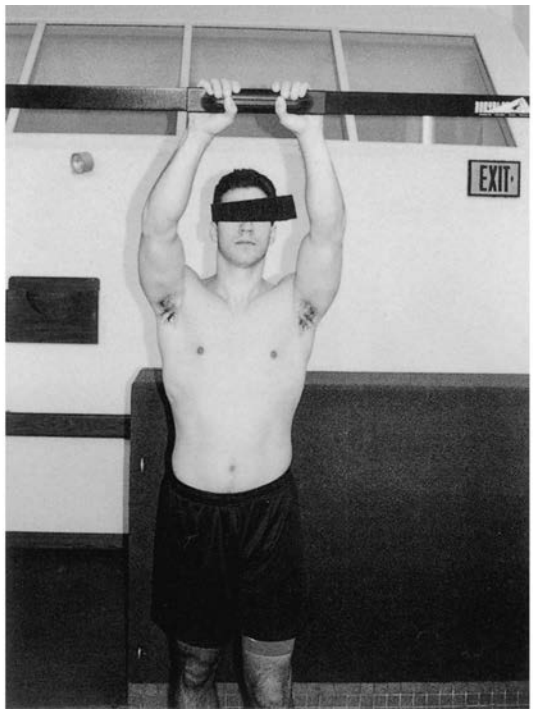


Figure 39-27 Bodyblade progression. **(A)** 30 degrees plane of scapula (POS). **(B)** 60 degrees POS. **(C)** 90 degrees sagittal with "plus." **(D)** Overhead. **(E)** 90 degrees POS. (Reproduced with permission from Kelly MJ. Anatomic and biomechanical rationale for rehabilitation of the athlete's shoulder. *J Sport Rehabil* 1995;4:122–154.)

resistance exercises are progressed to full range of motion and increasing resistance. Eccentric activity also may be emphasized. PNF techniques employing diagonal patterns, specific techniques of recruiting and sequencing isometrics, and concentric and eccentric muscle activity are performed.

Strengthening Equipment

Weight training using variable resistive devices or barbells is started with the same nonprovocative to provocative philosophy. This program can be progressed in the clinic,



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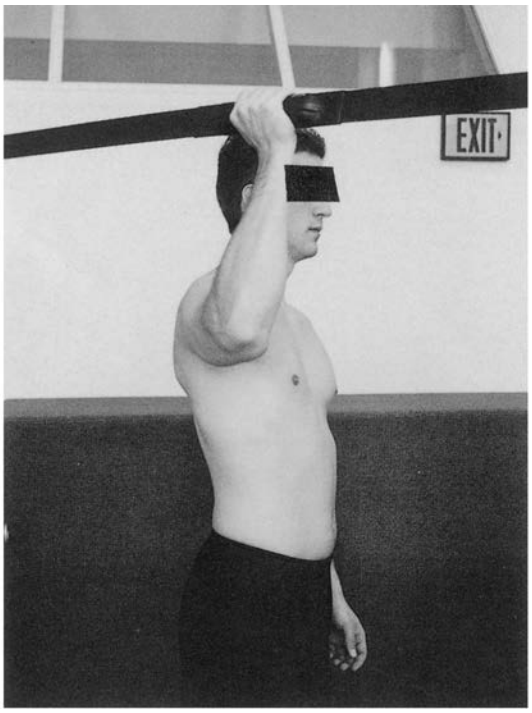

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Figure 39-27 (*continued*)

or the patient can be given indications for progression of the home program. Certain exercises that require shoulder abduction and full external rotation should be prohibited, particularly the military press, chest flies, and behind-the-neck latissimus pull-downs. These exercises may be modified to the POS.

The upper body ergometer (UBE) and rowing ergometer can be used to further improve strength and endurance. The UBE may be used earlier in the rehabilitative process; however, care is always required when considering the patient's position on the UBE. Placing the patient in a sitting position so that the machine's rotation axis is level with the glenohumeral joint requires the patient to repetitively cycle against resistance in the impingement zone. The prudent clinician initially has the patient perform the exercise in the standing position to avoid the potential rotator cuff trauma. Both the forward and backward directions are performed. We find the UBE beneficial in patients who have glenohumeral instability but do not advocate its use in patients with a reactive rotator cuff or postoperative cuff repair. A rowing ergometer can be used and is very effective in improving strength and endurance of the shoulder extensor and scapula retractor muscles while improving the patient's cardiovascular condition. The rowing ergometer is used in high-demand patients who fulfill stages III and IV criteria.

An extremely useful device for rehabilitating patients with rotator cuff tendinopathy and/or glenohumeral instability is the Bodyblade (Hymanson Inc., Playa Del Ray, CA) (Fig. 39-27). This device enhances strength, dynamic control, proprioception, and endurance training. Small to large oscillations of a fiberglass rod are performed in multiple positions (following the nonprovocative to provocative philosophy) and various time intervals of 10 to 60 seconds. Oscillating the blade requires short excursion, high-speed cocontraction muscle activity of the rotator cuff–deltoid–biceps complex in addition to the scapular muscles. Therefore, dynamic stabilizing training is uniquely achieved compared with other forms of exercise. Although exercising with this device may appear easy, it can be extremely difficult and can significantly challenge the patient's shoulder muscle strength, control, and endurance. Because the device can be used in any position, selective patients in phase II can use this device. Typically the patient is placed in a slightly abducted position and oscillates the blade, primarily through elbow flexion and extension. This requires proximal stabilization at the glenohumeral and scapulothoracic joints while the distal elbow is moved. The progression of the Bodyblade is based on the patient's demands. This device is well suited to the patient with glenohumeral instability but is rarely used in the older postoperative patient following a rotator cuff repair.

Plyometric training using weighted balls can be used to enhance neuromuscular control, strength, and proprioception by reproducing the physiologic stretch-shortening

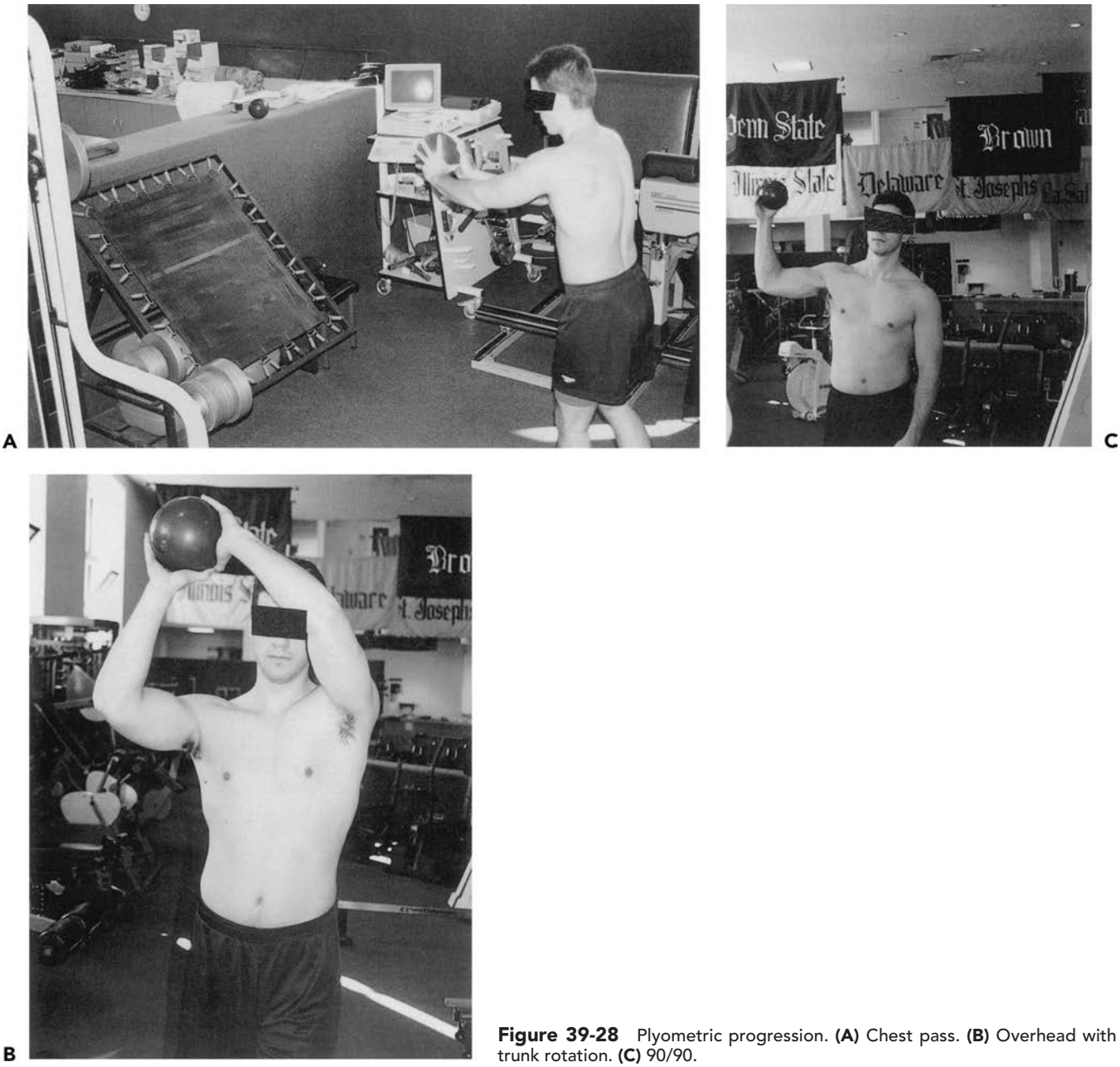

Figure 39-28 Plyometric progression. **(A)** Chest pass. **(B)** Overhead with trunk rotation. **(C)** 90/90.

cycle of muscle in multiple shoulder positions.[103] This activity can be used for any type of patient, but as with isokinetics, it is usually reserved for the younger patient or athlete. By catching and/or throwing a weighted ball (2 to 10 lb), the adductors/internal rotators are eccentrically loaded, and thus stretched, which is followed by a concentric shortening phase. Plyometric exercise creates a "quick" muscle contraction, as opposed to most other strengthening, which is slow and controlled. Initially a chest pass–type throw with both hands is used requiring approximately 90 degrees of elevation at ball release (Fig. 39-28A). The ball can then be arced to incorporate greater elevation (Fig. 39-28B). Sagittal plane motion may be more provocative to the athlete with posterior glenohumeral instability, and progression of difficulty should include horizontal adduction when arcing. True eccentric loading of the internal rotators is begun in the POS at 60 to 80 degrees and progressed to more provocative positions of elevation in the POS, eventually progressing to coronal plane positioning (Fig. 39-28C). The patient is in a functional standing or throwing position. The ball is thrown at the patient, so the velocity can be varied (varying the force of impact), yet placement of the arm may be consistent or varied, which is determined by the rehabilitation specialist. Constant

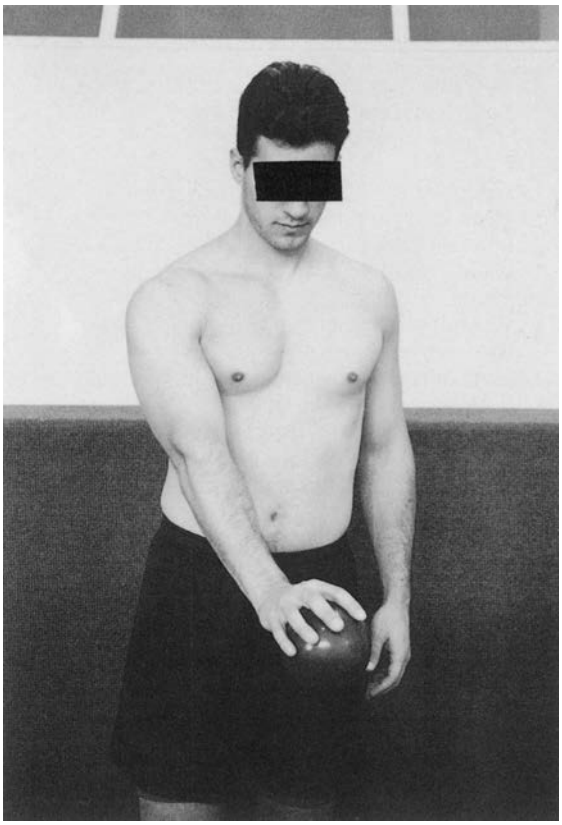

Figure 39-29 Plyometrics emphasizing glenohumeral and scapular decelerators. (Reproduced with permission from Kelly MJ. Anatomic and biomechanical rationale for rehabilitation of the athlete's shoulder. *J Sport Rehabil* 1995;4:122–154.)

assessment is made of the scapular muscle to achieve balance. Typically, with repetitive throwing or catching while in humeral elevation, the patient begins to overcompensate with the scapular elevators and correction is required. Catching the ball from a vertical drop while standing or side lying is a technique to eccentrically load the posterior cuff and scapular decelerators (Fig. 39-29).

The patient who must return to work is gradually progressed to work-simulated activities. Using variable resistance units that require the patient to push, pull, or lift can do this. Specific job activities such as lifting containers can be easily reproduced in the clinic with crates filled with varying weights. Work stations are available in many rehabilitation centers that allow the patient to use tools for various tasks. The task can be elevated or lowered depending on the patient's status. Education regarding lifting, tool use, and modification and ergonomic modification should all be addressed.

Phase IV

The patient is now performing full motion against resistance in provocative positions without symptoms. By this time most individuals other than athletes and laborers should have returned to all necessary activities. If they still have difficulty performing some overhead or lifting tasks such as painting a ceiling or picking up a bag of leaf clippings, they are taught the biomechanics of lifting to modify the task—by using a ladder, splitting the clippings, or using a wagon. In reality, this is usually discussed and performed in the previous phases.

During this phase the athlete should continue with the rotator cuff activities with a bias toward sport-specific positions. They will enter a modified off-season conditioning program. Allowing accommodating reentry philosophies encourages a gradual return to their sport. The pitcher will continue with an interval-throwing program, and the tennis player will continue with a return to noncompetitive forehand and backhand strokes, eventually progressing to technique serving strokes. The swimmer performs low-intensity interval training. Gradually the intensity of the activity is increased.

The laborer may be placed in a structured work-hardening program that is gradually increased in intensity. Education regarding lifting biomechanics and ergonomic modifications should be strongly emphasized during this phase. A functional capacity evaluation may help determine objective capabilities and limitations. Depending on the employer and job requirements, the patient may begin work that specifies job restrictions. This is not always possible; therefore, the patient must stay out of work until symptoms abate and/or soft tissue healing time parameters are satisfied. Occasionally, retraining or reeducation of the individual is required due to excessive job demands.

SUMMARY

This chapter presented principles, goals, rationales, and shoulder rehabilitation interventions. The common goals of the rehabilitation process include reduction of pain and inflammation, facilitation of collagen healing, improvement in ROM and strength, and optimization of proprioception and endurance. Goals are achieved by gradually increasing the program from nonprovocative to provocative positions, progressively increasing the connective tissue load through stretching, and challenging the neuromuscular system. Matching the rehabilitation program to the patient's demands protects the tissue and maximizes outcome.

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40 Disease-Specific Methods of Rehabilitation

Brian G. Leggin *Martin J. Kelley*

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SUMMARY 1291

Rehabilitation of the shoulder is a critical component to the recovery of function following surgical procedures. However, the quantity of rehabilitation does not always equate to quality. Each patient requires a different level of intervention. Supervised therapy three times per week is not necessary for all patients. Many patients need only instruction in a home program and periodic evaluation and progression of the rehabilitation program. Therefore, it is incumbent upon therapists, surgeons, and the patient to administer the appropriate amount of rehabilitation following shoulder surgery.

We believe in a team approach to shoulder rehabilitation. The team consists of the surgeon, the therapist, and most importantly, the patient. Frequent and effective communication among team members is essential. The surgeon must provide the patient and therapist with surgical findings, precautions, and contraindications. The therapist must instruct the patient on an appropriate program and

guide its progression. The patient needs to receive the information, follow through with adherence to precautions and exercise program, and communicate to the therapist and surgeon about progress. When complications develop or progress is not as expected, the therapist must make necessary modifications to the rehabilitation program. If these modifications fail to yield expected results, the therapist should inform the surgeon and make appropriate recommendations. When all members of the team perform their roles effectively, the result is a successful outcome.

A previous chapter discussed the specific exercises and modalities employed in shoulder rehabilitation (Chapter 39). This chapter will discuss the rehabilitation principles and guidelines for common shoulder disorders utilizing those exercises and modalities. The approach to rehabilitation when complications following shoulder surgery occur will also be discussed.

EXAMINATION

The postoperative rehabilitation program will depend largely on the pathology, type of surgery performed, and tissue healing. The patient's impairments and functional limitations must also be considered. There are a few pieces of information that should be gleaned from the history. It is important to note the patient's age, occupation, and recreational activities, as well as the patient's goals following surgery and rehabilitation. Whether the patient received any presurgery therapy will help the clinician understand the patient's conception of rehabilitation. It is also extremely important to communicate with the surgeon who performed the procedure and, if possible, to obtain a copy of the operative note. Gaining this information helps the clinician understand the quality of the soft tissue that was repaired, as well as any nuances of the surgery that may affect the rehabilitation process. Frequently, patients ask the therapist about what was done during surgery. Having a copy of the operative report or communicating directly with the surgeon will allow the therapist to answer that question and further gain the confidence of the patient.

The postoperative physical examination will not include special tests such as impingement signs or instability testing. The examination will be predicated and modified based on the stage of rehabilitation. For example, active range-of-motion (ROM) testing is almost always contraindicated in the first 6 weeks after surgery.

Pain is a parameter that should always be measured, especially during the first 3 months after surgery. The intensity of shoulder pain may vary depending upon the position or activity the patient is engaging in at any one time. Some patients experience very little pain after surgery, especially when their arm is immobilized. However, when they are able to start moving and using their upper extremity, pain may increase. Therefore, a complete assessment of pain should include pain experienced at rest as well as with activities of daily living and more strenuous activities.

Shoulder ROM is an important outcome of shoulder surgery. However, because of the multiplanar motion of the shoulder, it can be difficult to reproduce and compare with results reported by other clinicians.[1] In response to the need to standardize measurements, the American Shoulder and Elbow Surgeons (ASES) recommend that four functionally important ranges of motion be documented: forward elevation, external rotation with the arm at the side, external rotation in the 90-degree abducted position, and internal rotation with the hand up the back.[2,3] Forward elevation is defined as the maximum angle the arm makes with the trunk when the patient is asked to raise his or her arm above the head with the elbow held straight. It should be noted that this plane of motion is not considered to be true flexion or abduction. Experience tells us that most patients will raise their arm somewhere between true flexion and the plane of the scapula. The angle that the arm makes with the thorax is measured in the upright position for active motion, whereas passive measurement is made in the supine position. We will discuss when it is appropriate to measure each of these motions following specific surgical procedures.

Muscle force assessment is another commonly reported impairment used to document effectiveness of surgical or therapeutic intervention. As with ROM testing, it is important to have standardized testing protocols for reproducibility in a clinical setting. A variety of methods for quantifying muscle performance are available, including manual muscle testing (MMT), handheld isometric dynamometry, and isokinetic dynamometry. MMT is the most widely used method of clinical evaluation of muscle strength.[4] This technique, however, has been criticized for its subjectivity and lack of reliability within the good and normal ranges.[5,6] Isokinetic devices have fallen out of favor for use in the clinic because they are nonportable, are relatively expensive, and require elaborate setup and stabilization procedures.

Handheld dynamometry has been demonstrated to be reliable in both patient and nonpatient populations.[7–11] This method of muscle force measurement is portable, is relatively inexpensive, and does not require elaborate setup. Handheld dynamometry can also detect subtle differences in strength that MMT cannot. To accurately compare results of muscle force measurements, standardization of test position, stabilization, and protocol must be established. Measuring shoulder muscle performance of internal and external rotation with the arm at the side in neutral rotation and forward elevation at 45 degrees in the plane of the scapula has been demonstrated to be reliable and is the authors' preferred method of shoulder muscle force measurement.[11]

Impairment measures alone are not adequate measures of outcome.[12] It is also important to document any functional difficulties the patient may have. Several tools have been developed and used to document outcomes of patients with shoulder pathologies, including generic health status measures and condition-specific tools.[3,13–22] Condition-specific tools have been demonstrated to be more responsive than general health status measures in patients with shoulder disorders.[14] In the authors' practice, we use the Penn Shoulder Score (PSS) and the ASES Shoulder Score Index.[3,22] Outcome measurement is discussed in more detail in a later chapter (Chapter 41).

TABLE 40-1
POSTOPERATIVE PRECAUTIONS AND INSTRUCTIONS

Able to use arm for waist-level activities and basic activities of daily living (in appropriate patient)
No leaning on elbows
No sleeping on the involved side (emphasize arm supported in the plane of the scapula)
No sudden movements
No lifting and carrying with involved arm
No pushing or pulling
Importance of home exercise emphasized
Use of ice emphasized

REHABILITATION PRINCIPLES

The principles of rehabilitation after shoulder surgery remain constant regardless of the type of surgery performed. First, pain is always respected and rarely encouraged. Constant reevaluation after the introduction of a new exercise or technique is necessary to help prevent increased symptoms. Any increase in pain should be addressed through patient education on precautions, positioning, and proper exercise technique. Second, treatment is prioritized based on the impairments identified during the examination. Tissue healing is always taken into consideration prior to initiating any rehabilitation program. Third, exercise and techniques are advanced in motion, resistance, and movement planes based on symptoms and the functional demands of the patient. To assess their effect, modalities are introduced to the program one at a time. Range of motion should be restored in a protected, pain-free manner. Once the appropriate time frame for tissue healing has passed and pain and passive range of motion have improved, active range of motion and muscle performance can be assessed and addressed.

Emphasis is placed on the performance of a home exercise program. As such, no more than two or three new exercises are introduced at any one time. This helps the patient digest the information given and hopefully improves compliance and proper performance of the exercises. It also helps the therapist assess the effect a new set of exercises has on the patient. Improvements in these impairments lead to the introduction of activities to replicate the patient's functional demands.

Patient Education

The importance of patient education cannot be emphasized enough. The patient must understand and adhere to the precautions and instructions outlined by the surgeon and therapist, especially during the first 6 weeks after surgery (Table 40-1). The patient needs to be educated about the healing process and the importance of protecting the surgical repair. The patient should also be instructed in proper positioning of the arm for comfort and to promote healing. Many patients report that while at rest or sleeping, the most comfortable position is with the arm supported in the plane of the scapula. From a biomechanical standpoint, this also appears to be a more advantageous position.

Modalities

In addition to avoiding aggravating activities and positions, modalities have been advocated for pain reduction and relief of inflammation. Much of the evidence for their use is anecdotal or a product of questionable experimental design. A variety of modalities can be used during the rehabilitation process. The four main objectives for using modalities are to (a) reduce pain, (b) reduce inflammation, (c) improve range of motion, and (d) muscle reeducation. Most modalities achieve their physiologic effect by the transfer of thermal energy. These modalities include hot packs, cold packs, ultrasonography, diathermy, and infrared. Another mode of achieving a physiologic effect is through the use of electric stimulation. Electric stimulation modalities include transcutaneous electric nerve stimulation (TENS), as well as low-voltage, high-voltage, interferential, and direct current stimulation.

Range of Motion

Range-of-motion and stretching exercises are designed to prevent adhesions and/or fibrosis, reduce pain, allow collagen healing, and increase tissue length. Postoperative ROM exercises are performed within the direction and protected range allowed with respect to tissue healing and type of surgery performed. Stretching into provocative positions or aggressive stretching should not be performed to avoid putting the surgical repair at risk.

When restoring normal ROM of the shoulder, one should consider which structure might limit the motion. Studies have shown that external rotation with the arm at the side is most limited by the subscapularis and the coracohumeral ligament.[23–25] External rotation with the arm at 45 degrees appears to be limited by the subscapularis and middle fibers of the anterior glenohumeral ligament.[24] The inferior glenohumeral ligament limits external rotation when the arm is abducted to 90 degrees.[24] Gerber and colleagues simulated capsular contractures in cadavers and measured changes in elevation and rotation ROM. They found that restriction of the anterior capsule restricted external rotation ROM and posterior contractures restricted internal rotation ROM.[23] Contracture of the superior capsular structures limited rotation motions with the arm adducted.[23] Contracture of the inferior structures yielded restriction in abduction and rotation in the more elevated positions.[23]

We have divided our ROM exercises into phase I and phase II. They have been discussed in more detail in a previous chapter (Chapter 39). Phase I exercises may be initiated within the first 6 weeks postoperatively and include pendulums, passive or active assisted elevation, and external rotation with the arm positioned at 45 degrees in the plane of the scapula (Chapter 39, Fig. 39-7). Phase II ROM exercises are considered more provocative to surgically repaired tissue and may not be initiated until at least 6 weeks after surgery. These exercises include extension, internal rotation, and cross-body adduction (Chapter 39, Fig. 39-9). The patient is asked to take the extremity to a position of tolerable stretch and hold the position for 10 to 20 seconds. Each exercise is repeated 10 times, three to six times per day at home.

Strengthening

Muscular strength refers to the capacity for active tension development by a muscle.[26] Multiple factors influence muscle strength.[27] Strength gains occur by two types of neuromuscular changes: increased motor unit recruitment and hypertrophy.[28] Appropriately loading a muscle will result in hypertrophy or increased muscle mass and, subsequently, increased muscle force production. However, significant strength gains have been shown to occur in the absence of hypertrophy due to improved motor unit recruitment and synchronization.[27] Enhancing synchronization of the rotator cuff, deltoid, and scapular muscles, also referred to as neuromuscular training, is generally the rationale for improving shoulder function.

There are several methods therapists can use for improving strength and neuromuscular control. These include manual resistance, elastic resistance, free weights, and machines. Regardless of the method, the underlying principle guiding the therapist is that exercises should begin in nonprovocative or supported positions with a gradual progression toward potentially provocative or functional positions.

Our strengthening exercise program is also categorized by phases and has been discussed in more detail in a previous chapter (Chapter 39). Phase I exercises include external and internal rotation with the arm at the side, and extension with elastic resistance (Chapter 39, Fig. 39-21). Patients are typically asked to perform 10 repetitions with the lightest resistance. They are able to add a second set of 10 when the first set is performed without difficulty. A third set is added when there is no difficulty with the first two. When all three sets become easy, the patient may progress to the next level of resistance. Phase II of the strengthening exercise program is added when the patient can perform all three of the phase I exercises with the third level of resistance. These exercises include abduction to 45 degrees, forward elevation below shoulder level, and external rotation with the arm supported at 45 degrees (Chapter 39, Fig. 39-24).

The scapular strengthening program has also been divided into phases. Phase I is meant to address all portions of the trapezius muscle. Scapular retraction can be performed at three different positions: waist level, above the head, and ground level. Retraction with the resistance above the head should only be performed if this is a pain-free position for the patient. Phase II of the scapular strengthening program includes combination movements such as horizontal abduction with scapular retraction, horizontal adduction with scapular protraction, and scapular retraction with glenohumeral external rotation.

Some patients may go on to more strenuous exercises, which we term phase III. These include activities in more provocative and functional positions. Exercises with a Bodyblade (Hymanson Inc., Playa Del Ray, CA) or plyometrics with a weighted ball can be employed to enhance strength, dynamic control, proprioception, and endurance (Chapter 39, Figs. 39-27 to 39-29). Instruction in proper performance and use of variable resistance machines or free weights can also be introduced at this time.

General Rehabilitation Guidelines

As a general guideline, the authors recommend that the patient perform most of the rehabilitation exercises at home during the initial 6 to 12 weeks after surgery. Physical therapy visits are coordinated with the patient's postoperative physician visits at specified time intervals. Individual patient need may dictate more frequent physical therapy visits. The patient typically returns to the physician 7 to 10 days after surgery. In most cases, the patient is instructed in phase I ROM exercises and asked to perform the exercises three to six times per day until their next physician visit at 6 weeks postsurgery. We recommend supervised therapy for patients who fit one or more of the following criteria: unable to demonstrate exercises properly,

passive forward elevation ROM less than or equal to 100 degrees, or less than 10-degree improvement in passive forward elevation during the first visit. When the patient exceeds these criteria, he or she may be allowed to continue with the home program. At the 6-week postoperative visit, patients may be instructed in phase II ROM exercises and phase I strengthening. Phase I of the scapular strengthening exercises may also be introduced at this time. Patients may be progressed to phase II strengthening exercises at the 12-week postoperative visit.

ROTATOR CUFF DYSFUNCTION

Subacromial Decompression with Intact Rotator Cuff

Patients with a prominent anterior acromion who have failed nonoperative management including rest, modalities, nonsteroidal antiinflammatory drugs, exercise, and a subacromial injection of cortisone will be considered for acromioplasty. Open acromioplasty has been an effective procedure with long-term satisfactory results ranging from 80% to 90% in most studies.[29–32] Arthroscopic acromioplasty has demonstrated similar results to open acromioplasty and is now the preferred method of treatment.[33] The major advantage of arthroscopic over open decompression is that deltoid detachment is avoided.[34] Use of the arthroscope allows inspection of the glenohumeral joint, as well as the undersurface of the rotator cuff, and any pathology encountered can then be addressed.[35] Finally, an arthroscopic decompression is less invasive and can be performed more easily on an outpatient basis.[35]

Following the procedure, the patient is sent home with the arm immobilized in a sling. The patient is allowed to remove the sling on the second postoperative day as long as there is no discomfort. The postoperative instruction packet includes instructions for the patient to perform pendulum exercises, elbow active range of motion (AROM), and hand squeezes four to six times per day. Most patients will be seen by a physical therapist for two to three visits after this type of surgery. Andersen and colleagues demonstrated that a self-training exercise program yielded similar results to physical therapist–supervised (six visits) program following subacromial decompression.[36]

The patient will return to the surgeon 7 to 10 days postoperatively for suture removal and further instructions. At this time, the patient will be instructed by a physical therapist in phase I ROM exercises including supine passive forward elevation and external rotation (Table 40-2). A majority of patients will be discharged from therapy with this home exercise program to be performed four to six times per day. The next postoperative visit occurs 4 to 6 weeks after surgery. At this point, the patient is instructed in phase II ROM exercises (extension, internal rotation, cross-body adduction), phase I rotator cuff strengthening (external rotation, internal rotation, extension), and scapular retraction with resistance. At the 8- to 12-week postoperative visit the patient will be progressed to phase II strengthening exercises and, possibly, phase II scapular strengthening exercises. Patients with lower demands (nonathletes or laborers) will continue to progress their home exercise program and possibly add a variable resistance program.

Beyond 12 to 16 weeks postoperatively, the patient who must return to work may be gradually progressed to work-simulated activities. Patients are instructed in proper lifting techniques, tool use, and modification of activities. The overhead athlete will be progressed to activities simulating his or her sport demands. Rotator cuff strengthening at 90 degrees abduction, plyometric exercise with weighted balls, and use of the Bodyblade may all be included in this phase of rehabilitation.

Rotator Cuff Repair

Among the many presentations of rotator cuff tears are degenerative or partial-thickness tears, an acute extension of degenerative or partial-thickness tears, chronic full-thickness tears, massive tears involving more than one tendon, associated biceps tendonitis or ruptures, and associated traumatic arthritis of the glenohumeral joint secondary to cuff deficiencies.[37] The presence of a rotator cuff tear is not necessarily an indication for surgery.[35] The indications for surgical repair of rotator cuff tears are, therefore, the presence of pain or functional deficits that interfere with activities and have not responded to conservative measures.[35] Most surgeons continue nonoperative treatment for at least 3 to 4 months before considering repair; when weakness is prominent or progressive, more timely repair may be considered.[35]

Although the best method for repair of full-thickness rotator cuff tears has been controversial, complete arthroscopic repair techniques have been evolving as an alternative to traditional open and mini-open repairs.[38–41] Neer reported the results of anterior acromioplasty in combination with rotator cuff mobilization and repair in 1972.[31] The surgical fundamentals emphasized in that report substantially improved the reliability of the outcomes of repairs of rotator cuff tears.[38] The fundamentals include (a) preservation or meticulous repair of the deltoid origin, (b) adequate decompression of the subacromial space by resection of any anteroinferior osteophytes, (c) surgical releases as necessary to obtain freely mobile muscle–tendon units, (d) secure fixation of the tendon to the greater tuberosity, and (e) closely supervised rehabilitation including early passive range of motion within a protected range.[38]

The current options for rotator cuff repair include the following: (a) arthroscopically assisted open repair, which consists of arthroscopic subacromial decompression followed

TABLE 40-2
PENN PRESBYTERIAN MEDICAL CENTER SHOULDER AND ELBOW SERVICE ACROMIOPLASTY GUIDELINES—INTACT ROTATOR CUFF

Phase I: 0–4 Weeks Postoperative

Goals:
1. Patient education
2. Permit healing
3. Control pain and inflammation
4. Initiate ROM exercises

Treatment:
Immediately postoperative or postoperative day 1:
1. Immobilized in sling
   Use sling for comfort and public only
2. Pendulums
3. Hand squeezes
4. Elbow AROM
   Supine PROM forward elevation (in appropriate patient)

Postoperative days 7–10:
1. Pendulums
2. Supine PROM forward elevation and external rotation
3. Heat and ice
   Active scapular exercises (shoulder shrugs, scapular retraction)

Phase II: 4–6 Weeks Postoperative

Goals:
1. Improve to full ROM
2. Improve neuromuscular control and strength

Treatment:
1. Continue all stretches
2. Add phase II stretches (extension, IR, and cross-body adduction)
3. Progress to phase II strengthening exercises when at green for all phase I strengthening (abduction, forward elevation, and external rotation at 45 degrees in POS with arm supported)
4. Advanced scapular strengthening
5. Manual resistance for rotator cuff, deltoid, and PNF

Phase III: 6–12 Weeks Postoperative

Goals:
1. Full pain-free ROM
2. Optimize neuromuscular control
3. Improve endurance
4. Initiate return to functional activities

Treatment:
1. Continue all stretches and strengthening; progress rotator cuff exercises into POS abduction
2. Appropriate variable resistance and/or free-weight resistance
3. Strengthening above 90 degrees
4. Plyometrics*/Bodyblade
5. Work-/sport-specific exercise*

Phase IV: 12–16 Weeks Postoperative

Goals:
1. Return to sport,* occupation,* or desired activities
2. Promote concept of prevention

Treatment:
1. Work hardening*
2. Sport-specific training*

*Applies to athlete or laborer.
AROM, active range of motion; IR, internal rotation; PNF, proprioceptive neuromuscular facilitation; POS, plane of the scapula; PROM, passive range of motion; ROM, range of motion.
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by open repair of the rotator cuff through a lateral deltoid-splitting approach; (b) mini-open arthroscopically assisted repair, which includes arthroscopic subacromial decompression, release of adhesions, placement of tagging sutures, and débridement of the tendon edges followed by a mini-open deltoid splitting approach to obtain suture management and bone–tendon fixation; and (c) complete arthroscopic repair, in which subacromial decompression, release of adhesions, and bone–tendon fixation are all carried out in an arthroscopic fashion.[38] Early reported experience with complete arthroscopic rotator cuff repairs has been promising, and the technique has become increasingly popular among experienced shoulder surgeons as a preferred means to obtain repair of the rotator cuff.[38] In experienced hands, the technique appears to offer less pain and morbidity as well as quicker recovery than do alternative techniques such as open or mini-open repair.

Rehabilitation following rotator cuff repair may vary based on surgical technique, cuff tear size, tissue quality, amount of tension at the repair site, patient age, patient goals, functional demands of the patient, and systemic disease processes. The prognosis following repair has been correlated to the rotator cuff tear size, presurgery atrophy, and presurgery ROM restrictions.[42–47] The amount of postoperative interaction the patient has with the surgeon and therapist is dictated by individual patient need. Typically, the therapist sees the patient at the time of postoperative visits with the surgeon. These visits usually occur 7 to 10 days, 6 weeks, 3 months, and 6 months postoperatively. Approximately 70% of patients in our service are managed in this way. More frequent visits to a therapist may be called for if the patient is not progressing as expected. An assessment of the amount of therapy needed in the first 6 weeks after surgery is made at the first postoperative visit. Patients are

expected to achieve the following at that visit: at least 100 degrees of passive forward elevation, at least 30 degrees of passive external rotation, and no difficulty performing the home exercises. Patients who do not meet those criteria will be seen in physical therapy on at least a weekly basis until the range of motion improves sufficiently.

Two studies have demonstrated that patients achieve a good outcome after rotator cuff repair regardless of the amount of contact with a physical therapist.[48,49] Roddey et al. demonstrated that with a therapist available for questions, patients who utilized a videotape method for their home exercise program instruction had self-reported outcomes equal to patients instructed in their home program personally by a physical therapist.[48] However, one must question whether there was really much difference in the treatment technique between the two groups. In addition, 30% of the patients who entered the study dropped out. It is unclear whether they may have entered supervised physical therapy. Hayes and colleagues randomized patients following rotator cuff repair into an individualized physical therapy treatment group and a standardized unsupervised home exercise regime.[49] By 24 weeks postoperative, most patients demonstrated favorable outcomes regardless of rehabilitation mode. However, 6% of the patients in the home exercise group sought individualized physical therapy by 6 weeks postoperative, 19% sought physical therapy by 12 weeks postoperative, and 28% sought physical therapy by 24 weeks postoperative.

The rehabilitation program we are presenting consists of a core set of stretching and strengthening exercises. The timing of when these exercises are introduced to the patient depends on the type of procedure performed and size of the rotator cuff tear (Tables 40-3 and 40-4). We will discuss the rehabilitation process after rotator cuff repair through four phases.

Phase I

When beginning rehabilitation after rotator cuff repair, a therapist must know the size of the tear and tendon involvement, quality of the tissue and ease of tendon mobilization, surgical technique, presurgery treatment, and the patient's goals. Patients who have had repair of a small or medium cuff tear will most likely be immobilized in a sling, with or without a small abduction pillow, to be used for comfort and when they are in public places. The sling is used when dressing or bathing for the first 7 to 10 days. It is then used predominantly when the patient is in a public place.

Patients who have a large or massive rotator cuff repair are oftentimes immobilized in an abduction pillow or brace for the first 3 to 6 weeks postoperatively. The rationale for bracing in this position stems from work to examine the passive tension generated in the supraspinatus musculotendinous unit at the time of repair in patients undergoing repair of long-standing rotator cuff rupture.[50] This study found that shoulder adduction increases the amount of passive tension of the muscle. Therefore, positioning the arm at 30 to 45 degrees of elevation in the plane of the scapula would seem to allow healing while reducing the risk of damage to the rotator cuff repair site. Patients immobilized in this way are only allowed to remove the brace for exercising, bathing, and dressing, but must keep the arm passively supported at 45 degrees in the plane of the scapula during those activities. If possible, it is helpful to instruct a family member in the techniques of donning and doffing the brace and supporting the arm during bathing and dressing. We also recommend that a patient utilize a slightly deflated beach ball for support during bathing.

Patients begin with pendulum exercises, elbow AROM, and hand squeezes within the first week after surgery. At the first postoperative visit, patients are instructed in phase I stretching exercises, which are supine passive forward elevation and external rotation with a cane or stick. Emphasis is placed on the patient achieving a tolerable, submaximal stretch several times per day rather than aggressive short bouts of stretching. The patient is asked to perform 10 to 20 repetitions with at least a 10-second hold, four to six times per day at home. The therapist also must assess whether the patient requires more supervised physical therapy during these initial 6 weeks after surgery. We have found that patients who achieve greater than 100 degrees of passive forward elevation or a 10-degree improvement in forward elevation during the first visit do well continuing with the home program on their own. In addition, patients must be able to demonstrate independence with the performance of these exercises. Patients who do not fit these criteria will be recommended for more supervised therapy. The surgeon may hold off beginning these ROM exercises until 3 to 6 weeks postoperatively for some patients with large or massive cuff tears.

Phase II

This stage typically begins 6 weeks after surgery. Patients are asked to continue performing the phase I ROM exercises. The supine forward elevation exercise may now be performed with a cane or stick to achieve end-range forward elevation passive range of motion (PROM). Patients are instructed in phase II ROM exercises and phase I strengthening exercises at this time. Caution must be employed with patients who have had large or massive rotator cuff tears. Restrictions in internal rotation ROM are to be expected due to the nature of the repair. Therefore, this exercise must be performed submaximally. Pain or weakness with the strengthening exercises may necessitate shorter arcs of motion. In some cases of a complex repair or where the integrity of the repair may be in question, rotator cuff strengthening may not be initiated until 8 to 12 weeks postsurgery.

TABLE 40-3
PENN PRESBYTERIAN MEDICAL CENTER SHOULDER AND ELBOW SERVICE REHABILITATION GUIDELINES FOR SMALL/MEDIUM ROTATOR CUFF TEARS FOLLOWING SURGICAL REPAIR

Phase I: 0–6 Weeks Postoperative

Goals:
1. Patient education
2. Permit healing
3. Control pain and inflammation
4. Initiate ROM exercises

Treatment:
Immediately postoperative or postoperative day 1:
1. Immobilized in sling
   Use sling for comfort and public only
2. Pendulum
3. Hand squeezes
4. Elbow AROM

Postoperative days 7–10:
1. Pendulums
2. Supine PROM forward elevation and ER (at 45 degrees in POS)
3. Heat and ice
4. Active scapular exercises (shoulder shrugs and scapular retraction)

Phase II: 6–8 Weeks Postoperative

Goals:
1. Improve to full ROM
2. Improve neuromuscular control and strength
3. Emphasize normal scapulohumeral rhythm

Treatment:
1. Continue all ROM exercises
2. Add phase II ROM exercises (extension, IR, and cross-body adduction)
3. Phase I strengthening (ER, IR, extension)
4. Submaximal manual resistance (ER/IR) with arm supported
5. Resisted scapular strengthening (with arms below shoulder height)

Phase III: 8–12 Weeks Postoperative

Goals:
1. Full pain-free PROM
2. Optimize neuromuscular control
3. Improve endurance
4. Initiate return to functional activities

Treatment:
1. Resisted scapular strengthening
2. Manual resistance for rotator cuff and deltoid
3. Progress to phase II strengthening (abduction, forward elevation, and ER at 45 degrees in POS with arm supported when at green for all phase I exercises)
4. Appropriate variable resistance and/or free-weight resistance
5. Strengthening in 45- to 90-degree position (keep pain free and in POS)

Phase IV: 16 Weeks–6 Months Postoperative

Goals:
1. Return to sport, occupation, or desired activities*
2. Promote concept of prevention

Treatment:
1. Work- or sport-specific exercises*
2. Work hardening*
3. Gradual return to sport or desired activities*

*Applies to athlete or laborer.
AROM, active range of motion; ER, external rotation; IR, internal rotation; POS, plane of the scapula; PROM, passive range of motion; ROM, range of motion.
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Patients who are not progressing as expected may be referred to supervised therapy. To help advance PROM, glenohumeral mobilizations and gentle, relatively pain-free manual stretching can be performed. To augment strengthening, manual resistance can be applied with alternating isometrics beginning with the arm supported at 45 degrees in the plane of the scapula (POS) and neutral rotation. Scapular strengthening exercises can also begin at this time period. Exercises should be performed with the arms below shoulder height to avoid increasing pain.

Phase III

This phase begins 12 weeks after surgery. The patient should have nearly full PROM for forward elevation (FE) and external rotation. It should be expected that internal rotation ROM will be only slightly better than at the beginning of phase II. It is important to assess AROM at this point. Many patients, especially those with large or massive rotator cuff repairs, may not be able to achieve greater than 90 degrees of active forward elevation. The therapist must evaluate whether this deficiency is due to weakness, stiffness, or lack of neuromuscular control. If near full PROM is present and there is good rotator cuff strength with the arm at the side, lack of neuromuscular control is the suspected culprit. It may have been 6 to 12 months since the rotator cuff worked in functional positions, and it is reasonable to suspect that it needs to be "retrained" to work in the elevated or end-range positions. In addition, the deltoid has not been used in this manner and most likely lacks the necessary strength to elevate the arm against gravity.

Patients who are unable to actively elevate the arm against gravity should also perform strengthening exercises

TABLE 40-4

PENN PRESBYTERIAN MEDICAL CENTER SHOULDER AND ELBOW SERVICE REHABILITATION GUIDELINES FOR LARGE/MASSIVE ROTATOR CUFF TEARS FOLLOWING SURGICAL REPAIR

Phase I: 0–6 Weeks Postoperative

Goals:
1. Patient education
2. Permit healing
3. Control pain and inflammation
4. Initiate ROM exercises

Immediately postoperative or postoperative day 1:
1. Patients may be immobilized in sling or abduction brace
   If sling, use for comfort and public
   If abduction brace, immobilized for 3–6 weeks
2. Pendulums
3. Hand squeezes
4. Elbow AROM

Postoperative days 7–10:
1. Pendulums
2. Supine PROM forward elevation and ER in appropriate patient
3. Heat and ice

Phase II: 6–12 Weeks Postoperative

Goals:
1. Improve to full ROM
2. Improve neuromuscular control and strength

Treatment:
1. Continue all ROM exercises (add phase I ROM exercises if not performing)
2. Add gentle phase II ROM (IR, cross-body adduction, and extension)
3. Submaximal manual resistance (ER/IR) with arm supported
4. Phase I strengthening (ER, IR, extension)
5. Resisted scapular strengthening (with arms below shoulder height)

Phase III: 12–16 Weeks Postoperative

Goals:
1. Full pain-free ROM
2. Optimize neuromuscular control
3. Improve endurance
4. Initiate return to functional activities

Treatment:
1. Continue all ROM and strengthening
2. Progress to phase II strengthening when at green for all phase I exercises (abduction, forward elevation, ER at 45 degrees in POS with arm supported)
3. Manual resistance for rotator cuff and deltoid

Phase IV: 16 Weeks–6 Months Postoperative

Goals:
1. Return to work, sport, or desired activities*
2. Promote concept of prevention

Treatment:
1. Work hardening
2. Gradual return to work or desired activity
3. Progress Bodyblade into elevated positions
4. Work-/sport-specific exercises

*Applies to athlete or laborer.
AROM, active range of motion; ER, external rotation; IR, internal rotation; POS, plane of the scapula; ROM, range of motion.
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in the supine position (Fig. 40-1). In this position, gravity is virtually eliminated and patients can "practice" raising their arm past 90 degrees while strengthening the deltoid. When the patient can comfortably perform 30 repetitions of this exercise, the head is slightly elevated to add the weight of gravity to the exercise. This sequence continues until the patient is able to raise the arm while standing upright. This exercise can be enhanced with the use of weighted balls or elastic resistance tied to the foot (Fig. 40-2).

Patients who are able to raise their arm against gravity and are able to perform the phase I strengthening exercises with green Thera-Band will be instructed in phase II strengthening exercises. These exercises are designed to begin training the rotator cuff and deltoid for functional demands. However, as always, pain is respected and the intensity of the exercises must be monitored.

Phase IV

This phase typically begins at 16 weeks and continues for up to 6 months postoperatively. At this time, lower-demand patients continue to gradually progress their home exercise program. Patients are encouraged to approach overhead activities with caution and whenever possible to use ladders or stools to raise their hand closer to the task so that the elbow can remain below shoulder level. They are again instructed in the biomechanics of lifting in an effort to reduce the risk of rotator cuff overload.

For the athlete, sport-specific training can begin utilizing plyometrics to enhance neuromuscular control, strength, and proprioception. Recommendations and instruction for proper use of gym equipment should also be done at this time. Patients should be encouraged to avoid exercises with the arm behind the plane of the body. Latissimus

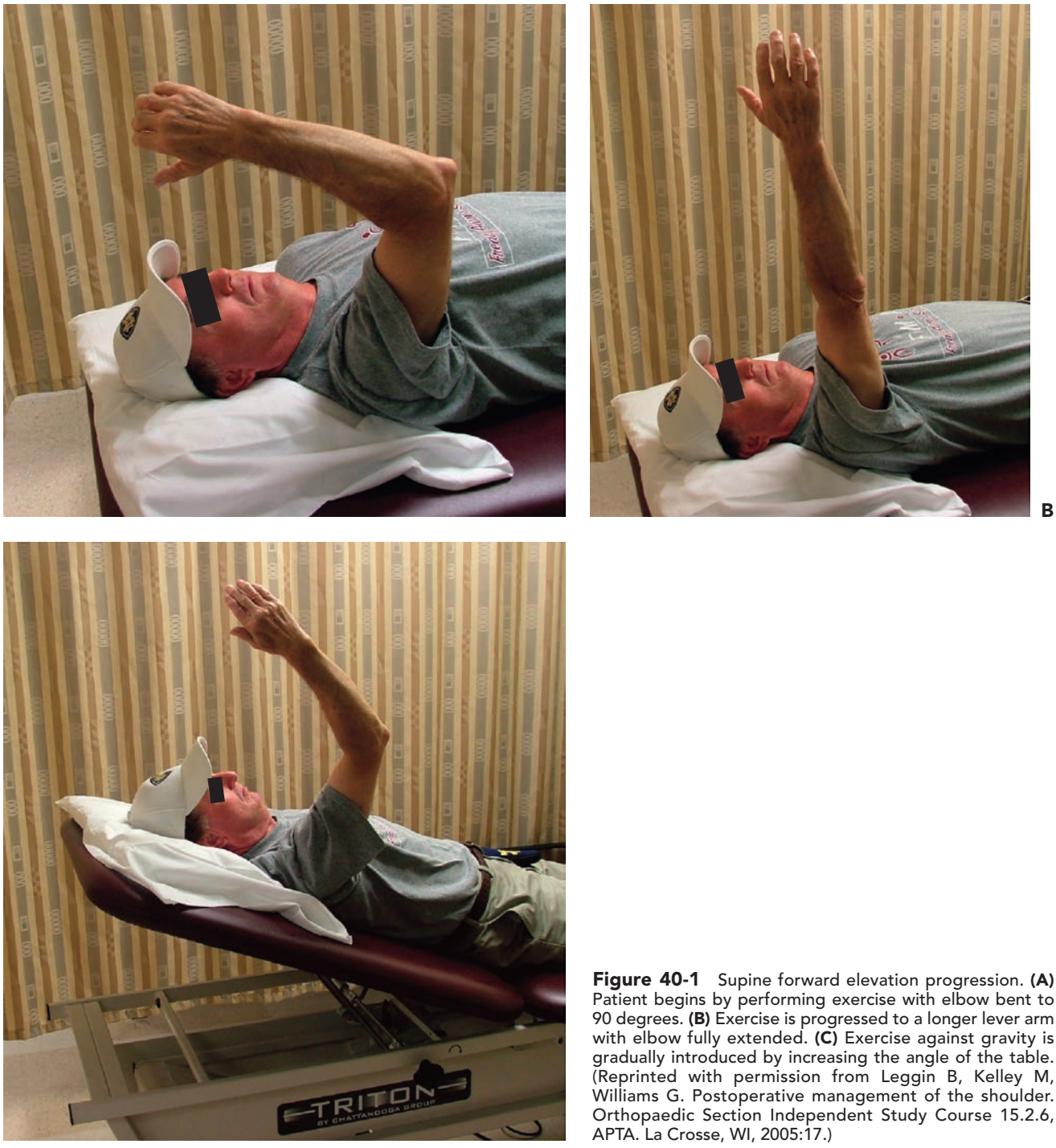

Figure 40-1 Supine forward elevation progression. **(A)** Patient begins by performing exercise with elbow bent to 90 degrees. **(B)** Exercise is progressed to a longer lever arm with elbow fully extended. **(C)** Exercise against gravity is gradually introduced by increasing the angle of the table. (Reprinted with permission from Leggin B, Kelley M, Williams G. Postoperative management of the shoulder. Orthopaedic Section Independent Study Course 15.2.6, APTA. La Crosse, WI, 2005:17.)

pull-downs should be performed to the chest, not behind the head. Caution should be employed when performing any type of "pushing" exercise such as chest press or shoulder press. It is safer to perform these exercises with a machine to allow for greater safety.

The patient who must return to work is gradually progressed to work-simulated activities. Emphasis is placed on simulating work activities in a safe, effective manner. The patient is educated on proper lifting mechanics and ergonomic modifications.

Latissimus Dorsi Tendon Transfer

Irreparable rotator cuff tears are characterized by the inability to achieve a direct repair of the native tendon to the proximal humerus despite mobilization of the remaining

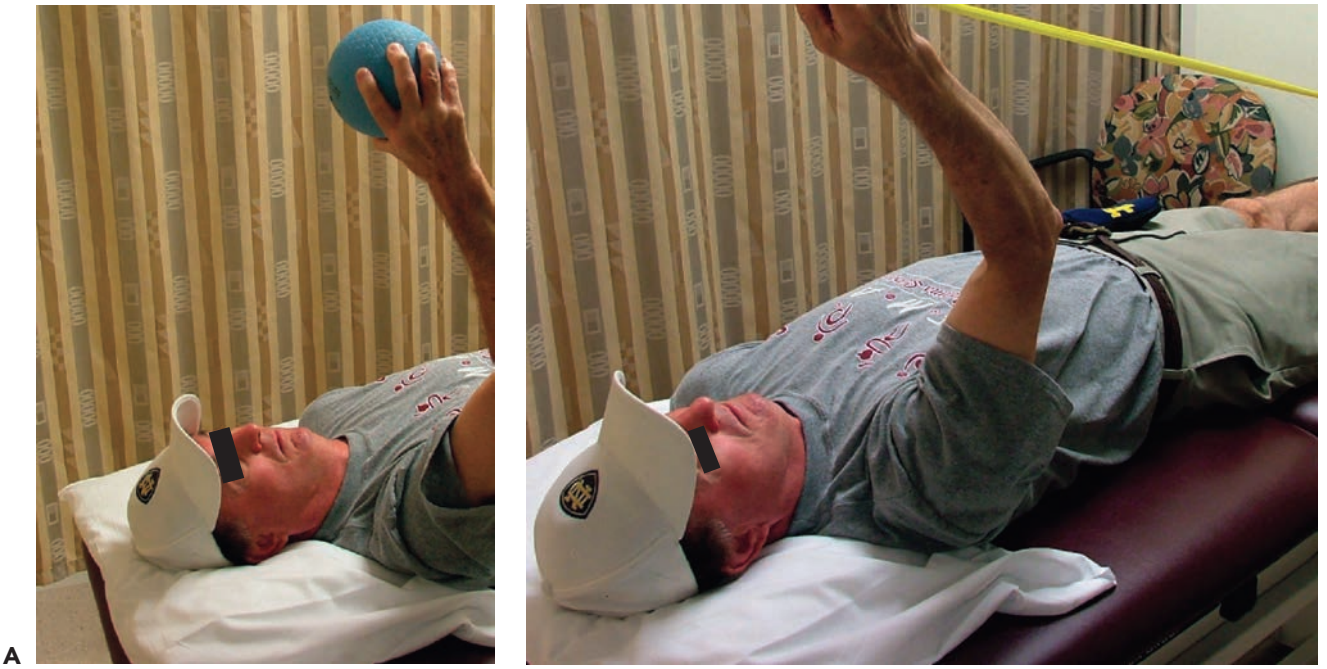

Figure 40-2 Supine forward elevation progression with weighted ball **(A)** and elastic resistance **(B).** (Reprinted with permission from Leggin B, Kelley M, Williams G. Postoperative management of the shoulder. Orthopaedic Section Independent Study Course 15.2.6, APTA. La Crosse, WI, 2005:17.)

tissue with conventional techniques of soft tissue release.[51] Surgical options that restore tendon continuity in recurrent, irreparable defects are limited by the duration of time since injury and inferior tissue quality.[51] Gerber et al.[52] described the technique of latissimus dorsi tendon transfer as a reconstructive option for irreparable posterosuperior rotator cuff defects. By replacing damaged and atrophied rotator cuff tissue with a healthy extrinsic tendon, latissimus dorsi tendon transfer is thought to restore an external rotation moment at the glenohumeral joint through both active contraction of the transferred muscle and the passive effect of tenodesis.[51–53]

Gerber reported that more than 80% of the 16 patients in his original study achieved good to excellent results.[53] The mean gain in active forward flexion was 52 degrees.[53] The mean adjusted Constant score was 73% overall and 82% when patients with a subscapularis lesion were excluded.[53] Aoki et al.[54] reported on 12 shoulders in 10 patients with irreparable rotator cuff tears treated with transfer of the latissimus dorsi. They reported excellent results in four shoulders, good in four, fair in one, and poor in three. Active forward elevation improved from a preoperative average of 99 degrees to a postoperative average of 135 degrees. Electromyography (EMG) revealed that nine of the 12 transferred muscles showed activity that was synergistic with the supraspinatus on external rotation with abduction.

Warner and Parsons[51] compared outcomes for 16 patients who underwent latissimus dorsi transfer as a salvage reconstruction for a failed rotator cuff repair with outcomes for six patients who underwent a primary reconstruction for irreparable cuff defect. There was a statistically significant difference in Constant score between groups, which measured 55% for the salvage group compared with 70% for the primary group.[51] Late rupture of the tendon transfer occurred in 44% of patients in the salvage group compared with 17% in the primary group.[51] Rupture had a statistically significant effect on the Constant score, with a mean decline of 14%. Therefore, these authors concluded that salvage reconstruction of failed prior rotator cuff repairs yields more limited gains in satisfaction and function than primary latissimus dorsi transfer.[51] Factors associated with more limited outcomes among patients in this study included poor tendon quality, severe fatty degeneration, and deltoid detachment.[51]

Rehabilitation following latissimus dorsi tendon transfer is very similar to the rehabilitation following massive rotator cuff tears, with a few caveats. For the first 6 weeks after surgery, patients may be immobilized in an abduction pillow or brace. Patients will perform pendulum exercises, hand squeezes, elbow AROM, supine passive forward elevation, and external rotation. After 6 weeks, gentle phase II ROM exercises (extension, cross-body adduction, internal rotation) will be added.

Six to 8 weeks after surgery, phase I strengthening exercises (internal rotation, external rotation, extension) with elastic resistance can be added. We have noticed that patients who do well following this procedure have greater internal rotation strength on the involved side than the

uninvolved side. Although the insertion of the latissimus dorsi has been relocated to the position of an external rotator, the muscle still contracts upon resisted adduction of the arm. Therefore, the latissimus needs to be retrained to act as an external rotator during elevation of the arm.

We begin to retrain the latissimus dorsi by positioning the patient's arm on a table at 45 degrees elevation in the POS and neutral rotation. The patient is asked to perform an isometric adduction contraction. At the same time the therapist positions the arm in slight external rotation. The patient is asked to hold the arm in this position. This "place-and-hold" technique is performed for all ranges of external rotation until the patient is able to perform a continuous arc of external rotation. The exercise is gradually progressed to greater degrees of elevation up to 90 degrees.

Another training technique we utilize is to position the patient supine with the elbow bent to 90 degrees. The therapist places his or her hands on the medial wrist and medial elbow of the patient. The patient is asked to simultaneously adduct and internally rotate into the therapist's hands while also raising the arm overhead. This simulates active elevation with a contraction of the latissimus to assist with centralization of the humeral head. This exercise is progressed by gradually raising the angle of the table and subsequently adding gravity until the patient can raise the arm against gravity. The latissimus never contracts without volitional control of the patient. Some patients are able to actively contract the latissimus while elevating the arm. These patients seem to achieve greater overhead function and power than those who are unable to learn this maneuver.

Beyond 12 weeks after surgery, emphasis is placed on improving the strength of the deltoid with both the supine elevation progression and elastic resistance in standing. Scapular strengthening exercises may also be added anytime after the 6-week postoperative period. Many patients are able to begin using the arm at shoulder level at this point. The patient will continue performing a home exercise program aimed at maximizing shoulder function for up to 6 to 12 months after surgery.

Glenohumeral Instability

Glenohumeral instability is defined as abnormal symptomatic translation of the humeral head relative to the glenoid. The incidence of anterior instability (80%) far exceeds that of posterior instability; however, some believe the true incidence of posterior instability (greater than 79%) is not appreciated due to spontaneous relocation and poor diagnostics.[55–57] The incidence of instability differs for those above and below 40 years of age, with a recurrence rate of more than 79% in those under 30 and 15% in those over 40.[56,58,59] The difference in recurrence rate is mostly related to activity level but also to connective tissue differences and associated pathology. One should recognize that the incidence of a rotator cuff tear in the older patient (over 40 years) who experiences an anterior dislocation can be as high as 85%.[60,61] Subluxation occurs when partial dissociation of the humerus and glenoid occurs, whereas dislocation occurs when the humerus and glenoid fully separate.

Instability can be classified several ways, the simplest of which are traumatic and atraumatic. Atraumatic instability can be further classified as voluntary and involuntary. Traumatic instability can result from a high-velocity uncontrolled end-range force causing a breach in the capsulolabral–bone interface or Bankart lesion. The acronym TUBS (*t*raumatic, *u*nilateral, *B*ankart lesion, *s*urgery indicated) is used to describe the characteristics of the traumatic group. Commonly these individuals require surgery to repair the Bankart lesion and to remain active and without recurrence of instability. A second acronym, AMBRI (*a*traumatic, *m*ultidirectional, *b*ilateral, *r*ehabilitation effective, *i*nferior capsular shift if surgery), is used to describe those who suffer from atraumatic instability. These individuals have a patulous capsule due to increased connective tissue elasticity, capsular stretching, or a combination of these two factors with dynamic stabilizer weakness. Commonly these individuals have symptomatic translation in multiple directions and are therefore described as having multidirectional instability. A strengthening program for the glenohumeral and scapulothoracic muscles may be beneficial to this group, but if they do not respond, a capsular shift may be required.[62]

A Bankart repair is performed to correct unidirectional shoulder instability by reattaching the detached labrum and associated glenohumeral ligaments with little disruption to the length or attachment of other structures around the shoulder. An open Bankart repair may involve detachment and later reattachment of the humeral insertion of the subscapularis and a reattachment of the labrum to the anterior glenoid with sutures through bone or with suture anchors.[63] It may also be necessary to reduce any capsular redundancy by tightening the anterior capsule with sutures. The disruption of the subscapularis has implications for postoperative rehabilitation. The surgeon will typically assess the amount of external range of motion available at the time of repair of the subscapularis. It is imperative the patient does not or is not stretched beyond this point during the first 6 to 8 weeks postoperatively. Greis et al. reported on several cases of subscapularis rupture within the first 4 weeks following surgery.[64] It should also be noted that open anterior stabilization can be associated with a 12-degree loss of shoulder external rotation.[65]

Recently, arthroscopic techniques to repair anterior unidirectional glenohumeral instability have been advocated. The advantages of arthroscopic repair include less invasive surgery, no damage to the subscapularis, and, therefore, less loss of external rotation than open procedures. Although there is no damage to the subscapularis during the arthroscopic repair, external rotation range of motion should still be restricted to 30 to 45 degrees during the first

6 to 8 weeks after surgery. Patients who have undergone arthroscopic stabilization may be held from performing PROM exercises for the first 6 weeks after surgery if the surgeon feels that they have adequate PROM during the first 7 to 10 days after surgery. These patients typically return 6 weeks after surgery with near full PROM.

A recent study compared two rehabilitation programs following arthroscopic Bankart repair.[66] One group was immobilized for 3 weeks after surgery prior to initiating ROM exercises. The second group began ROM and submaximal isometric exercises on the third postoperative day. There was no difference in recurrence rate, shoulder scores, return to activity, pain score, and ROM between the two groups at the final follow-up evaluation. However, the group who began rehabilitation sooner demonstrated a faster return to functional ROM and activity, as well as more satisfaction with the rehabilitation program.

Rehabilitation Following Bankart Repair

Phase I

Patient education is important in the early phases following surgery. Patients are instructed to use their arm for waist level activities when tolerated. They are cautioned about lifting anything, particularly by pushing the hands together (internal rotation), sleeping on the surgical side, leaning on the elbow, and making sudden movements. They are encouraged to use ice frequently throughout the day. When sitting, the patient is encouraged to position the arm on a pillow or armrest in slight abduction and the arm in neutral rotation. This helps to prevent prolonged internal rotation contractures.

The patient will be shown pendulum exercises the day of surgery and is to perform them four to six times per day (Table 40-5). Hand and wrist exercises are also encouraged.

TABLE 40-5
PENN PRESBYTERIAN MEDICAL CENTER SHOULDER AND ELBOW SERVICE REHABILITATION GUIDELINES FOLLOWING BANKART PROCEDURE

Phase I: 0–4 Weeks (Exercise 4–6 Times per Day)

Goals:
1. Patient education
2. Permit capsuloligamentous labral healing
3. Control pain and inflammation
4. Initiate ROM exercises

Treatment:
Postoperative day 1:
1. Educate patient on precautions
2. Pendulum exercises
3. Elbow AROM, hand-squeeze exercises
4. Ice (instruct patient on use of ice at home)

Postoperative days 7–10:
1. Continue with pendulum exercises
2. Phase I stretching
   Forward elevation
   ER at 45 degrees in POS (limit range to 30 degrees)

Phase II: 4–6 Weeks

Goals:
1. Decreased pain and inflammation
2. Normal arthrokinematics of glenohumeral and scapulothoracic joint
3. Improve strength

Treatment:
1. Continue with above treatment
2. Phase II stretching (extension, IR, cross-body adduction)
3. Manual resistance for glenohumeral and scapulothoracic stabilization
4. Phase I strengthening (at 6 weeks; ER, IR, extension)
5. Add shoulder shrugs and scapular retraction

Phase III: 6–12 Weeks

Goals:
1. Increase strength of rotator cuff and deltoid
2. Increase strength of scapular muscles
3. Increase total arm strength (biceps, triceps, forearms, etc.)
4. Initiate strengthening in provocative positions

Treatment:
1. Continue with above (decrease frequency of stretching exercises)
2. Add phase II strengthening when at green for phase I strengthening (abduction, forward elevation, ER at 45 degrees in POS); progress strengthening to more provocative positions
3. Variable resistance and/or free-weight resistance
5. Bodyblade progression
6. Plyoball progression (begin with chest pass)

Phase IV: 12–16 Weeks

Goals:
1. Initiate return to sport or occupational activity*

Treatment:
1. Bodyblade in overhead positions
2. Plyoball throwing
3. Work-/sport-specific activities*

*Applies to athlete or laborer.
AROM, active range of motion; ER, external rotation; IR, internal rotation; POS, plane of the scapula; ROM, range of motion.
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The patient will typically be asked to use the sling for comfort or when out in public. At 1 to 2 weeks postoperative, the patient is examined at the surgeon's office. If the surgeon feels that there is inadequate shoulder range of motion, the patient will begin phase I PROM exercises of forward elevation utilizing the opposite hand, and external rotation with the arm at 45 degrees in the POS utilizing a cane or stick. External rotation should not exceed 30 to 45 degrees or the amount of motion achieved in the operating room for an open procedure. Assessment of generalized hyperelasticity and hypoelasticity should be performed and will guide the progression of ROM exercise. If a hyperelastic patient presents with 150 degrees of elevation and 45 degrees of external rotation within the first 6 weeks following surgery, ROM exercises will be discouraged. However, if a hypoelastic patient presents with significant restriction within the first 6 weeks postsurgery, ROM and stretching will be encouraged.

Phase II

Depending on the status of the patient and quality of repair, the patient will be progressed to phase II ROM and phase I strengthening exercises at 4 to 6 weeks. Either an elastic band or free weights (0.5 to 2 lb) can be used. Stretching is continued in all directions. If the patient is being followed in a supervised manner, manual resistance starting at 45 degrees in the POS can be initiated using alternating isometrics. Scapular muscle integration and appropriate scapulohumeral rhythm is encouraged in all strengthening exercises. The patient can be progressed to phase II strengthening if he or she is able to perform phase I exercises with the third level of elastic resistance or a 4-lb dumbbell. If excessive stiffness is demonstrated, more progressive stretching is performed and joint mobilization techniques can be utilized. Rotational strengthening exercises may be progressed to 45 degrees of elevation.

Phase III

At this time the patient can be progressed to light-weight isotonics, using free weights or variable resistance units. Many times these exercises can be initiated in the latter part of phase II. Strengthening of the biceps and triceps can be initiated. The shoulder should remain in a protected position (less than 20 degrees of elevation) and the scapula "fixed" to integrate scapular muscle function into the exercise. A rowing exercise also can be performed with the same type of resistive equipment. Elastic band or free-weight strengthening is progressed in resistance, repetition, and position of elevation. Diagonal patterns (D2 and D1) can be incorporated into the program. The patient can progress to latissimus pull-downs performed in front of the body. The upper body ergometer (UBE) may be used with motion performed in each direction. The Bodyblade may be used in nonprovocative positions with progression to functional positions and increased time intervals of up to 60 seconds (Chapter 39, Fig. 39-27). The Plyoball progression may be used, particularly if the patient is a competitive or recreational athlete or laborer (Chapter 39, Figs. 39-28 and 39-29). Stretching should continue in this phase until full elevation is achieved and external rotation stretching is progressed to 90 degrees of abduction (90/90 position). External rotation at neutral still may be lacking up to 20 degrees, but external rotation at 90 degrees of elevation should be within 10 degrees of the other side. One must remember that collagen tissue continues to remodel for up to 12 months; therefore, further gains will be achieved over time.

The Athlete

The throwing athlete will be progressed at an accelerated pace, relative to external rotation stretching in phase II since this motion is critical to performance; however, stability cannot be sacrificed. In phase III the throwing or swimming athlete will be advanced to the provocative positions for their glenohumeral dynamic stabilizer exercises. Specific manual techniques may be emphasized for the glenohumeral dynamic stabilizers and scapular muscles. Cardiovascular exercise is strongly encouraged in the group.

Phase IV

Patients will be progressed to phase IV at 12 to 16 weeks and can begin to participate in their particular activities. The swimmer is encouraged to perform slow strokes. The basketball player can begin shooting. The thrower can begin light throwing of a tennis ball; the patient then moves back into an interval program over weeks 14 to 20. Patients can progress their variable resistance for a free program. Chest pressing can be initiated after 14 weeks; however, the degree of horizontal abduction may be limited and weight will be added gradually. We also encourage patients to begin chest presses with a machine that will allow restricted and protected movement.

Rehabilitation Following Anterior Capsulorrhaphy

Anterior capsulorrhaphy is performed to reduce the volume within a redundant capsuloligamentous complex (CLC). The degree of CLC redundancy can vary significantly. Thus, one may encounter a throwing athlete with minimal redundancy and "stiff" connective tissue as opposed to an individual with a very patulous CLC and "loose" connective tissue who has undergone unsuccessful stabilization procedures or has true multidirectional instability. These two types of athletes would be progressed very differently, but with the ultimate goal of allowing CLC

healing and optimizing neuromuscular control. In the throwing athlete, near-normal ROM would be encouraged, whereas in the individual with failed previous surgeries, ROM restrictions of 10 to 20 degrees may be encouraged. Phase I may be slightly more protracted after capsulorrhaphy as compared with phase I after an open Bankart procedure.

Phase I

Patient education is emphasized and is identical to that of the postoperative Bankart patient. The patient's arm is placed in a sling following surgery. The initiation of pendulum exercises varies based on the patient's connective tissue elasticity and the surgeon's preference. Pendulum exercises typically are started within the first 4 weeks (Table 40-6). Some patients may not begin any type of ROM exercises until 6 weeks.

Phase II

After 4 to 6 weeks the patient may begin PROM forward elevation with the opposite hand and passive external rotation with the arm placed in 45 degrees in the POS. Forward elevation may be limited to 90 degrees and external rotation may be limited to neutral until 6 to 8 weeks after surgery. At this point, the patient can begin phase II ROM exercises and phase I strengthening exercises using elastic band and/or free weights (0.5 to 2 lb). Elevation exercises are continued, moving toward full range. External rotation stretching may be limited to no more than 45 degrees in the POS. External rotation at 90 degrees in the POS is checked and should not progress beyond 70 degrees. The goal is for this patient to have some degree of tightness by the end of 12 weeks. Approximately 10 to 20 degrees of limited motion in all planes is desired at 12 weeks because continued collagen remodeling will occur over time. This may vary depending on the extent of the capsulorrhaphy

TABLE 40-6
PENN PRESBYTERIAN MEDICAL CENTER SHOULDER AND ELBOW SERVICE REHABILITATION GUIDELINES FOLLOWING ANTERIOR CAPSULORRAPHY

Phase I: 0–4 Weeks

Goals:
1. Patient independent with precautions and home exercise program prior to discharge from hospital (typical inpatient hospital stay = 1 day).
2. Permit capsular healing
3. Control pain and inflammation
4. ROM exercises will be initiated depending on surgeon's preference

Postoperative day 1:
1. Educate patient on precautions
2. Pendulum exercises (25 times in each direction), depending on surgeon
3. Elbow AROM, hand-squeeze exercises
4. Ice (instruct patient on use of ice at home)

Phase II: 4–6 Weeks

Goals:
1. Decreased pain and inflammation
2. Normal arthrokinematics of glenohumeral and scapulothoracic joint
3. Improved strength

Treatment:
1. Continue with above treatment
2. Add phase I stretching (forward elevation and ER in POS); limit ER to 45 degrees
3. Manual resistance for glenohumeral and scapulothoracic stabilization
4. Add phase I strengthening
5. Add shoulder shrugs and scapular retraction
6. Bodyblade in POS

Phase III: 6–12 Weeks

Goals:
1. Increase strength of rotator cuff and deltoid
2. Increase strength of scapular muscles
3. Increase total arm strength (biceps, triceps, forearms, etc.)
4. Initiate strengthening in provocative positions

Treatment:
1. Continue with above (decrease frequency of stretching exercises)
2. Add phase II stretching (extension, IR, cross-body adduction)
3. Add phase II strengthening (abduction, forward elevation, ER at 45 degrees in POS)
4. Variable resistance and/or free-weight resistance
5. Bodyblade in nonprovocative positions with progression to functional positions
6. Plyoball progression (begin with chest pass)

Phase IV: 12–16 Weeks

Goals:
1. Initiate return to sport or occupational activity*

Treatment:
1. Bodyblade in overhead positions
2. Plyoball throwing
3. Work-/sport-specific activities*

*Applies to athlete or laborer.
AROM, active range of motion; ER, external rotation; IR, internal rotation; POS, plane of the scapula; PROM, passive range of motion; ROM, range of motion.
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and if the patient has hyperelastic tissue. Manual strengthening techniques using alternating isometrics in the midrange are strongly emphasized. Although the capsule has been tightened, it is critical that the glenohumeral dynamic stabilizer function be optimized. Short arc manual resistive exercises and/or the use of free weights are utilized in an effort to maximize these muscles in their stabilization function across the glenohumeral joint. Scapular muscle integration is again strongly emphasized. Use of the UBE may begin, again performed in both directions. The Bodyblade can be started toward the end of the phase at 10 to 12 weeks, starting in the nonprovocative position.

Phase III

The patient follows the same regimen as described for that following the Bankart procedure. Again, strong emphasis is placed in midrange function with strengthening being performed and progressed toward the end range. The use of the Plyoball will be based on the functional demands of the patient. If the patient is an athlete or laborer and therefore requires use of the arm in provocative positions, then plyometrics will be utilized in phase III. Various resistance exercises are also performed.

Phase IV

The patient is progressed to activities of phase IV at approximately 16 weeks. If the patient is a throwing athlete, an interval program is initiated. For a swimming athlete, progression will be slow, with initial work on stroke and technique and finally distance. The patient will not be encouraged to aggressively stretch as many swimmers tend to do because such an action may stretch out the reconstruction. A gradual return of ROM will be encouraged in the next 6 to 9 months.

Rehabilitation Following Thermal Capsulorraphy

The arthroscopic application of thermal energy to selectively shrink the glenohumeral joint capsule has been developed over the past 8 to 10 years.[67–70] Several authors have reported 82% to 93% return to competition following thermal-assisted capsulorraphy procedures in overhead athletes.[71–74] However, the use of thermal capsulorraphy to correct shoulder instability may be declining due to poor outcomes reported with this procedure in some patients.[75]

Phase I

Since the early capsular tensile strength is presumed to be weak, the early rehabilitation program is more conservative than other stabilization procedures.[76] The goal in the immediate postoperative period of 4 to 6 weeks is to allow healing of the tissue. This phase consists of distal arm strengthening exercises and providing pain relief.[76] Patients are encouraged to keep their arm in a sling most of the day and perform hand squeezes and elbow ROM exercises. They are able to use their arm for waist-level activities and basic activities of daily living.

Phase II

This phase begins 4 to 6 weeks after surgery. Range of motion and rotator cuff strength are assessed at this time. Many patients present with full passive forward elevation and little restriction of external rotation. Patients who may have a restriction in these two motions will be instructed in pendulum exercises and phase I ROM exercises such as forward elevation and external rotation. The patient is encouraged to apply a gentle stretch to the end range of motion to allow gradual and progressive restoration of range of motion.

Phase I strengthening exercises with elastic resistance for external rotation, internal rotation, and extension are initiated at this time as well. Scapular strengthening exercises such as scapular retraction without resistance and progressing toward elastic resistance can also be added at this time. Manual resistance for alternating isometrics to promote stability and proprioception are also useful at this stage. Scapular muscle integration and appropriate scapulohumeral rhythm is encouraged in all strengthening exercises. Patients can be progressed to phase II strengthening if they are able to perform phase I exercises with the third level of elastic resistance or a 4-lb dumbbell.

Phase III

Patients should have achieved nearly full or full range of motion and rotator cuff strength relative to the opposite extremity by 12 weeks postoperative.[77] At this time the patient can be progressed to light-weight isotonics, using free weights or variable resistance units. Many times these exercises can be initiated in the latter part of phase II. Strengthening of the biceps and triceps can also be initiated. A seated row exercise with resistive equipment can be performed. Elastic band or free-weight strengthening is progressed in resistance, repetition, and position of elevation. Diagonal patterns (D2 and D1) can be incorporated into the program. The patient can progress to latissimus pull-downs performed in front of the body. It is very important to emphasize this position and discourage the performance of this exercise with the bar brought behind the head due to the potential stress on the anterior glenohumeral joint. The UBE may be used with motion performed in each direction. The Bodyblade may be used in nonprovocative positions with progression to functional positions and increased time intervals of up to 60 seconds (Chapter 39, Fig. 39-27). The plyoball progression may be

used, particularly if the patient is a competitive or recreational athlete or laborer (Chapter 39, Figs. 39-28 and 39-29).

The Athlete

The throwing athlete will be progressed at an accelerated pace, relative to external rotation stretching in phase II since this motion is critical to performance; however, stability cannot be sacrificed. In phase III throwing or swimming athletes will be advanced to the provocative positions for their glenohumeral dynamic stabilizer exercises. Specific manual techniques may be emphasized for the glenohumeral dynamic stabilizers and scapular muscles. Cardiovascular exercise is strongly encouraged in the group.

Phase IV

Patients will be progressed to phase IV at 12 to 16 weeks and can begin to participate in their particular activities. The swimmer is encouraged to perform slow strokes. The basketball player can begin shooting. The thrower can begin light throwing of a tennis ball; the patient then moves back into an interval program over weeks 14 to 20. Patients can progress their variable resistance for a free program. Chest pressing can be initiated after 14 weeks; however, the degree of horizontal abduction may be limited and weight will be added gradually. We also encourage patients to begin chest presses with a machine that will allow restricted and protected movement.

SLAP Lesions

Superior labrum anterior-posterior (SLAP) lesions have been classified into four distinct categories based on the labral injury and the stability of the labrum–biceps complex found at arthroscopy.[78] Subsequent authors have added additional classification categories and specific subtypes.[79–81] Type I lesions denote fraying and degeneration of the superior labrum with a normal biceps tendon anchor. Type II lesions may have fraying of the superior labrum, but their hallmark is a pathologic detachment of the labrum and biceps anchor from the superior glenoid. In type III SLAP lesions, the superior labrum has a vertical tear analogous to a bucket-handle tear in the meniscus of the knee. A type IV pattern involves a vertical tear of the superior labrum, but this superior labral tear extends to a variable extent up into the biceps tendon as well. The torn biceps tendon tends to displace with the labral flap into the joint, whereas the biceps anchor itself remains firmly attached to the superior glenoid. Last, a complex of two or more SLAP lesions may occur, with the most common presentation being a type II and a type IV.[82]

Surgical treatment of SLAP lesions is generally performed as described by Snyder et al.[78,82] Type I lesions, seen as significant fraying about the superior labrum, are débrided with a shaver placed through the anterior portal.[83] Type II lesions, in which the biceps origin and superior labrum are detached from the bony base of the glenoid, are treated, depending on the instability of the lesion and the age of the patient.[83] Some type II lesions are relatively stable and can be treated by débridement and bony abrasion alone.[83] When the labral detachment is significant and it can easily be pulled off of the superior glenoid, either repair or tenodesis should be considered.[83] Younger patients and those who engage in overhead activities are treated with repair. Tenodesis may be preferred for some patients generally over the age of 45 and who do not regularly participate in overhead activities.

Treatment of type III lesions is generally accomplished with débridement of the flapped labrum.[83] Type IV lesions are treated depending on the amount of biceps tearing. Those lesions with less than 25% extension into the biceps tendon are treated with débridement alone.[83] Those with more than 25% extension into the biceps tendon are treated with either repair in younger patients or tenodesis in older patients.[83]

The characteristics of rehabilitation following a SLAP lesion débridement or repair are very similar to those found in the rehabilitation following rotator cuff repair and Bankart lesion. Any position, which may create tension on the biceps, should be avoided during the first 6 weeks following surgery and approached with caution thereafter. These positions include shoulder extension, internal rotation behind the back, and using the arm to carry or lift objects with the elbow extended. In addition, external rotation with the arm at 90 degrees of abduction should be approached with caution. When a biceps tenodesis is performed, any resistive active motion of the elbow, either in flexion or supination, is avoided.[83]

Phase I

Patients are instructed in the precautions as described previously. A sling is used for comfort during the first 7 to 10 days following surgery. Phase I ROM exercises are then initiated and performed to tolerance (Table 40-7). The surgeon may request that external rotation range of motion be limited to 45 degrees in patients who have evidence of a peel-back tear. Patients are expected to achieve full passive forward elevation 6 weeks following surgery.

Phase II

This phase begins 6 weeks after surgery. Active and passive range of motion as well as rotator cuff and deltoid strength are assessed at this time. Patients are instructed in phase II ROM exercises (extension, internal rotation, cross-body adduction) and phase I of the strengthening exercises (external rotation, internal rotation, extension). Scapular retraction exercises with elastic resistance can also be performed at this time.

TABLE 40-7
PENN PRESBYTERIAN MEDICAL CENTER SHOULDER AND ELBOW SERVICE REHABILITATION GUIDELINES FOLLOWING SLAP REPAIR

Phase I: 0–6 Weeks (Exercise 3–5 Times per Day)

Goals:
1. Patient education
2. Permit capsuloligamentous labral healing
3. Control pain and inflammation
4. Initiate ROM exercises

Treatment:
Postoperative day 1—first postoperative visit:
1. Educate patient on precautions
2. Ice (instruct patient on use of ice at home)

Postoperative days 7–10:
1. Educate patient on precautions
2. Instruct in pendulum exercises
3. Phase I stretching
   Forward elevation
   ER at 45 degrees in POS (limit range to 30 degrees)

Phase II: 6–8 Weeks

Goals:
1. Decreased pain and inflammation
2. Normal arthrokinematics of glenohumeral and scapulothoracic joint
3. Improve strength

Treatment:
1. Continue with above treatment
2. Phase II stretching (extension, IR, cross-body adduction)
3. Manual resistance for glenohumeral and scapulothoracic stabilization
4. Phase I strengthening (at 6 weeks; ER, IR, extension)
5. Add shoulder shrugs and scapular retraction

Phase III: 8–12 Weeks

Goals:
1. Increase strength of rotator cuff and deltoid
2. Increase strength of scapular muscles
3. Increase total arm strength (biceps, triceps, forearms, etc.)
4. Initiate strengthening in provocative positions

Treatment:
1. Continue with above (decrease frequency of stretching exercises)
2. Add phase II strengthening when at green for phase I strengthening (abduction, forward elevation, ER at 45 degrees in POS); progress strengthening to more provocative positions
3. Variable resistance and/or free-weight resistance
4. Bodyblade in nonprovocative positions and progress to functional positions
5. Plyoball progression (begin with chest pass)

Phase IV: 12–16 Weeks

Goals:
1. Initiate return to sport or occupational activity*

Treatment:
1. Bodyblade in overhead positions
2. Plyoball throwing
3. Work-/sport-specific activities*

*Applies to athlete or laborer.
ER, external rotation; IR, internal rotation; POS, plane of the scapula; ROM, range of motion.
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Phase III

This phase begins 10 to 12 weeks after surgery and continues until week 16. Patients who are able to perform the phase I strengthening exercises with the green band are instructed in phase II strengthening (abduction, forward elevation, external rotation supported at 45 degrees). Advanced scapular strengthening exercises may be employed at this time. In addition, biceps strengthening with light weights may begin.

Phase IV

This phase typically begins at 16 weeks and continues for up to 6 months postoperatively. Patients are instructed in the biomechanics of lifting in an effort to reduce the risk of overload. For the athlete, sport-specific training can begin utilizing plyometrics to enhance neuromuscular control, strength, and proprioception. Recommendations and instruction for proper use of gym equipment should also be done at this time. Patients should be encouraged to avoid exercises with the arm behind the plane of the body. Latissimus pull-downs should be performed to the chest, not behind the head. Caution should be employed when performing any type of "pushing" exercise such as chest press or shoulder press. It is safer to perform these exercises with a machine to allow for greater safety.

The patient who must return to work is gradually progressed to work-simulated activities. Emphasis is placed on simulating work activities in a safe, effective manner. The patient is educated on proper lifting mechanics, ergonomic modifications, and common sense.

PROXIMAL HUMERUS FRACTURES

Fractures of the proximal humerus can produce as many as four major fracture fragments.[84] These include the head, the greater tuberosity, the lesser tuberosity, and the shaft of the proximal humerus. Neer introduced a four-part classification system based on the anatomic and biomechanical forces that contribute to the displacement of the fracture fragments.[85,86] Displacement is defined as greater than or equal to 1 cm of translation or 45 degrees of angulation from the fragment's normal anatomic position as seen on radiographs.[86] A proximal humerus fracture in which none of the major fragments is displaced is a one-part or nondisplaced proximal humerus fracture.

Management of proximal humerus fractures varies depending on the type and amount of displacement. A majority of proximal humerus fractures are nondisplaced and do not require surgery.[86–89] In these cases, the patient's arm is placed in a sling with the arm at the side. In some two-, three-, and four-part proximal humerus fractures, surgery is indicated. The preferred methods include closed reduction, open reduction and internal fixation, and prosthetic arthroplasty.[90] The type of fracture present as well as the medical and functional status of the patient determine the type of procedure to be performed.[90]

Communication between the therapist, surgeon, and patient is essential to successful rehabilitation following proximal humerus fracture.[85] The rate of exercise advancement is coordinated with the surgeon and depends on the severity of the fracture, stability of reduction, and formation of callus.[85] In most cases, the patient has never had a previous shoulder injury. Therefore, the patient should be educated on the importance of performing a home exercise program, techniques for pain relief, the length of the rehabilitation process, and expected outcome. Several factors affect outcome, including patient age and compliance with the home exercise program, complexity of the fracture, type of reduction, and soft tissue involvement.[85]

A detailed history should be obtained from the patient and should include mechanism of injury, presence of additional injuries, prior functional status, and patient's goals. Proximal humerus fractures result primarily from low bone mass and falls.[91–95] These fractures appear to be associated with fractures and fall history; medical conditions such as epilepsy, depression, and diabetes; use of seizure medication; and left-handedness.[91] To help prevent another proximal humerus fracture, recommendations for those at risk for a proximal humerus fracture may include moderate levels of physical activity, use of calcium carbonate tablets as a calcium supplement, other measures to reduce loss of bone mass and prevent falls, and maintaining a high dietary calcium intake.[91] If the physician has not provided a detailed description of the type and stability of the fracture, the therapist should obtain this information prior to initiating movement of the extremity. The therapist should also perform a good neurovascular examination of the distal extremity.

Phase I

The rehabilitation process may begin when the surgeon is confident that adequate stabilization of the fracture has been achieved. This can be as early as the first postoperative day in many cases. In those cases in which surgery has not been performed or less than ideal fixation has been achieved, the rehabilitation program may require modification.

The importance of performing the home exercise program four to six times per day should be emphasized to the patient. Pain is a major consideration in this population and should always be respected. Many patients with proximal humerus fractures have never had shoulder pain similar to the magnitude they now experience; therefore, it is occasionally more difficult to motivate them to perform their home exercise program. Initial exercises include pendulums, supine passive forward elevation with the opposite hand, and supine passive external rotation with a stick. Patients are slowly advanced to passive extension, internal rotation, and cross-body adduction at approximately 6 weeks postfixation. Special consideration is given to those patients with greater tuberosity fractures because these exercises provide tension to the rotator cuff and its attachment. If excessive tightness is determined, joint mobilization is implemented to facilitate the return of motion.

Phase II

Approximately 6 to 8 weeks postfixation, the patient should have achieved an improvement in passive ROM. Rotator cuff strengthening with isometrics and/or Theraband can begin at this time. In addition, scapular strengthening exercises also may be initiated. If the patient is being followed in supervised therapy, manual isometrics at 45 degrees in the POS can be introduced.

Phase III

Approximately 8 to 12 weeks postfixation, the patient should have 80% full passive ROM and good rotator cuff strength. Phase III strengthening exercises with the elastic band can begin at this time. These exercises include abduction to 45 degrees, forward elevation, and external rotation at 45 degrees. Progression of resistance for shoulder shrugs, scapular retraction, biceps curls, and triceps extension is also accomplished. For patients who are being followed in supervised therapy, manual resistance in unsupported positions can be progressed utilizing alternating isometrics and proprioceptive neuromuscular facilitation (PNF) diagonals.

GLENOHUMERAL ARTHRITIS

Several diseases affect the osteoarticular surface of the glenohumeral joint and can result in the need for surgical intervention. The most common among these disorders include degenerative osteoarthritis, traumatic arthritis, osteonecrosis, arthritis of glenohumeral instability, rheumatoid arthritis, rotator cuff arthropathy, and crystal-induced arthritis.[90,96–98] Other less common disorders include hemophilic arthropathy, Paget's disease, psoriasis, and ochronosis.[90] Arthritic conditions can cause deformity of the normal articulating surfaces, loss of the articular cartilage, and synovitis associated with generalized joint inflammation.[90] As a result, the patient may experience joint pain, instability, limited active and passive ranges of motion, and decreased strength, which limits the patient's daily, recreational, and work activities.[90] The primary indication for prosthetic arthroplasty is pain that limits functional activity and is not improved with conservative management.[90] Limited ROM in the absence of pain is not an indication for surgery.[98]

Prosthetic joint replacement can include total shoulder arthroplasty or hemiarthroplasty (proximal humeral replacement). Indications for hemiarthroplasty include conditions in which the glenoid is intact, such as acute proximal humerus fractures, avascular necrosis, and younger patients.[90,98–100] Another indication for hemiarthroplasty is patients who have massive, irreparable deficiencies of the rotator cuff such as those with cuff tear arthropathy, crystal-induced arthritis, and rheumatoid arthritis.[29,30,100] Patients with massive rotator cuff deficiency have a higher incidence of glenoid component loosening than patients with the same disease process but an intact rotator cuff.[100] Therefore, hemiarthroplasty is the preferred procedure for these patients.[90] Total shoulder arthroplasty in patients with an intact rotator cuff and significant glenoid degeneration will result in improved shoulder mechanics and postoperative pain level.[90,97,98]

Postoperative Rehabilitation

Good communication between the rehabilitation specialist and orthopedic surgeon is critical to successful outcome after shoulder arthroplasty. Rehabilitation after shoulder arthroplasty depends on the underlying diagnosis, integrity of the rotator cuff, and variations in surgical technique. The rehabilitation specialist should know the amount of external rotation and forward elevation achieved by the surgeon at the time of wound closure.[90] The kind of joint stability and quality of the subscapularis repair should be determined. The patient's goals and motivation to participate in the rehabilitation process are also significant factors affecting outcome. Neer recommended classifying patients into standard goals and limited goals categories.[98] Those patients with good preoperative ROM and rotator cuff function are placed into the standard goals category (Table 40-8). Patient's with poor postoperative ROM and/or poor or ruptured rotator cuffs are classified as having limited goals. It is essential for the rehabilitation specialist to have knowledge of as much of these factors as possible prior to initiating the rehabilitation program (Table 40-9).

Phase I

Patients are typically hospitalized for 2 days after surgery. During this time, emphasis is placed on patient education and a core of essential ROM exercises. The rehabilitation process begins the morning of the first postoperative day with patient education. The patient is told to expect swelling and discoloration of the affected extremity and occasionally of the chest wall as a result of the surgery. With the exception of special cases, patients are encouraged to refrain from using the sling unless they are in public or experiencing discomfort. They are also instructed in the use of ice for control of pain and inflammation. Patients can use their extremity for waist-level activities and bring their hand to the mouth with the elbow held at the side. Patients are also asked to not sleep on or make sudden movements with the operated side. In addition, they are asked to avoid lifting, carrying, pushing, pulling, and leaning on the affected side. Evaluation should include documentation of distal neurovascular status and passive arcs of forward elevation and external rotation. In addition, knowledge and documentation of external rotation, forward elevation, and ROM achieved by the surgeon in the operating room is important. To respect the healing of the subscapularis, the patient should be instructed to not exceed the external rotation limitation during the first 6 weeks after surgery.

It should be emphasized that all exercises are to be performed four to six times per day. To help maintain distal upper-extremity strength and decrease distal extremity swelling, the patient is instructed in hand-squeezing exercises and elbow-active ROM. The patient is also instructed in pendulum exercises, which are to be performed 25 times in each direction. Supine passive forward elevation with the opposite hand and external rotation with a stick or the opposite hand holding the forearm are instituted in the afternoon session on the first postoperative day. Patients are asked to perform these exercises in their room when they are not being seen by the therapist. Criteria for discharge from the hospital include independence with the exercise program and precautions, passive forward elevation of 120 degrees, and passive external rotation of 20 degrees.

Patients in the limited goals category often have bilateral shoulder disease or polyarticular arthritis and may require modification of the exercise program. This may include help from a family member or arrangement of home or outpatient therapy.

TABLE 40-8

PENN PRESBYTERIAN MEDICAL CENTER SHOULDER AND ELBOW SERVICE REHABILITATION GUIDELINES FOLLOWING TOTAL SHOULDER ARTHROPLASTY

Phase I: 0–3 Weeks (Exercise 4–6 Times per Day)

Goals:
1. Patient education
2. Allow healing of subscapularis
3. Control pain and inflammation
4. Initiate ROM exercises

Postoperative day 1 (a.m. session):
1. Educate patient on precautions
2. Pendulum exercises
3. Elbow AROM, hand-squeeze exercises
4. Ice (instruct patient on use of ice at home)

Postoperative day 1 (p.m. session):
1. Review precautions
2. Pendulums, elbow AROM, and hand squeezes
3. Supine passive forward elevation stretching in POS
4. Supine passive ER stretching in POS (within limits of range achieved in OR)
5. Ice

Postoperative days 2–5:
1. Continue with above until patient is independent with home exercises and precautions
2. Begin light ADLs (hand to mouth, writing, etc.)
3. Ice

Postoperative days 7–10 (first MD visit postoperative):
1. Review home exercise program
2. Add phase II stretching (if good tissue quality) PROM extension, IR, and cross-body adduction

Phase II: 3–8 Weeks

Goals:
1. Decreased pain and inflammation
2. Increased ADLs
3. Continue stretching until full PROM is achieved
4. Initiate strengthening exercises

Treatment:
1. Review all exercises and precautions
2. Add phase II stretching (if not already)
3. Initiate light isometrics for rotator cuff (may omit IR depending on healing of subscapularis)
4. Progress to phase I strengthening at 4–6 weeks (ER, IR, extension)
5. Scapular strengthening (shoulder shrugs, scapular retraction)

Phase III: 6–12 Weeks

Goals:
1. PROM full and pain free
2. Increase functional activities
3. Increase strength of scapular stabilizers

Treatment:
1. Phase II strengthening (abduction, forward elevation, ER at 45 degrees in POS)
2. Upper-extremity PNF diagonals
3. Progress resistance of shoulder shrugs, scapular retraction, biceps, triceps

Phase IV: 12–16 Weeks

Goals:
1. Full functional activities
2. Return to work or sport*

Treatment:
1. Work- or sport-specific training*
2. Suggest modifications to work, sport, or functional activities*

*Applies to athlete or laborer.
ADL, activity of daily living; AROM, active range of motion; ER, external rotation; IR, internal rotation; OR, operating room; PNF, proprioceptive neuromuscular facilitation; POS, plane of the scapula; PROM, passive range of motion; ROM, range of motion.
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Phase II

Patients continue all stretching exercises until full passive ROM is achieved. At 3 to 6 weeks after surgery, patients in the standard goals category should be able to perform many of their waist-level activities of daily living. At 6 weeks after surgery, phase II ROM exercises (extension, internal rotation, and cross-body adduction) and phase I strengthening exercises with elastic bands can be instituted and progressed. When the patient is able to perform all phase I exercises with the green band, phase II strengthening exercises can begin. In addition, shoulder shrugs and scapular retraction exercises can begin.

Patients who are having difficulty achieving full passive ROM may be followed in supervised therapy. Glenohumeral joint mobilizations and gentle manual stretching can be initiated to help improve ROM. The patient may also be given a pulley for home use to help achieve full passive forward elevation. Manual resistance to external and internal rotation with the arm supported at 45 degrees in the POS can be initiated using alternating isometrics.

Patients in the limited goals category will begin the phase II stretching exercises at 3 to 6 weeks postoperatively. They may also initiate submaximal rotator cuff isometrics and scapular strengthening at this time.

TABLE 40-9

PENN PRESBYTERIAN MEDICAL CENTER SHOULDER AND ELBOW SERVICE REHABILITATION GUIDELINES FOLLOWING TOTAL SHOULDER ARTHROPLASTY—LIMITED GOALS

Phase I: 0–3 Weeks (Exercise 4–6 Times per Day)

Goals:
1. Stress importance of precautions and performance of home exercise program
2. Allow healing of subscapularis
3. Control pain and inflammation
4. Initiate ROM exercises (instruct family member, etc., in exercises)

Postoperative day 1 (a.m. session):
1. Educate patient on precautions
2. Pendulum exercises
3. Elbow AROM, hand-squeeze exercises
4. Ice (instruct patient on use of ice at home)

Postoperative day 1 (p.m. session):
1. Review precautions
2. Patient performs pendulums, elbow AROM, and hand squeezes
3. Supine passive forward elevation stretching in POS*
4. Supine passive ER stretching in POS (within limits of range achieved in OR)*

Postoperative days 2–5:
1. Continue with above until patient is independent with home exercises and precautions
2. Add standing AAROM extension with stick
3. Begin light ADLs (hand to mouth, writing, etc.)
4. Ice

Postoperative days 7–10:
1. Review home exercise program

Phase II: 3–8 Weeks

Goals:
1. Decrease pain and inflammation
2. Increase ADLs

Treatment:
1. Review all exercises and precautions
2. Phase II stretching (PROM extension, IR, and cross-body adduction)
3. Submaximal rotator cuff isometrics

Phase III: 8–12 Weeks

Goals:
1. ROM full and pain free
2. Increase functional activities
3. Begin rotator cuff strengthening

Treatment:
1. Continue with above
2. Phase I strengthening (ER, IR, extension)

Phase IV: 12–16 Weeks:

Goals:
1. Return to functional activities
2. Continue to improve strength

Treatment:
1. Continue with all stretches and strengthening
2. Add scapular strengthening
3. Add phase II strengthening if able

*Instruct patient or caregiver.
ADL, activity of daily living; AAROM, active assisted range of motion; AROM, active range of motion; ER, external rotation; IR, internal rotation; OR, operating room; POS, plane of the scapula; PROM, passive range of motion; ROM, range of motion.
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Phase III

Twelve weeks after surgery, the patient should have 80% full and pain-free passive ROM, as well as good rotator cuff strength. Phase II strengthening exercises with the elastic band can usually begin if they have not already. These exercises include abduction to 45 degrees, forward elevation, and external rotation with the arm supported at 45 degrees. Progression of resistance for shoulder shrugs, scapular retraction, biceps curls, and triceps extension is accomplished. For patients who are being followed in supervised therapy, manual resistance in unsupported positions can be progressed utilizing alternating isometrics and PNF diagonals. Patients with limited goals should have adequate soft tissue healing and stability of the glenohumeral components and can begin phase I strengthening exercises.

Evaluation of the integrity of the subscapularis may also be performed at this time. Miller and colleagues[101] performed a retrospective review of 41 patients following total shoulder arthroplasty. Terminal internal rotation was evaluated by the lift-off and belly-press examinations. Abnormal results were found for 25 of 37 lift-off examinations (67.5%) and 24 of 36 belly-press examinations (66.6%). Of 25 patients with an abnormal lift-off finding, 92% reported reduced subscapularis function.

Phase IV

The patient will be progressed to this phase at approximately 16 weeks postsurgery. This phase includes work- or sport-specific training as well as suggestions for modification of work, sport, or functional activities. Patients are discouraged from participating in heavy work or recreational

activities that result in high loads and forces to the glenohumeral joint.[90] Golf, swimming, bicycling, aerobics, and running activities are acceptable activities for patients following shoulder arthroplasty. To decrease the load on the shoulder during these activities, the therapist should emphasize proper mechanics and improving overall flexibility. Patients with severe arthritis or poor soft tissues with massive rotator cuff tears typically have lower functional demands and are typically satisfied with their ability to perform normal daily activities.

MULTIDIRECTIONAL AND VOLUNTARY INSTABILITY

Multidirectional instability is defined as dislocation or subluxation in a combination of anterior, posterior, or inferior directions. The basic pathology is congenital or acquired patulous capsuloligamentous complex that lacks the stabilizing barrier and compression effect. The degree of instability may vary from those being able to participate in athletic events to those unable to lift the arm without subluxing. Typically, the labrum is intact but fraying or tearing may occur with repetitive instability events.[102] Often individuals with multidirectional instability are athletes such as swimmers and gymnasts who commonly have generalized hyperelasticity. Patients may describe diffuse achy pain with activity or at rest. Instability can be sensed with normal daily or overhead high-demand activities and weakness or paresthesias may be reported. Atraumatic posterior instability may be most notable.

An interesting patient group labeled as having multidirectional instability are those who habitually or voluntarily sublux. This includes patients who can sublux at will but are asymptomatic and do not require treatment. However, the symptomatic group may sublux/dislocate for secondary gain or psychiatric reasons but some appear to have developed an unconscious coordinated muscle firing sequence resulting in instability. EMG and biofeedback studies have identified several different abnormal firing patterns involving a combination of increased activation of the anterior deltoid and pectoralis major in conjunction with decreased activation of the posterior rotator cuff and serratus anterior.[103–105] Significant medial scapular winging can be associated with voluntary and involuntary instability (usually posterior). Although abnormal muscle activation patterns can result in posterior instability, we also believe that immediate posterior subluxation during elevation causes serratus anterior "shutdown."

Examination

Instability testing typically reveals increased translation in all directions and an excessive sulcus sign. Posture and scapular position are noted. A depressed and downwardly rotated scapula may predispose the joint to instability.[106] Inadequate scapula muscle integration is identified by poor scapular stabilization during active range motion or resisted motion testing in multiple positions. Patients are stratified into those with and without scapular winging. Patients with scapular winging usually are considered to have abnormal firing patterns, symptomatic voluntary instability, or significant compromise of the CLC (i.e., patulous CLC, large SLAP lesions). If patients present with scapular winging during sagittal plane elevation, they are asked to repeat elevation with the arm maintained in external rotation. Elimination of scapular winging confirms posterior instability. This is called an external rotation stabilization maneuver (Fig. 40-3). Our belief is that many patients immediately posteriorly sublux, causing the serratus anterior

A B

Figure 40-3 **(A)** Posterior subluxation and scapular winging present during elevation. **(B)** Scapular external rotation stabilization maneuver reduces the joint and eliminates the winging.

to reduce firing. We have measured multidirectional instability patients who demonstrate significant scapular winging using three- dimensional telemetry and electromyography and found limited scapular upward rotation and posterior tilt in conjunction with dramatic reduced firing of the serratus anterior (16% maximal voluntary contraction). Our studies have shown scapular kinematics and serratus anterior activation to normalize when the patient elevates while maintaining external rotation. Some patients, usually those with voluntary instability or those with an unconscious abnormal firing pattern, require serratus anterior preactivation to reduce the joint and prevent scapular winging. The patient is asked to protract and slightly elevate the scapula with the arms at the side followed by shoulder elevation in the sagittal plane while maintaining shoulder external rotation (Fig. 40-4). The patient must reach slightly forward to suppress attempts to downwardly rotate the scapula. The ability to dramatically reduce scapular winging and improve elevation or achieve full elevation identifies the posterior instability component and abnormal muscle recruitment patterns. If winging persists, the examination algorithm presented in the Scapular Muscle Dysfunction section is followed.

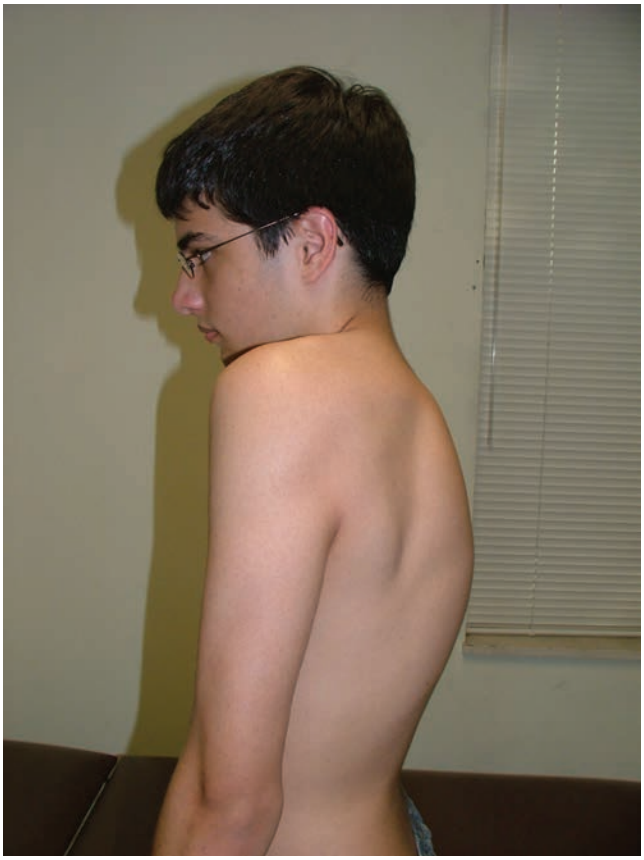

Figure 40-4 Preactivating the serratus anterior by protracting and slightly elevating the scapula while keeping the arms at the side.

Intervention

All patients demonstrating multidirectional instability without scapular winging are initially treated with glenohumeral stabilizer strengthening exercises in nonprovocative positions with scapular muscle integration.[107,108] Special attention is directed toward activating scapular muscles in isolation and then in combination with arm motion. Strengthening must be pain free and the patient's shoulder is palpated to identify subluxation while exercising. Manual resistance is applied since immediate feedback is gained for position or resistance accommodation. Initially, isometrics are utilized in varying planes and degrees of elevation. If stable and pain free, the patient is progressed to short arc motions and eventually full arc motions. Elastic bands or free-weight exercises are progressed from nonprovocative to provocative elevated positions. Functional strengthening and proprioceptive training can be achieved manually with the Bodyblade (Hymanson Inc., Playa Del Ray, CA) and Plyoball as discussed in Chapter 39. Depending on the degree of instability and the patient's demands, a limited or complete upper-extremity strengthening program is initiated. Closed-chain exercises may be utilized in certain athletic populations such as gymnasts or wrestlers. Patients who respond to a conservative approach but are felt to be high risk for future instability events are strongly discouraged from performing bench pressing, flys, overhead presses, or pushups.

Patients with scapular winging and a positive external rotation stabilization maneuver (including those requiring serratus anterior preactivation) are treated with a specific program. First poor resting scapular positioning is addressed through postural retraining and scapular retractor muscle activation. The essential component of intervention with this group is making the patient aware of repetitive subluxations. The vicious cycle of instability, pain, and muscle deactivation resulting in more instability must be broken. We show patients their abnormal movement pattern when elevating either in the mirror or by video. They are then instructed in an accentuated corrected movement pattern by preactivating the serratus anterior (protract and slightly elevate the scapula) and elevating in external rotation. The change in symptoms and range is dramatic and has a significant visual feedback effect if played back on video. The patient is instructed that during daily activities requiring forward reaching, they are to do so by using this accentuated movement pattern. This reduces the number of subluxations, breaking the vicious cycle, and it "uninstalls" the abnormal pattern and "installs" an appropriate pattern. Biofeedback may also be used for muscle retraining.[104,105] Along with postural exercise, the only other initial exercise the patient may receive is to isolate the serratus anterior by protracting and slightly elevating the scapula (arms at side). They are to do this 10 to 20 times 10 times a day. When symptoms and subluxation events reduce, patients are asked to do the

same but lift the arm to 90 degrees in the sagittal plane while maintaining external rotation. If they sublux or the scapula wings, they are not ready to progress. Typically by 1 to 2 weeks, patients have integrated appropriate muscle activation so that elevation occurs with a normal movement pattern, the glenohumeral joint remains stable, and the scapula is fixed on the thoracic wall—they have installed the new program.

Once the symptoms are controlled, subluxation events are significantly reduced, and the movement pattern looks normal, the patient is started on glenohumeral strengthening with scapular muscle integration, already discussed previously. Some patients may require further unconventional strengthening by having them hold a stick with the elbows bent at 90 degrees and in supination. They perform an isometric for the external rotators by attempting to pull the stick apart and elevate the arms in the sagittal plane while maintaining the isometric pull.

Since some patients function with asymptomatic instability, success is returning them to this status. Returning a patient back to a high level of functioning can be difficult.[109] If stability is improved but cannot be fully regained for low-demand activities, surgery is considered. Patients are required to go through a conservative rehabilitation program, especially those with scapular winging. We have found the response to the movement retraining helpful in surgery selection and improved postsurgical outcomes.

Multidirectional instability is a challenge to both the therapist and surgeon. Recognition of the pathology and using a symptom-dependent exercise progression program can return patients back to symptom-free activities. Patients with scapular winging and abnormal firing patterns require special attention since most will fail if the subluxation frequency and abnormal firing patterns are not addressed.

SCAPULAR WINGING AND DYSKINESIA

Scapular dyskinesia is an alteration in the normal position or motion of the scapula during coupled scapulohumeral movements.[110] Scapular winging may be considered a type of scapular dyskinesia characterized by significant scapular medial border displacement during shoulder motion. Quantitative scapular kinematic studies have shown abnormal scapular motion associated with certain pathologies.[111–113] Unfortunately, the equipment used to attain these measurements is not clinically applicable. The clinician is left with visual inspection and linear measurements that have low reliability.[114] Insight regarding the origin, examination, and intervention of scapular dyskinesia continues to develop.

Kuhn[115,116] described a classification system for scapular winging: primary, secondary and voluntary. Primary winging is related to neurologic, osseous, and soft tissue pathology. The two most common reasons for neurologic scapular winging are long thoracic nerve, affecting the serratus anterior, and spinal accessory nerve palsy, affecting the trapezius. Kuhn discussed a rarely seen winging due to rhomboid weakness created by a dorsal scapular nerve palsy or C-5 radiculopathy. Mixed palsies due to brachial plexopathy can also occur. Osseous malformations such as osteochondromas, clavicular malunions, or scoliosis cause scapular winging. Soft tissue abnormalities such as muscle ruptures, congenital absence anomalies, or scapular bursitis can result in scapular winging. Secondary winging is related to pathology at the glenohumeral joint or related tissues and the acromioclavicular joint. Glenohumeral joint instability, typically posterior, is a common cause of scapular winging. Scapular winging or dyskinesia is often related to poor scapular motor control in an intact neural system. This is referred to as motor control dyskinesia, and although it is seen in conjunction with other shoulder pathology, it is commonly encountered among asymptomatic individuals. The third category causing scapular winging is voluntary, nonpathologic and pathologic. Patients without true pathology but who coordinate muscle activity to cause scapular winging represent the nonpathologic group, while those with true pathology (i.e., instability and symptomatic voluntarily wing) are categorized as pathologic.

Examination

Kelley described an examination algorithm for scapular muscle assessment to determine if winging was due to neurologic, secondary causes and motor control dyskinesia.[117] The patient is first observed standing for resting winging and obvious atrophy. If resting winging is noted, the patient is checked for a scoliosis demonstrated by a thoracic rib hump during trunk flexion. Active range of motion of both shoulders is assessed in the standing position by elevating in the sagittal plane. Significant scapular winging that normalizes beyond 90 degrees during sagittal plane flexion elevation is related to motor control dyskinesia (typically the serratus anterior). If medial winging persists beyond 90 degrees, a long thoracic nerve palsy or posterior glenohumeral instability is suspected. Posterior instability causes scapular winging for two reasons: reflexive serratus anterior "shut down" resulting from immediate posterior subluxation or abnormal shoulder girdle muscle activation. The patient is then asked to elevate the arm in the sagittal plane while the arm is maintained in full external rotation to differentiate between instability or a long thoracic nerve palsy. This is referred to as the external rotation stabilizing maneuver (Fig. 40-3). Sometimes preactivating the serratus anterior by scapular protraction and slight elevation is required, especially in those patients who habitually sublux or have developed an unconscious abnormal firing pattern (discussed in the Multidirectional and Voluntary Instability section). Elimination of scapular winging with the external rotation stabilizing maneuver identifies

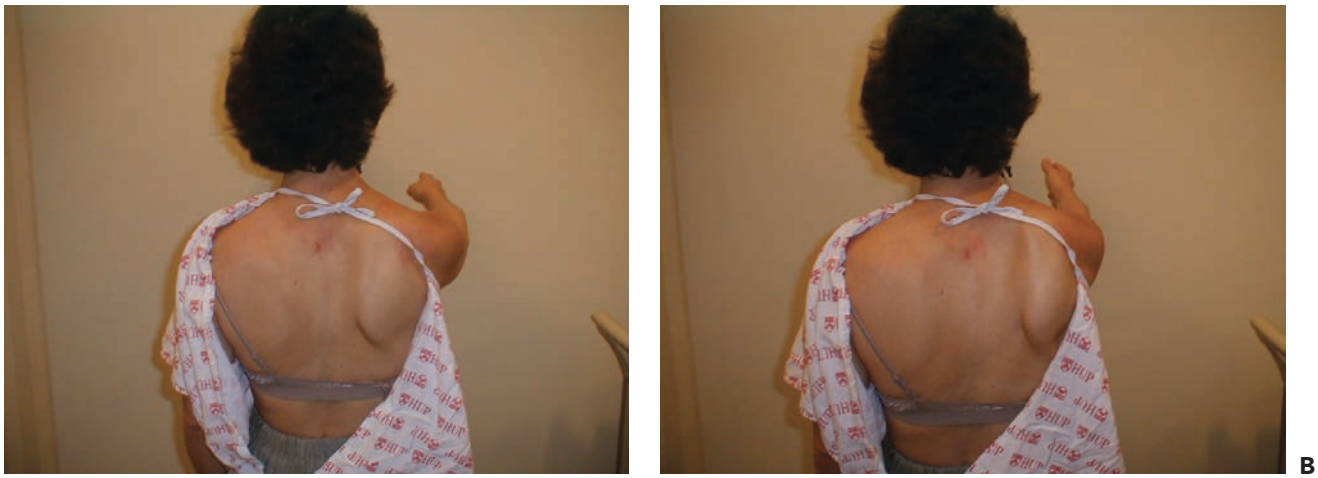

Figure 40-5 **(A)** Plus sign is performed by having the patient elevate to 90 degrees and then reach forward. **(B)** The scapula should protract along the thoracic wall but if winging increases, it is a sign of complete paralysis.

the underlying pathology as posterior instability. Maintaining humeral external rotation during elevation tightens the capsuloligamentous complex in addition to contracting the external rotators, both of which prevent posterior subluxation and allow normal serratus anterior activation. Further instability special tests can be performed to confirm instability.

If humeral external rotation does not eliminate scapular winging, the patient is asked to perform scapular protraction and slight scapular elevation while the arm is at 90 degrees of sagittal plane flexion. This is called a "plus" sign. The lower trapezius commonly attempts to stabilize the medial border during forward flexion in the presence of a long thoracic nerve palsy. When protraction at 90 degrees is performed, the lower trapezius reflexively deactivates due to antagonistic inhibition and the serratus anterior is left to control scapular movement. If scapular winging increases during attempted protraction, the patient is considered to have absent serratus anterior activity. Inability to protract the scapula on the thoracic wall is referred to as a positive "plus" sign (Fig. 40-5). If the patient protracts and the scapula fully moves forward on the thoracic wall or the movement is incomplete, motor dyskinesia or partial serratus anterior activity due to a recovering palsy is suspected, respectively. A recovering long thoracic nerve palsy cannot be ruled out if complete scapular protraction is achieved since enough fibers may be innervated to complete the task. Next the patient is asked to place the arm at 135 degrees of sagittal plane elevation and the examiner resists, pushing into shoulder extension while palpating the scapular inferior border.[118] Easy posterior displacement of the inferior border with minimal resistance is a sign of significant serratus anterior weakness and considered to be a resolving neurologic insult. Patients with very dramatic scapular dyskinesia but an intact neurologic system and void of glenohumeral subluxation will easily maintain the scapula fixed on the thoracic wall during resisted shoulder flexion at 135 degrees.

Identification of a spinal accessory nerve injury is assisted by the presence of trapezius atrophy and a depressed and protracted shoulder girdle. Coronal plane abduction is then performed and scapular movement noted. If good sagittal plane elevation is present but the patient cannot lift above 90 degrees in the true coronal plane (without pain), a spinal accessory nerve palsy is suspected. The trapezius is further examined by performing the "flip" sign.[119,120] The "flip" sign is performed with the examiner standing to the patient's side and resisting the involved shoulder's external rotators. The examiner visually examines the scapula to determine if the scapular medial border "flips" from the thoracic wall.[120] A positive "flip" sign occurs when significant displacement of the medial border occurs off the thoracic wall. The mechanism for this sign is unopposed pull of the infraspinatus and posterior deltoid by the middle and lower trapezius. Interestingly, the rhomboid muscle does not spontaneously activate to stabilize the scapula in this position. Further isolation to determine manual muscle testing strength grade of the middle and lower trapezius can be performed with the patient prone as described by Kendall and McCreary.[118]

Intervention

Rehabilitation will be discussed for patients with neurologic involvement, glenohumeral instability, and motor control dyskinesia.

Neurologic Involvement

Rehabilitation of the individual with either a long thoracic or spinal accessory nerve palsy begins with understanding

pathology. There is nothing we can do to facilitate reinnervation other than creating a friendly environment for nerve regeneration. Postural education and supporting the arm are important to eliminate the dependent weight of the extremity, minimize scapular depression, avoid separation of headed neck, and reduce pain. The most important aspect of rehabilitation in this patient population is improving scapular muscle compensatory activity. Individuals with serratus anterior paralysis are encouraged to activate the rhomboid and trapezius to gain greater stabilization and upper extremity "power." The patient is instructed in exercise to isolate and integrate the rhomboid and middle and lower trapezius by retracting and/or elevating the scapula. Phase I strengthening exercises (external rotation, internal rotation, and extension) are performed by first preactivating the trapezius and rhomboid. Resisted elbow flexion and extension exercises are performed with scapular muscle integration. Teaching scapular muscle preactivation encourages scapular stability during distal functional movement patterns. Diagonal patterns simulating a "backhand" are performed emphasizing scapular retraction. Manual therapy is performed to isolate and challenge the retractors. As the nerve recovers and serratus anterior muscle activation is noted, the serratus may first be activated supine, performing a scapular protraction with the arm positioned at 90 degrees. Exercise is progressed to standing and then with resistance. Progression to weight-bearing exercise is attempted in a graduated manner.

A patient with a spinal accessory nerve palsy is encouraged to isolate and integrate the serratus anterior and rhomboid. The same exercises previously described are performed, in addition to forward flexion with a "plus." The progression includes diagonal patterns and gravity-minimized and antigravity positions.

Intermittent visits are encouraged when treating both long thoracic or spinal accessory nerve palsies in conjunction with a consistent home exercise program since recovery following is time dependent.

Glenohumeral Instability

Rehabilitation of patients with primary instability (usually posterior) and scapular winging is achieved by attempting to stabilize the glenohumeral joint. Commonly, these patients have a history of voluntary subluxation. Voluntary instability or patients trapped in an unconscious pattern activate/deactivate scapular, axiohumeral, and glenohumeral muscles to sublux the glenohumeral joint and create scapular winging. Patients must be made aware of their volitional involvement. Subluxation typically occurs with any attempted forward movement of the arm. The external rotation stabilizing maneuver is performed. This maneuver can prevent the subluxation, suppress abnormal muscle firing patterns, and eliminate scapular winging. The patient must understand the inflammatory nature of the vicious cycle; subluxation leads to pain and joint irritation resulting in reflexive muscle shutdown, which results in instability. The cycle must be interrupted and the frequency of subluxations reduced. Once this is achieved, typical strengthening of the glenohumeral and scapulothoracic stabilizers can be performed.

Motor Control Dyskinesia

Rehabilitation of individuals with scapular motor control dyskinesia requires a thorough examination and integration of anatomechanics and kinesiology. Some degree of motor control dyskinesia is quite common in the general population, but most remain asymptomatic. Weight lifters or those who previously performed heavy bench pressing or high-repetition pushups tend to demonstrate poor eccentric scapular muscle control. Symptomatic dyskinesia occurs in throwing athletes who demonstrate significant resting scapular depression and protraction. Burkhart et al.[121] described the SICK scapula in which the abnormal scapular resting position results in altered kinematics and ultimately rotator cuff tendinopathy and glenohumeral instability. Pectoralis minor stretching helps to reestablish a normal resting position, allowing appropriate scapular posterior tilting and rotation during elevation. Isolation through manual techniques and/or active range of motion is essential to teach scapular muscle control.[121,122] Unfortunately, many therapists believe dyskinesia is related to inadequate "strength"; however, we believe it is related to poor muscle "control." Aggressive scapular muscle strengthening can increase symptoms due to provocative positioning. Once appropriate scapular muscle isolation and coordination is achieved, resistance is applied either manually, with elastic bands, or with free weights. The patient is progressed to strengthening in functional positions and return to activity/sports.

Rehabilitation of scapular winging and dyskinesia requires an algorithmic examination approach to identify the pathology and the appropriate intervention. Aggressive strengthening before motor control is established leads to symptom perpetuation and frustration. Scapular muscle isolation and eventual integration into functional movement patterns will improve outcomes.

SUMMARY

Proper rehabilitation of the shoulder is essential to the recovery of patients treated both conservatively and postoperatively. Successful rehabilitation is dependent on effective communication and interaction between the physician, therapist, and patient. Each of these team members has a defined role in the rehabilitation process and must fulfill his or her responsibilities for the desired outcome to be achieved.

This chapter presented principles and rationales for rehabilitation of various shoulder pathologies. In addition, guidelines for instruction and exercise progression were illustrated. Whether the patient will be followed in regular supervised therapy or seen in the office at specific intervals, extensive patient education is essential to successful rehabilitation. The patient must understand the pathology and rationale for each phase of the rehabilitation process. Constant reevaluation by the physician and therapist is important to make necessary program modifications if the patient is not achieving preset goals.

The common goals of the rehabilitation process include reduction of pain and inflammation, facilitation of collagen healing, improvement in ROM and strength, and optimization of proprioception and endurance. This is achieved by gradually increasing the program from nonprovocative to provocative positions. It is the rehabilitation specialist's and surgeon's responsibility to identify when to implement the appropriate modalities or exercises to improve the impairment and thereby increase the patient's function.

Prospective studies that demonstrate rehabilitation of impairments and functional limitations associated with various shoulder pathologies are needed in the literature. Once these studies exist, a dialogue between physicians and therapists can be further developed. The end result will be more efficient and successful rehabilitation for the most important member of the team—the patient.

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41 Measurement of Shoulder Outcomes

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INTRODUCTION

Documenting outcome of medical and therapeutic treatment is receiving increased emphasis from patients, administrators, and third-party payers. To survive in today's health care environment, providers must document quality of treatment, compare results of different forms of treatment, and compare results reported by other clinicians. Keller has stated that the quality of medical care is based on a combination of the "efficacy" of a procedure or technology as proven in laboratory studies, the "effectiveness" of that procedure when used in the community, and the "appropriateness" of the procedure when applied to a given patient.[81]

Patient-oriented outcomes research focuses on the effectiveness and appropriateness of a treatment. Until recently, the orthopedic literature has consisted of efficacy research. Although reports such as these may be related to the success of the procedure, they do not tell us what effect the procedure had on the patient. Because there is little evidence of patient outcome, treatment recommendations have been left to the personal experience of a limited number of surgeons who most often have a special interest or expertise in the area being reported. It cannot be assumed, then, that patients with similar pathologies from different geographic locations are being treated with the same procedure, let alone achieving the same outcome.

Although the recent trend has been toward reporting clinical assessment and patient self-reported outcome, no one system or tool has been widely used and accepted. In a systematic review encompassing 610 articles relating to surgery on the shoulder, a total of 44 different outcome

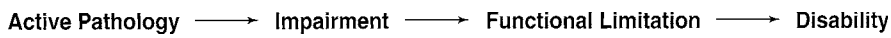

Figure 41-1 Nagi disablement scheme.

scores were encountered.[63] Of these, 22 (50%) used clinical assessment, 21 (47.7%) used a self-report tool, and 1 (2.3%) used both clinical assessment and a self-report tool.[63] These authors also reported that the overall pattern of the application of an outcome score was highly variable and at times inappropriate.[63] They identified changes made to self-report questionnaires, often without proper testing of the modification and without justification.[63]

Although Codman set the groundwork for reporting outcomes over 80 years ago, there is still much work to be done in this area. Common guidelines and terminology must be agreed upon for an outcome tool to become widely used and accepted. This chapter will present (a) a brief history of outcome reporting, (b) the concept of disablement, (c) criteria for validation of outcome measures, and (d) a review of existing shoulder scales.

DISABLEMENT

Before we discuss measurement tools, it will be helpful to classify outcomes through the use of a disablement scheme. "Disablement" is a global term that reflects all the diverse consequences that disease, injury, or congenital abnormalities may have on human functioning at many different levels.[76] Saad Nagi introduced the first disablement scheme in 1965 (Fig. 41-1).[111,112] The World Health Organization followed with the International Classification of Impairments, Disabilities, and Handicaps in 1980.[66,153] The goal of these schemes is to describe the pathway from active pathology to various consequences. For simplification, we will only discuss the Nagi scheme.

Nagi described active pathology as the interruption or interference of normal processes and the efforts of the organism to regain a normal state.[111,112] Active pathology can result from degenerative disease processes, infection, trauma, or other causes. Attempts have been made to accurately describe pathologies, such as classification of fractures, size of rotator cuff tear, shape and size of acromion, and degree of instability. Follow-up typically consists of x-rays to document fracture healing, arthroplasty position, or acromial shape, and magnetic resonance imaging for size of rotator cuff tear or degree of healing of rotator cuff repair. Again, this tells us about the efficacy of the treatment, but nothing about its effect on the patient.

Active pathology typically leads to an impairment of some kind. Impairment is an anatomic or physiologic abnormality or loss that can be a direct result (e.g., decreased active range of motion [ROM] due to a frozen shoulder) or secondary result (e.g., decreased strength secondary to disuse) of the pathology. Much of the clinical orthopedic literature has focused on impairment data such as pain, ROM, strength, and stability as an outcome.

Pain is one of the most commonly measured impairments in shoulder literature. Pain has been measured and reported in various ways, including visual analog scales (VASs), numeric rating scales, descriptive scales, and the amount and type of pain medication. The visual analog scale has been reported to be reliable and valid.[125,133] The typical VAS consists of a 10-cm horizontal or vertical line with anchor points such as "no pain" and "worst possible pain" at either end of the line. The patient is asked to place a mark on the line indicating the amount of pain he or she is experiencing. Pain is then calculated by measuring the distance of the mark from one of the anchor points. A disadvantage of measuring pain in this way is that it can be very time consuming for the clinician, because every scale must be individually measured. It should also be noted that the 10-cm line will decrease with each photocopy, so printed forms should be used.[68]

Another method used to record pain is a numeric rating scale. Much like the VAS, a numeric rating scale also uses anchor points. However, rather than using a line, numbers are typically arranged from 0 to 10. The patient is asked to circle the number that best correlates to his or her level of pain. Ferraz et al. found the numeric rating scale to be the most reliable among both literate and illiterate subjects.[48] Good correlation of numeric rating scales to VASs has been reported.[152] Williams et al. also favored numeric rating scales for telephone administration and longer-term follow-up.[152]

A descriptive pain scale typically provides the patient with verbal descriptors such as none, mild, moderate, and severe.[33,34,68] "Moderate" is most often chosen, even in patients who rate pain as mild or severe by other methods.[68] Another concern is that there are not enough options for patients to accurately record pain in the same rank order.[107]

Quantifying pain by the amount and type of pain medication, such as over-the-counter, nonsteroidal antiinflammatories, or narcotics, also has been used.[45,121] Although this is useful information to have on hand, the accuracy of this type of pain scale must be questioned, because various psychological and psychosocial issues may taint the results. Pain scales that include descriptors as well as amount of medication in a single item should be regarded with caution, because multiple variables can be confusing for patients.[45]

Shoulder ROM is an important outcome of shoulder surgery. Goniometric measurement of ROM has been reported to be reliable.[22,50,107,122] However, because of the multiplanar motion of the shoulder, it can be difficult to reproduce and compare results reported by other clini-

cians.[122] In response to the need to standardize measurements, the American Shoulder and Elbow Surgeons (ASES) recommends that four functionally important ranges of motion be documented: forward elevation, external rotation with the arm at the side, external rotation in the 90-degree abducted position, and internal rotation.[25,121] Forward elevation is defined as the maximum angle the arm makes with the trunk when the patient is asked to raise his or her arm above the head with the elbow held straight. It should be noted that this plane of motion is not considered to be true flexion or abduction. Experience tells us that most patients will raise their arm somewhere between true flexion and the plane of the scapula. The angle that the arm makes with the thorax is measured in the upright position for active motion, whereas passive measurement is made in the supine position.

External rotation with the arm at the side is measured with the elbow flexed to 90 degrees and the forearm in the sagittal plane. External rotation at 90 degrees of abduction is measured by asking the patient to begin with the arm in the same position as for external rotation at the side. The patient is then asked to abduct the arm to 90 degrees. Once at 90 degrees of abduction, the patient is asked to externally rotate the shoulder. Active motion is measured with the patient upright, whereas passive motion is measured with the patient supine. Internal rotation is measured by the position reached by the outstretched hitchhiking thumb up the back, in reference to the posterior anatomy. Common reference points include the greater trochanter, buttock, sacrum, waist line, and spinous processes of the lumbar and thoracic vertebrae.

Strength assessment is another commonly reported impairment to document effectiveness of surgical or therapeutic intervention. As with ROM testing, it is important to have standardized testing protocols for reproducibility in a clinical setting. A variety of methods for quantifying muscle performance have been available, including manual muscle testing (MMT), handheld isometric dynamometry, and isokinetic dynamometry.

MMT is the most widely used method of clinical evaluation of muscle strength.[102] Some investigators have recommended using MMT when documenting shoulder outcomes.[45,121] This technique, however, has been criticized for its subjectivity and lack of reliability within the good and normal ranges.[7,23]

In an attempt to quantify the results of MMT procedures, handheld dynamometry was introduced in 1949.[102] Although several investigators have demonstrated handheld dynamometry to be reliable in both patient and nonpatient populations, problems include an upper limit to recording muscle force, difficulty in maintaining the device perpendicular to the limb segment, and dependency on the strength of the tester.[1,14–19,27,30,43,89,98,102,115,120,143,151]

Several isokinetic devices have become available for use in the clinic, including those that have the capability of measuring concentric, eccentric, and isometric strength. The reliability of these dynamometers has been well documented.[28,35,44,47,71,86,89,97,117,134] However, these devices are nonportable and relatively expensive, and require elaborate setup and stabilization procedures. Therefore, use in the clinic is time consuming and impractical.

The Isobex 2.1 (Curor Ag, Niederwanten, Switzerland) is an isometric dynamometer that is lightweight and portable. Fixation to a wall is attained by suction cups. A cable mechanically measures force output. Reliability of this device for measuring shoulder strength is comparable with or better than isokinetic and handheld dynamometry.[89]

To accurately compare results of strength measurements, standardization of such variables as test position, dynamometer, stabilization, and protocol must be established. Several studies have advocated testing shoulder strength in the plane of the scapula and 15 to 45 degrees of abduction. Proponents of this test position argue that it avoids the impingement arc, avoids the apprehension position, and reduces passive tension on the rotator cuff.[132] However, positioning the arm in the scapular plane at 45 degrees for rotational testing would require appropriate stabilization of the limb, which may make reproducibility and efficiency in the clinic difficult. Testing elevation at 45 degrees in the plane of the scapula would not require stabilization. A recent study by the authors demonstrated that measuring isometric shoulder strength for internal and external rotation with the arm at the side in neutral rotation is highly reliable. In addition, elevation tested at 45 degrees in the plane of the scapula is also reliable.[89]

Interpreting strength testing data has typically consisted of comparison with the uninvolved extremity and comparison with normative data.[132] It is difficult to compare results with the normative data available in the literature because test position and dynamometer vary for each study. Although we prefer comparing ROM and strength data to the opposite uninvolved side, this method is not without controversy. The influence of hand dominance in both athletic and nonathletic populations has been debated in the literature.[28,117,132] In patients who have bilateral shoulder involvement, age-, gender-, and hand dominance–matched normative values would be ideal.

Instability is also an impairment commonly reported. The ASES recommends grading the amount of instability of the glenohumeral joint in the following way: 0 = no translation, 1 = mild (0 to 1 cm translation), 2 = moderate (1 to 2 cm translation or translates to the glenoid rim), and 3 = severe (greater than 2 cm translation or over the glenoid rim).[141] This should be noted for anterior, posterior, and inferior translation. In addition, the presence of apprehension or a positive relocation test should be noted. The ASES also recommends having the patient rate his or her level of shoulder instability on a VAS.[121]

Although knowledge of these parameters is important to the overall understanding of pathology, they do not describe

the total effect of the pathology on the person. Nagi used the term "functional limitations" to describe limitations in performance at the level of the whole person. Physical functioning involves the patient's ability to perform activities of daily living (ADLs), dress, reach, push, pull, lift, carry, and work. However, all impairments do not lead to functional limitations. For example, a patient with limited shoulder ROM may not be able to reach the back of his or her opposite shoulder. However, this may not be a functional loss, because the patient may use a long-handled scrub brush to wash the axilla and back of the opposite shoulder.

Disability is the limitation in performance of socially defined roles and tasks within a sociocultural and physical environment.[76] A patient who is unable to perform or participate in usual work, school, leisure, and personal care activities can be considered disabled. It is important to note that not all impairments and functional limitations lead to disability. Disability largely depends on the patient's desired activity level. For example, a limitation in the ability to raise the arm above shoulder level may not be a disability for a computer programmer who works below shoulder level. However, this functional limitation would be a devastating disability for a painter or professional tennis player. We also can illustrate disability through the use of shoulder scoring systems. The computer programmer with a shoulder score of 90 on a 100-point scale can have no perceived disability. However, moderate disability can be perceived by the painter or tennis player with the same 90-point score.[34,69]

The disablement process can be halted at each step by appropriate surgical or therapeutic intervention. In addition, the process can be expedited or slowed by several factors, including age, gender, lifestyle, education, social support, motivation, and environment.[75] It is important to determine which of these factors weigh most heavily on patient outcome after shoulder surgery. Self-report questionnaires are now a common way to determine patient outcome after surgical or therapeutic intervention.

DEVELOPMENT OF SCORES

Several instruments have been reported to document outcome with little regard for the process of selecting and constructing the scale. This has caused confusion as to the best way to construct and validate outcome measurement tools for use in the clinic.[84] Clinicians should choose an outcome tool that will best suit the purpose of its use.[84] Kirshner and Guyatt classified outcome assessment tools as either discriminative, predictive, or evaluative.[84] A discriminative index is used when no "gold standard" is available to validate the measure. Items in this type of measure must be important to patients and performed by almost all of the patients to be studied. The most appropriate number of response options in this type of scale is two. Either the patient can or cannot perform the activity. This type of instrument allows comparison among different groups. However, a dichotomous scale such as this cannot detect small changes in a patient's status over time.

A predictive index is used to determine whether a patient has a specific condition or is likely to develop that condition. The patient can then be classified into a specific category. Based on a comparison of other patients in that category, one can predict the outcome the patient will achieve. However, this type of index will not measure outcome.

An evaluative index is used to quantify the amount of change the treatment intervention has provided. An evaluative index must be able to measure all clinically important effects of treatment.[84] These scales must be responsive to change over time. It is this type of scale that we are most interested in to document within-person change over time.

Several investigators have outlined requirements for the development of new outcome measures.[21,56,58,61,74,77,78,81,84,142] For an index to be considered useful in the clinic, there are several steps that must be followed, including item selection, item scaling, item reduction, determination of reliability, determination of validity, and determination of responsiveness.[126]

Item Selection

The first step in constructing an evaluative instrument is to select a set of items that are relevant to the overall function of the type of patient to be evaluated. This can be accomplished through literature review, personal experience, consultation with colleagues, review of existing instruments, and interview with patients.[58,61,84,137] The likelihood that patient response to a particular item will change with medical or therapeutic intervention is an important consideration.[84] In addition, all clinically important treatment effects must be included in the tool.[84]

Item Scaling

Once the items of the instrument are established, the response options or range that patients have for responding to each item must be determined. Each item must be sensitive to clinically important changes in status. Typical response options include a VAS, NRS, or a Likert scale with multiple options. The optimal number of response options for a Likert scale has not been reported. However, increasing response options will increase item responsiveness. The typical Likert scale includes five to nine options. For a tool designed to discriminate between patients who can or cannot perform an activity, a simple "yes" or "no" is all that is necessary. These response options are fine for tools designed to detect between-person differences, but not small changes in an individual patient's status. Therefore, it is recommended that either a Likert scale, an NRS, or a VAS be used with an evaluative tool.

Item Reduction

Because we are interested in measuring change over time, it would be counterproductive to include items in the scale that do not change after a particular intervention. This stage of scale development is item reduction. A method of reducing the items of a scale is to administer the scale to a group of patients before and after an intervention that produces change in a patient's status. Items that are unresponsive to change are then deleted from the questionnaire.

Reliability

Once the questionnaire is in its final format, it is ready for reliability testing. Reliability is the extent to which a measurement is consistent and free from error.[58,61,84,119,129,142] Reliability is tested by administering the questionnaire to the same subject at two points in time. Although there may be small changes in within-person measures, they may be statistically and clinically insignificant.[126] Intrarater reliability refers to the stability of data recorded by one individual across two or more trials.[119,129] Interrater reliability is variation between two or more raters who measure the same group of subjects.[119,129] Test–retest reliability means that a measure will remain stable over repeated measures in time, provided that no change in status has taken place.[129] An intraclass correlation coefficient (ICC) or kappa coefficient statistic is commonly used to express reliability.[119] The extent to which the items of a scale reflect the same dimension is known as internal consistency.[49] Internal consistency also provides an index of a scale's ability to differentiate among clients at an instant in time.[49] A coefficient alpha is typically used to determine internal consistency of a self-report tool.[38]

Error and Change

The error associated with a single application of any clinical or functional measurement tool can be analyzed with the standard error of measurement (SEM). The SEM provides an estimate of how reliably a scale estimates an individual's "true score," that is, the score that would be obtained for the person if the scale measured perfectly, without error.[42,105,126] The SEM is a representation of measurement error expressed in the same units as the original measurement.[13] The SEM can be calculated by using either Cronbach's α or the intraclass correlation coefficient.[49] Whereas error estimates based on Cronbach's α are specific to an instant in time, error estimates derived from test–retest reliability can be generalized over a time interval equal to that applied in the test–retest reliability study.[49] The SEM can be calculated with the formula: SEM = SD × [square root (1 – reliability coefficient)]. The SEM carries with it 68% confidence bounds. To achieve a higher confidence level, the SEM can be multiplied by the z value associated with the 90% confidence level (z = 1.65) or the 95% confidence level (z = 1.96).

The error associated with multiple applications of a clinical or functional measurement tool can be determined by calculating the minimal detectable change (MDC). The MDC can be used to make clinical judgments regarding whether a patient has improved or not. It can be calculated with the formula: MDC = SD × [square root (1 – reliability coefficient)] × square root of 2. The MDC also carries with it 68% confidence bounds. To achieve a higher confidence level, the MDC can also be multiplied by the z value associated with the 90% confidence level (z = 1.65) or the 95% confidence level (z = 1.96).

The minimal clinically important difference (MCID) is the smallest difference in a score that is considered to be worthwhile or important.[64] The approach used to establish the MCID is not fixed.[8] Nine different methods have been used to calculate the MCID for an instrument.[8,150] A commonly used approach is for the patient to rate his or her change on a global scale (much worse to no change to much better).[8,72] The mean change of those who report becoming slightly worse or slightly better is used to calculate the MCID.

Validity

Validity concerns the extent to which an instrument measures what it is intended to measure.[119] Validating a new instrument is an ongoing process as new information becomes available.[49] In a shoulder score, we want to know if it can evaluate change in the magnitude or quality of a variable from one time to another. There are several types of measurement validity including face, content, construct, and criterion.[49]

Face validity is the simplest and weakest form of validity. If the items of an index appear to make sense to the person using it, then the scale has face validity. Content validity is satisfied when it is proven that the scale measures all important aspects of the condition to be examined. A more formal way of measuring validity of an index is to demonstrate that the results of the new scale correlate to an external "gold standard." This is also known as criterion validity.

Construct validity refers to how well one instrument performs when compared with instruments of similar or dissimilar purposes and dimensions.[108] Convergent construct validity examines the correlations among similar instruments.[108] Divergent construct validity is assessed by examining the correlations between dissimilar instruments.[108] Correlation coefficients are calculated for each of these analyses and can range from −1 to 1, with 1 indicating a positive relationship and −1 a negative relationship.[108] A high positive correlation coefficient is desired when the two scales being examined are, in theory, similar in nature. A high negative relationship is expected when assessing divergent validity.

Before we can declare an evaluative index useful in the clinic, it must be able to detect improvement, regression, or stability of a condition over time.[140] "Sensitivity to change," "responsiveness," or "longitudinal validity" are terms that have been used when examining a measure's ability to detect change over time.[2,41,49,96,140] We will use the term "responsiveness" when discussing a measure's ability to detect change. There are several methods for assessing responsiveness.[84] Stratford et al. have discussed several study designs that can be used to determine responsiveness of an index.[140] The simplest design compares an initial score with a follow-up score after treatment intervention. More sophisticated designs compare the outcome of a group who received a treatment of known efficacy with that of a placebo group. Another design involves comparing the clinician's and patient's global ratings of change with the change in score. Whichever design is chosen, an adequate sample size must be selected.

There are many methods of assessing responsiveness. The two most commonly employed methods are the standardized response mean (SRM) and the effect size (ES).[80,90] The SRM is calculated with the formula: SRM = mean change (follow-up scores – initial scores) ÷ SD of change scores. The ES is calculated with the formula: ES = mean change (follow-up scores – initial scores) ÷ SD of initial scores. Interpretation of the SRM and ES values is not standardized.[108] Guyatt et al.[59,61] suggested that an SRM greater than 0.80 demonstrates an acceptable level of responsiveness. Cohen[31] recommended that an ES or SRM of 0.2 represents a small effect, 0.5 a moderate effect, and 0.8 or more a large effect. The greater the "effect" is, the more responsive the measure is and therefore the more likely it is to reflect actual change in a patient's function and disability.[108]

CRITICAL REVIEW OF SHOULDER SCORES

Several tools have been developed and used to document outcome of surgical and therapeutic treatment of shoulder pathology. These include generic quality-of-life or health status measures, region-specific measures, condition-specific tools, and joint-specific tools.[12,29,87] Although many of these tools have been used in various reports, no one tool has been widely accepted and utilized.[55,127] The lack of universal acceptance could be attributed to several factors including the wording of questions, specific content of the scale, allocation of point values, and lack of or inadequate measurement properties.[24]

Shoulder self-report tools should measure components of pain, patient satisfaction, and function.[55] Many tools contain some of these components but do not adequately assess each dimension. For this reason, some authors have advocated the use of a generic health measure, a shoulder-specific measure of function, and a measure of patient satisfaction.[11,52,85] We will discuss the advantages and disadvantages of each type of measure used to assess outcome after shoulder surgery or intervention.

Generic Measures

Generic measures quantify a patient's perception of his or her overall state.[12] The object of using generic measures is to quantify overall health rather than that related to a specific condition.[12] Therefore, they would cover health issues such as heart disease, diabetes, or other comorbid conditions and the shoulder problem.[12] Although generic measures tend to be less sensitive to changes in orthopaedic disorders, they provide a valuable broad view of the patient's overall health.[11,12,20,60,118,123]

The most commonly used generic health measure in the orthopaedic literature is the SF-36.[12,106,144,145,148] This index consists of 36 items derived by Ware and colleagues from the Medical Outcomes Survey.[141,144,145,148,149] It measures eight health concepts: physical functioning (10 items), social functioning (two items), role limitations due to physical problems (four items), role limitations due to emotional problems (three items), mental health (five items), energy/fatigue (four items), bodily pain (two items), and general health perception (five items).

Gartsman and colleagues[52] found that the SF-36 documented significant differences between U.S. general population norms and patients with five common shoulder conditions (glenohumeral instability, rotator cuff tear, adhesive capsulitis, glenohumeral arthritis, and impingement). In the great majority of comparisons in this study, patients with shoulder conditions scored significantly lower than the U.S. general population norms. In addition, they reported that these five common shoulder conditions had an effect on an individual's perception of his or her general health that ranks with those of several major medical conditions (hypertension, congestive heart failure, diabetes mellitus, myocardial infarction, and clinical depression).[52]

However, the SF-36 may not be sensitive enough to document change over time in patients with shoulder pathology. Mossberg and McFarland[110] found lower physical functioning scores in patients with lower-quarter involvement than in those with upper-quarter involvement. They were not surprised by this finding, given that nearly all items in the physical functioning subscale are in some way associated with the use of the lower extremities. Beaton and Richards[11] found lower correlations of five shoulder-specific questionnaires with the SF-36 than with each other. They also found the scores on the physical function section of the SF-36 to be higher than the scores on the shoulder-specific measures in the same patients. Dawson et al.[39] and Angst et al.[3] found similar results in their studies on patients following rotator cuff repair and total shoulder arthroplasty.

Although the authors of the SF-36 considered it a "short-form" measure, some consider a questionnaire with 36 questions too lengthy.[51] Therefore, the SF-12 Health Survey was developed to provide a shorter alternative to the SF-36.[146,147] The SF-12 contains a subset of 12 items from the SF-36 including one or two items from each of the eight SF-36 scales. The SF-12 items and summary scores have reproduced the SF-36 summary measures with the same interpretations.[51,146,147] To date, a comparison of these two measures in patients with shoulder disorders has not been performed.

Disease-Specific Measures

To detect very focused types of changes in patients with shoulder disorders, some investigators have advocated the use of disease-specific assessment tools.[82,83,93,130] Rowe et al. were one of the first to attempt to accomplish this with their Rating Sheet for Bankart Repair.[130] This 100-point scoring system awards 50 points to the patient who reports no recurrence of dislocation, subluxation, or apprehension. The remaining 50 points are derived from ROM and function. There have not been any reliability or validity studies on this scoring system. Although this scoring system appears well suited for the patient with shoulder instability, it would not be useful for a patient who has a comorbid shoulder disorder. Using disease-specific tools makes it difficult to compare the functional limitations and disability associated with different shoulder conditions.

Region-Specific Measures

Region-specific measures are applicable across many joints or disorders and usually are designed for the entire upper limb.[12] They offer a practical alternative to disease-specific or joint-specific tools in that they can be used in the context of all the various disorders encountered in a busy practice.[12] However, a tool must have adequate measurement properties in all the patient groups in which it is applied.

The Disabilities of the Arm, Shoulder, and Hand (DASH) outcome measure is a commonly used tool for the upper extremity.[9,104] The DASH is a 30-item questionnaire that evaluates symptoms and physical function with a five-response option for each item.[9] A scoring algorithm allows a score out of a possible 100 points. A higher score on the DASH reflects greater disability. The measurement properties of the DASH have been examined (Table 41-1).[9,67,99,104]

Joint-Specific Measures

A number of tools that are applicable to any condition of the shoulder are available. Although they have been used extensively in the literature to report outcome of surgical or therapeutic intervention, evidence of psychometric properties is lacking and many times the scales are modified by the authors. The most commonly used scales will be discussed regarding content and measurement properties.

Neer Rating Sheet

Neer published the first system to document outcome in 1972, when he discussed his results of anterior acromioplasty.[113] In 1982, he discussed results of total shoulder replacement, using the same system.[114] Although the Neer rating sheet is not a cumulative scoring system, it does take into account the important aspects of pain, motion, strength, function, and patient satisfaction. Neer graded his results as excellent, satisfactory, or unsatisfactory, depending on a combination of these parameters. In addition, x-ray findings, postoperative complications, patient's compliance with physical therapy, and patient's general physical condition were documented at each follow-up visit. Although no reliability or validity data are available, this early method of tracking shoulder outcomes has formed the basis from which more recent outcome scoring systems have been derived.

Constant Shoulder Score

The Constant Shoulder Score (Fig. 41-2) was described in 1987 by Constant and Murley.[33,34] It is a 100-point scoring system in which 35 points are derived from the patient's report of pain and function. The remaining 65 points are allocated for objective assessment of ROM and strength. A significant contribution of Constant's work is age- and gender-matched normative data based on a study of 900 individuals with no known shoulder dysfunction. The Constant Shoulder Score has been used internationally for reporting outcome of various shoulder conditions.[40,53,54,57,70,73,79,94,116,124,128,135,136,154] This may be why investigators at the 1992 International Shoulder Surgeons' meeting were required to present clinical data using the Constant Shoulder Score.[127] The European Shoulder and Elbow Society also requires results of clinical data to be reported using the Constant Shoulder Score.[87]

The content of the score appears to include all relevant aspects of shoulder outcome, with the exception of whether or not the patient is satisfied with his or her shoulder. However, each item of the scale requires a significant degree of interpretation by the patient. There is only one pain scale in which the patient is asked to rate the most severe pain experienced at rest, during sleep, or with various activities. Clinical experience tells us that patients experience varying degrees of pain with different activities. One pain scale appears to be inadequate to gain a true picture of the patient's pain. There is also concern that report of function is not specific to any particular activity and therefore is left to interpretation by the patient.

TABLE 41-1
CHARACTERISTICS AND PSYCHOMETRIC PROPERTIES OF SHOULDER SCALES

Scale	Dimensions (% of Total Score)	Test–Retest Reliability (ICC)	Internal Consistency	Error Estimate: SEM and MDC	Responsiveness: SRM and ES
Penn Shoulder Score (PSS)[88]	Pain: 30% Satisfaction: 10% Function: 60% Range: 0–100	Pain: 0.88[88] Satisfaction: 0.93[88] Function: 0.93[88] Total: 0.94[88]	$\alpha = 0.93$[88]	SEM (90% CI):[88] Pain ± 3.8 Satisfaction ± 1.3 Function ± 6.1 Total ± 8.5 MDC (90% CI):[88] Pain ± 5.2 Satisfaction ± 1.8 Function ± 8.6 Total ± 12.1 MCID: 11.4	SRM: 1.27[88] ES: 1.01[88]
American Shoulder and Elbow Surgeons (ASES)[121]	Pain: 50% Function: 50% Range: 0–100	Pain: 0.79[109] Function: 0.82[109] Total: 0.84[109]	$\alpha = 0.86$[109]	SEM (90% CI):[109] Pain ± 8.4 Function ± 6.7 Total ± 11.0 MDC (90% CI):[109] Pain ± 11.8 Function ± 9.5 Total ± 15.5 MCID: 6.4	SRM: 1.54[109] 0.54[83] ES: 1.4[109]
Constant Shoulder Score[33]	Pain: 15% Function: 20% Clinician assessment (ROM and strength): 65% Score range: 0–100	Total: 0.80[32]	Not tested	SEM (95% CI): ± 17.7[32]	SRM: 0.59[83]
Disabilities of the Arm, Shoulder, and Hand (DASH)[104]	Symptoms: 16.7% Disability: 83.3% Optional: sports, performing arts, or work module Score Range: 0–100	Total Score: 0.92[104]	$\alpha = 0.96$[104]	SEM: 7.6 (90% CI)[104] MDC: 12.8 (90% CI)[104]	SRM: 1.13[104] 0.70[83]
Shoulder Pain and Disability Index (SPADI)[125]	Pain: 50% Disability: 50% Score Range: 0–100	Pain: 0.64[125] 0.70–0.91[36] Disability: 0.64[125] 0.57–0.84[36] Total: 0.66[125] 0.84–0.91[36]	Pain: $\alpha = 0.86$[125] Disability: $\alpha = 0.93$[125] Total: $\alpha = 0.95$[125]	SEM (95% CI):[36] Pain ± 15.3 Disability ± 11.3 Total ± 9.3 MDC: not calculated	SRM: 1.23[10] 1.38[65]
University of California, Los Angeles (UCLA) Shoulder Scale[45]	Pain: 29% Function: 29% Satisfaction: 14% ROM: 14% Strength: 14% Score Range: 0–35	Pain: 0.59–0.78[36] Function: 0.51–0.89[36] Satisfaction: 0.79[36]*	Not tested	SEM: not calculated MDC: not calculated	SRM: not calculated ES: not calculated
Simple Shoulder Test (SST)	Function: 100% Score Range: 0–12	ICC: 0.99[10]	$\alpha = 0.85$[126]	SEM: ± 22.8[126†]	SRM: not calculated ES: not calculated

* Postsurgical patients only.
†Converted score range to 0–100.
CI = confidence interval; ES = effect size; ICC = intraclass correlation coefficient; MCID = minimal clinically important difference; MDC = minimal detectable change; ROM = range of motion; SEM = standard error of measurement; SRM = standardized response mean.

A. Subjective Assessment:

Pain during functional use:

15 14 13 12 11 10 9 8 7 6 5 4 3 2 1 0
None Mild Moderate Severe

Daily work: (work without restriction = 4 points, 75% of normal work = 3 points, 50% of normal work = 2 points, ...etc.; undisturbed sleep = 2 points, interrupted sleep = 1 point, no sleep = 0 points)

Work	Recreation	Sleep
0	0	0
1	1	1
2	2	2
3	3	
4	4	

Ability to work at the level of:

2	the waist
4	the xiphoid process
6	the neck
8	the head
10	above the head

Figure 41-2 Constant Shoulder Score.[1,19] (Adapted with permission from Constant CR, Murley AHG. A clinical method of functional assessment of the shoulder. *Clin Orthop* 1987;214:160–164.)

The objective assessment of external rotation is also questionable.[55] The patient is awarded 4 points for placing the hand behind the head with the elbow held back, whereas 8 points are awarded for hand placement on top of the head with the elbow held back. The difference of shoulder external rotation between these two hand positions seems negligible.

The method of measuring strength has not been standardized. Constant advocated testing isometric abduction strength with a spring balance at 90 degrees of abduction. He also stated that testing can be performed at the highest level achieved by patients, if they are unable to achieve 90 degrees of abduction. This inconsistency makes reproduction of results difficult. Furthermore, Conboy et al. recently questioned the validity of measuring shoulder power in a single arc of movement.[32] Bankes and colleagues[5] proposed a standard method of shoulder strength measurement for the Constant score. Their method involved a fixed spring balance and a standard test position with the arm at 90 degrees of abduction in the scapular plane, elbow extended, and forearm pronated.[5] Patients unable to achieve this test position as a result of pain or deformity are given a value of zero. The patient is asked to pull upward on the spring balance to create a 5-second isometric contraction. The highest value achieved in three maximum pulls provides the strength score.

Although the procedures and statistical analysis for determining reliability are not reported, the investigators report an average observer error of 3% among three different observers testing 100 abnormal shoulders.[33] Conboy and colleagues[32] examined the reliability of the Constant Shoulder Score in 25 patients with either arthritis, dislocation, or impingement. They found an interobserver standard deviation of 8.86 with a 95% confidence limit that the measurement of a single observer would be within 17.7 points of the true score.[32] Therefore, studies examining pre- and postintervention measures using the Constant Shoulder Score should be examined with caution. Another concern is that because such a large portion of the Constant Shoulder Score is derived from objective impairment measures, patients who are unable to return

Constant Shoulder Score
(continued)

B. Objective Assessment:

Flexion:	Abduction:
0 = 0–30º	0 = 0–30º
2 = 31–60º	2 = 31–60º
4 = 61–90º	4 = 61–90º
6 = 91–120º	6 = 91–120º
8 = 121–150º	8 = 121–150º
10 = >150º	10 = >150º

External Rotation:

2 = Hand behind head, elbow held forward

4 = Hand behind head, elbow held back

6 = Hand on top of head, elbow forward

8 = Hand on top of head, elbow back

10 = Full elevation from top of head

Internal Rotation:

0 = Hand to lateral thigh

2 = Dorsum of hand to buttock

4 = Hand to lumbosacral junction

6 = Dorsum of hand to waist (LV 3)

8 = Dorsum of hand to TV 12

10 = Dorsum of hand to interscapular region (TV 7)

Strength of abduction: (90º of abduction or highest level patient can achieve)

Trial 1: ____

Trial 2: ____

Trial 3: ____

Total = ____ / 3 = ____

Figure 41-2 (*continued*)

to the clinic will be lost to follow-up, which may lead to incomplete outcome studies.

American Shoulder and Elbow Surgeons

The ASES Standardized Shoulder Assessment Form (Fig. 41-3) was developed by the Research Committee of the ASES and published by Richards and colleagues in 1994.[121] The instrument consists of a patient self-assessment section and a clinician assessment. The patient self-report section consists of a 100-point scale with two equally weighted dimensions, pain and activities of daily living. Patients are asked to record their pain "today" on a 10-point numeric rating scale with endpoints of "no pain at

Pain:

How bad is your pain today?

0 1 2 3 4 5 6 7 8 9 10

No pain at all — Pain as bad as it can be

Activities of daily living: (Circle the number that indicates your ability to do the following activities
0 = unable to do; 1 = very difficult to do; 2 = somewhat difficult to do; 3 = not difficult)

Activity	Right Arm	Left Arm
1. Put on a coat	0 1 2 3	0 1 2 3
2. Sleep on your painful or affected side	0 1 2 3	0 1 2 3
3. Wash back/do up bra in back	0 1 2 3	0 1 2 3
4. Manage toileting	0 1 2 3	0 1 2 3
5. Comb hair	0 1 2 3	0 1 2 3
6. Reach a high shelf	0 1 2 3	0 1 2 3
7. Lift 10 lb above shoulder	0 1 2 3	0 1 2 3
8. Throw a ball overhand	0 1 2 3	0 1 2 3
9. Do usual work (list): _____	0 1 2 3	0 1 2 3
10. Do usual sport (list): _____	0 1 2 3	0 1 2 3

Figure 41-3 American Shoulder and Elbow Surgeons Standardized Shoulder Assessment Form.[141] (Adapted with permission from Richards RR, An K, Bigliani LU, et al. A standardized method for the assessment of shoulder function. *J Shoulder Elbow Surg* 1994;3:347–352.)

all" and "pain as bad as it can be."[121] The number circled is subtracted from 10 and multiplied by 5 for a possible 50 points for this section. The activities of daily living section consists of 10 questions, each with four response options ranging from "unable to do" to "not difficult." The total of the responses is calculated and multiplied by 5/3 to yield a possible score of 50 points. The measurement properties for this scale have been examined (Table 41-1).[85,109]

Although the index does include the important aspects of pain and function, it does not include the patient's satisfaction with his or her shoulder. In addition, only one pain scale is not specific to activity or time of day. The ADL section includes three items that may not be normal ADLs for some patients. This can present a problem, because there are only 10 items in this section. Sallay and Reed[131] attempted to establish normative ASES scores across age and gender. They found a mean ASES score of 95.8 points in 293 individuals who felt that their shoulder was normal. However, these authors modified the ADL subscale by offering patients an additional response option of "I don't play sports" for that item. This item was dropped from the overall score for the patients who did not play sports and the function score was calculated based on the remaining nine questions. Therefore, these normative data need to be approached with caution since the validated form of this score does not include that response option.

The advantage of this scale is its ease of administration. It can be completed by the patient independent of an examiner or by phone interview. The clinician assessment portion provides a standardized method for clinicians to follow.

Shoulder Pain and Disability Index

The Shoulder Pain and Disability Index (SPADI; Fig. 41-4) was described by Roach and colleagues in 1991.[125] This 100-point system incorporates a VAS for all items. There are five items for pain and eight for functional limitations/disability. Scoring is based on the severity of pain and disability reported. The higher the score is, the more severe the disability is.

The items of this scale relate mostly to self-care and dressing. Therefore, the SPADI does not appear to adequately measure occupational and recreational disability.[65] The investigators report fair reliability with high internal consistency of the scale (Table 41-1). They also report sensitivity to change and good correlation to objective measures of ROM. Williams and colleagues recognized the difficulties posed when using a VAS in the clinic or for

PAIN SCALE

A. How severe is your pain:

			Score
1. At its worst?	No pain	Worst pain imaginable	
2. When lying on involved side?	No pain	Worst pain imaginable	
3. When reaching for something on a high shelf?	No pain	Worst pain imaginable	
4. Touching the back of your neck?	No pain	Worst pain imaginable	
5. Pushing with the involved arm?	No pain	Worst pain imaginable	

DISABILITY SCALE

A. How much difficulty did you have:

1. Washing your hair?	No difficulty	So difficult required help	
2. Washing your back?	No difficulty	So difficult required help	
3. Putting on an undershirt or pullover sweater?	No difficulty	So difficult required help	
4. Putting on a shirt that buttons down the front?	No difficulty	So difficult required help	
5. Putting on your pants?	No difficulty	So difficult required help	
6. Placing an object on a high shelf?	No difficulty	So difficult required help	
7. Carrying a heavy object of 10 lb or more?	No difficulty	So difficult required help	
8. Removing something from your back pocket?	No difficulty	So difficult required help	

Figure 41-4 Shoulder Pain and Disability Index.[3] (Adapted with permission from Roach KE, Budiman-Mak E, Norwarat S, Lertratanakul Y. Development of a shoulder pain and disability index. *Arthritis Care Res* 1991;4:143–149.)

telephone administration.[152] They changed the format to a numeric rating scale, studied the correlation to the original version of the SPADI, and assessed its responsiveness to change over various time periods. The investigators found good correlation between the VAS and numeric scaled SPADI. They also found significant changes at each time period that correlated with the patient's global status rating. Although these studies provided evidence of reliability and validity of the SPADI, it should be noted that all of the patients in the study by Roach et al.[125] were men, and 98% of the subjects studied by Williams et al.[152] were also men. Cook and colleagues[36] reported acceptable reliability and

internal consistency of the SPADI in both nonsurgical and postsurgical shoulder patients.

University of California, Los Angeles End-Result Score

The University of California, Los Angeles (UCLA) End-Result Score (Fig. 41-5) was first used by Ellman and colleagues[45] in 1986. This is a 35-point scale. The items measured include pain (10 points), function (10 points), active forward flexion (5 points), strength of forward flexion (5 points), and patient satisfaction (5 points). A score of 34 to 35 is considered an excellent result and a score of 29 to 33 a good result. Any score less than 28 is considered a poor result.

This scale appears to include all aspects relevant to rating the shoulder. However, the UCLA score uses descriptive items for pain and function. Each item contains multiple descriptions that may make it difficult for the patient to understand. The report of patient satisfaction is also questionable. The patient has two choices for this item. Five points are awarded if he or she is "satisfied and better," whereas 0 points are awarded for "not satisfied and worse." There is no option in between. Limiting patient responses to these two options results in only gross estimates of patients' levels of satisfaction.[36] This method of measuring patient satisfaction also makes it difficult to use this scale prior to treatment, because many patients are typically not satisfied and worse when seeking medical attention.[55] Cook and colleagues[36] were unable to calculate a valid reliability value for this item in a group of 24 nonsurgical patients because eight patients changed their satisfaction responses between test administrations.

Strength of forward flexion is assessed with manual muscle testing. We have already discussed the problems associated with this method of strength testing. In addition, the position of this measurement is not standardized. Reliability has only been examined for the pain and function questions in both nonsurgical and postsurgical patients.[36] Reliability for the satisfaction question has been examined in postsurgical patients only.[36]

	Points
Pain:	
Present all of the time and unbearable; strong medication frequently	1
Present all of the time but bearable; strong medication occasionally	2
None or little at rest, present during light activities; salicylates frequently	4
Present during heavy or particular activities only; salicylates occasionally	6
Occasional and slight	8
None	10
Function:	
Unable to use limb	1
Only light activities possible	2
Able to do light housework or most activities of daily living	4
Most housework, shopping, and driving possible; able to do hair and dress and undress, including fastening brassiere	6
Slight restriction only; able to work above shoulder level	10
Normal activities	
Active forward flexion:	
>150º or more	5
120º–150º	4
90º–120º	3
45º– 90º	2
30º– 45º	1
<30º	0
Strength of forward flexion (manual muscle testing):	
Grade 5 (normal)	5
Grade 4 (good)	4
Grade 3 (fair)	3
Grade 2 (poor)	2
Grade 1 (muscle contraction)	1
Grade 0 (nothing)	0
Satisfaction of the patient:	
Satisfied and better	5
Not satisfied and worse	0

Figure 41-5 University of California, Los Angeles End Result Score. (Adapted with permission from Ellman H, Hanker G, Bayer M. Repair of the rotator cuff: end-result study of factors influencing reconstruction. *J Bone Joint Surg Am* 1986;68: 1136–1144.)

Most likely due to its ease of administration and the fact that it was one of the earlier introduced shoulder scores, the UCLA score has been used frequently in the literature for both retrospective and prospective studies.[2,4,26,45,46,62,92,95,138,139] Burkhart et al.[26] reported pre- and postoperative values for the UCLA score in a study of 14 patients who received partial repair of a massive rotator cuff defect. All but one of these patients were satisfied with their result. However, the average postoperative score was 27.6. This is considered a poor result under the guidelines set forth by the original investigators. Therefore, it would seem more practical to only report numeric results of the UCLA score and omit using terms such as "excellent" or "good."[55]

Simple Shoulder Test

The Simple Shoulder Test (SST) (Fig. 41-6) was developed by the shoulder service at the University of Washington.[6,91,100,101] The questionnaire consists of 12 functional items, derived from a review of other scales and the most frequent complaints of patients seen in their practice. The SST does not directly assess pain, ROM, or strength. Instead, these parameters are evaluated indirectly through each of the items on the questionnaire.[87] Two of the questions relate to pain, seven to function, and three to ROM.[11] Response to each item requires a simple "yes" or "no." The goal of the developers was to provide an assessment tool that does not require a clinician, elaborate equipment, or computer for calculation. Not surprisingly, excellent reliability values have been reported for this scale.[11] Beaton and Richards[11] also reported good correlation of the SST to other shoulder instruments. However, the SST has very poor precision as indicated in reports on its standard of measurement with 95% confidence interval.[37,126]

A dichotomous scale such as the SST provides excellent reliability and discriminates between patients who can and cannot perform a particular activity.[84] However, this type of scale would not perform well as an evaluative index. For example, a patient who had mild pain at night that occasionally disrupted sleep would answer the same as a patient who had severe pain at night that prevented sleep on a regular basis. There is no provision for the patient to assess satisfaction with the function of the shoulder.

Penn Shoulder Score

The Penn Shoulder Score (PSS) (Figs. 41-7 and 41-8) is a joint-specific self-report measure developed by the

Answer each question below by circling "yes" or "no" (please do not leave questions unanswered)

1. Is your shoulder comfortable with your arm at rest by your side?	Yes	No
2. Does your shoulder allow you to sleep comfortably?	Yes	No
3. Can you reach the small of your back to tuck in your shirt with your hand?	Yes	No
4. Can you place your hand behind your head with the elbow straight out to the side?	Yes	No
5. Can you place a coin on a shelf at the level of your shoulder without bending your elbow?	Yes	No
6. Can you lift 1 lb (a full pint container) to the level of your shoulder without bending your elbow?	Yes	No
7. Can you lift 8 lb (a full gallon container) to the level of your shoulder without bending your elbow?	Yes	No
8. Can you carry 20 lb at your side with the affected extremity?	Yes	No
9. Do you think you can toss a softball underhand 10 yards with the affected extremity?	Yes	No
10. Do you think you can toss a softball overhand 10 yards with the affected extremity?	Yes	No
11. Can you wash the back of your opposite shoulder with the affected extremity?	Yes	No
12. Would your shoulder allow you to work full-time at your regular job?	Yes	No

Figure 41-6 Simple Shoulder Test. (Adapted from Lippitt S, Harryman DT, Matsen FA. A practical tool for evaluating function: the simple shoulder test. In: Matsen FA, Fu FH, Hawkins RJ, eds. *The shoulder: a balance of mobility and stability.* Park Ridge, Illinois. American Academy of Orthopaedic Surgeons, 1993.)

PENN SHOULDER SCORE

Part I: Pain & Satisfaction: Please circle the number closest to your level of pain or satisfaction

	office use only
Pain at rest with your arm by your side: 0 1 2 3 4 5 6 7 8 9 10 No pain … Worst pain possible	_____ (10 – # circled)
Pain with normal activities (eating, dressing, bathing): 0 1 2 3 4 5 6 7 8 9 10 No pain … Worst pain possible	_____ (10 – # circled) (Score 0 if not applicable)
Pain with strenuous activities (reaching, lifting, pushing, pulling, throwing): 0 1 2 3 4 5 6 7 8 9 10 No pain … Worst pain possible	_____ (10 – # circled) (Score 0 if not applicable)
PAIN SCORE:	=____/30
How satisfied are you with the current level of function of your shoulder? 0 1 2 3 4 5 6 7 8 9 10 Not satisfied … Very satisfied	=____/10 (# circled)

Figure 41-7 Penn Shoulder Score pain and satisfaction subscales. (Reprinted with permission from University of Pennsylvania Shoulder and Elbow Service.)

authors.[88] It is a 100-point scale that consists of three subscales including pain, satisfaction, and function. The pain subscale consists of three pain items that address pain at rest, with activities of daily living, and with strenuous activities (see Fig. 41-7). Each item is based on a 10-point numeric rating scale with endpoints of "no pain" and "worst possible pain." Points are awarded for each item by subtracting the number circled from 10. Therefore, a patient can be awarded 30 points for the complete absence of pain. If the patient is unable to use the arm for strenuous activities, 0 points are scored for that item.

Patient satisfaction with shoulder function is also assessed with a 10-point numeric rating scale. The endpoints are "not satisfied" and "very satisfied." A maximum of 10 points for this section indicates that the patient is "very satisfied" with the current level of function of his or her shoulder.

PENN SHOULDER SCORE Part II: Function: Please circle the number that best describes the level of difficulty you might have performing each activity.	No difficulty	Some difficulty	Much difficulty	Can't do at all	Did not do before injury
1. Reach the small of your back to tuck in your shirt with your hand	3	2	1	0	X
2. Wash the middle of your back/hook bra	3	2	1	0	X
3. Perform necessary toileting activities	3	2	1	0	X
4. Wash the back of opposite shoulder	3	2	1	0	X
5. Comb hair	3	2	1	0	X
6. Place hand behind head with elbow held straight out to the side	3	2	1	0	X
7. Dress self (including put on coat and pull shirt off overhead)	3	2	1	0	X
8. Sleep on affected side	3	2	1	0	X
9. Open a door with affected side	3	2	1	0	X
10. Carry a bag of groceries with affected arm	3	2	1	0	X
11. Carry a briefcase/small suitcase with affected arm	3	2	1	0	X
12. Place a soup can (1–2 lb) on a shelf at shoulder level without bending elbow	3	2	1	0	X
13. Place a one-gallon container (8–10 lb) on a shelf at shoulder level without bending elbow	3	2	1	0	X
14. Reach a shelf above your head without bending your elbow	3	2	1	0	X
15. Place a soup can (1–2 lb) on a shelf overhead without bending your elbow	3	2	1	0	X
16. Place a one-gallon container (8–10 lb) on a shelf overhead without bending your elbow	3	2	1	0	X
17. Perform usual sport/hobby	3	2	1	0	X
18. Perform household chores (cleaning, laundry, cooking)	3	2	1	0	X
19. Throw overhand/swim/overhead raquet sports. (circle all that apply to you)	3	2	1	0	X
20. Work full-time at your regular job	3	2	1	0	X
SCORING: Total of columns = ____ (a) Number of "X's" × 3 = ____(b), 60 – ____(b) = ____ (c) (if no "X's" are circled, function score = total of columns) Function Score = ____(a) ÷ ____(c) = ____ × 60 ____/60					

Figure 41-8 Penn Shoulder Score function subscale. (Reprinted with permission from University of Pennsylvania Shoulder and Elbow Service.)

The function subsection (see Fig. 41-8) is based on a sum of 20 items, each with a 4-point Likert scale. The response options include 0 (can't do at all), 1 (much difficulty), 2 (with some difficulty), and 3 (no difficulty). A patient is awarded 60 points if all activities can be performed without difficulty. Because some items in this subscale may not be applicable to all patients, the response option "did not do before injury" is available. For scoring purposes, the total possible points for the function subscale are reduced by 3 when this option is circled. Scoring

Part III: ACTIVE RANGE OF MOTION

Points are based on a % of INV/UNINV shoulder AROM	AROM		PROM		% UNINV	PTS
	INV	UNINV	INV	UNINV		
Forward elevation					%	
External rotation at 0º abduction					%	
External rotation at 90º abduction					%	
Internal rotation (level of hitchhiking thumb)					%	
TOTAL POINTS =						/40

Forward elevation & External rotation AROM scoring	Internal rotation AROM scoring
96 – 100% = 10 points	<2 Spinal levels = 10 pts
85 – 95% = 9 points	
75 – 84% = 8 points	3 – 5 Spinal levels = 8 pts
65 – 74% = 7 points	
55 – 64% = 6 points	6 – 12 Spinal levels = 6 pts
45 – 54% = 5 points	
35 – 44% = 4 points	Sacrum = 4 pts
25 – 34% = 3 points	
15 – 24% = 2 points	Buttock = 2 pts
5 – 14% = 1 point	
0 – 4% = 0 points	Side of thigh = 0 pts

Figure 41-9 Penn Shoulder Score active range of motion (AROM) scoring. INV = involved; PROM = passive range of motion; UNINV = uninvolved. (Reprinted with permission from University of Pennsylvania Shoulder and Elbow Service.)

is based on a percentage of the total possible points. For example, a patient has a total score for the function subsection of 27 points. He or she responded, "did not do before injury" for two items. Therefore, the total possible points would be 54 (60 − 6). The final function score would be calculated: 27 ÷ 54 = 0.5; then 0.5 × 60 = patient's function subscale score of 30 points.

The total PSS maximum score of 100 indicates high function, low pain, and high satisfaction with the function of the shoulder. The PSS can be used in the aggregate or each subscale individually. Most patients complete the scale in approximately 5 minutes, and the clinician can typically calculate the final scores in less than 2 minutes.[108]

The measurement properties of the PSS have been examined (Table 41-1).[88] Two additional studies examined specific attributes of the PSS. Cook and colleagues[37] examined the error associated with the function subscale and compared it to other shoulder scales at differing levels of function. They found that the PSS had much better precision throughout all score ranges than the ASES and SST.[37] The PSS had precision nearly equal to that of the SPADI at the middle-range scores, but exhibited better precision at the high and low scores.[37] Michener and colleagues[109] demonstrated convergent construct validity of the ASES score by reporting correlation with the PSS of 0.78. The PSS has also been used as an outcome measure in two clinical trials involving patients with shoulder impingement syndrome and rotator cuff repair.[36,103]

There is also a separate, clinician- or impairment-based score that may be used in conjunction with the self-report scale. The 100-point impairment score consists of objective measures of ROM and strength (Figs. 41-9 and 41-10). Shoulder active range of motion (AROM) is recorded as recommended by the ASES and includes forward elevation, external rotation with the arm at the side,

Part IV: STRENGTH:				
Device: _____(kg lbs. ft/lbs) (circle one)	INV	UNINV	% Uninv	PTS
External rotation at side			%	
Internal rotation at side			%	
Elevation at 45º in POS			%	
TOTAL POINTS =				/60

Strength Scoring
>90% = 20 points
80 – 89% = 18 points
70 – 79% = 16 points
60 – 69% = 14 points
51 – 59% = 12 points
40 – 49% = 10 points
30 – 39% = 8 points
20 – 29% = 6 points
10 – 19% = 4 points
1 – 9% = 2 points
0% = 0 points

Figure 41-10 Penn Shoulder Score strength scoring. INV = involved; POS = plane of scapula; UNINV = uninvolved. (Reprinted with permission from University of Pennsylvania Shoulder and Elbow Service.)

external rotation at 90 degrees of abduction, and internal rotation.[25,121] All motions are measured in the seated position.

Fig. 41-9 demonstrates the scoring for each of the ROM parameters. Scoring for forward elevation and external rotation is based on a percentage of the opposite uninvolved side. Internal rotation scoring is based on the level that the hitch-hiking thumb can reach, until the patient is able to reach the lumbar or thoracic spinous processes. Scoring then is based on a comparison of the spinal level achieved by the opposite uninvolved extremity. Ten points can be awarded for each ROM parameter for a total of 40 points for this section.

Strength testing should be performed with an isometric dynamometer. Internal and external rotation is measured with the arm at the side and in neutral rotation. Elevation is measured at 45 degrees in the plane of the scapula. Scoring is based on a percentage of the opposite normal side (see Fig. 41-10). Ten points can be awarded for each position for a maximum of 30 points for this section. This method of recording strength has demonstrated good reliability with three different isometric dynamometers.[89] A limitation of basing a score on a percentage of the opposite side assumes that the "uninvolved" side is normal. In addition, this type of scoring would not be useful for patients with two "involved" shoulders or those who have undergone surgery on that shoulder.

SUMMARY

The necessity of documenting outcome of surgical and therapeutic intervention of shoulder disorders is receiving increased emphasis in health care today. More importantly, clinicians must be able to compare results of different forms of treatment, compare results reported by other clinicians, and learn the confounding factors that may affect outcome. To accomplish this, standardized methods must be universally accepted. Available outcome scoring systems must meet the necessary criteria before they can be used. Once these systems are in place, the most effective treatments will be offered for the least cost.

Improving the patient's pain, function, and satisfaction should be the goal of any treatment intervention. Knowledge of the factors that contribute to that improvement is important in determining their relationship to the overall patient outcome. A patient self-assessment tool that measures outcome for the general population of shoulder patients should be used. In addition, the level of improvement in impairments such as ROM and strength should be compared with the patient's assessment of outcome. Use of a generic health status measure provides data as to the effect a shoulder problem has on the patient's overall function. Integrating these data will help to guide treatment and predict outcome, thereby improving the efficiency of our health care system.

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Pain Management

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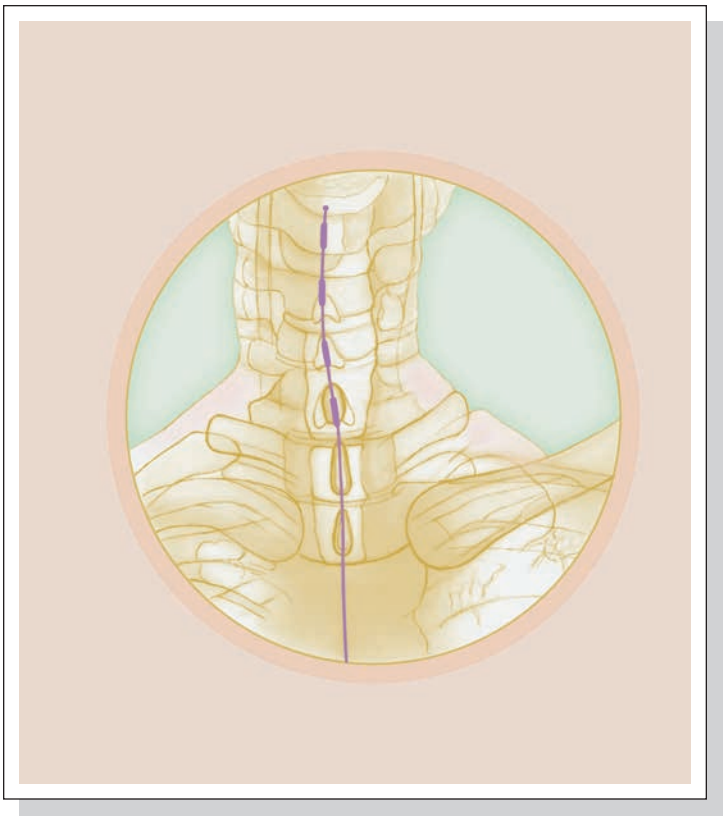

42 Medical Management of Chronic Shoulder Pain

Samer N. Narouze *Nagy A. Mekhail*

EPIDEMIOLOGY 1319

CLASSIFICATION 1319

CHRONIC SOMATIC SHOULDER PAIN 1320
Regional Myofascial Pain Syndrome 1320
Fibromyalgia 1321

CHRONIC NEUROPATHIC SHOULDER PAIN 1321
Cervical Pathology 1321
Brachial Plexopathy 1323
Thoracic Outlet Syndromes 1324
Entrapment Syndromes 1326

COMPLEX REGIONAL PAIN SYNDROME 1328
Sympathetically Maintained Pain 1328
Sympathetically Independent Pain 1328
Complex Regional Pain Syndrome 1329

PHARMACOLOGIC MANAGEMENT OF CHRONIC SHOULDER PAIN 1332
Acetaminophen 1333
Nonsteroidal Antiinflammatory Drugs 1333
Antiepileptics 1335
Opioids 1337
Antidepressants 1341
Centrally Acting Agents 1342

Pain is defined by the International Association for Study of Pain (IASP) as "an unpleasant sensory and emotional experience associated with actual or potential tissue damage or described in terms of such damage."[101]

EPIDEMIOLOGY

Pain is one of the most significant health care crises in the United States. Nearly half of Americans see a physician with a primary complaint of pain each year, making pain the single most frequent reason for physician consultation in the United States.[70]

At least 60 million Americans suffer from chronic pain problems. Disability due to pain is a major problem in the United States. Three-fourths of the disablement relate to pain.[89]

CLASSIFICATION

Pain is classified in many ways including acute or chronic, organic or psychogenic, and malignant or nonmalignant.[54–75]

Acute pain is defined as pain temporally related to a precipitating event. It is associated with autonomic nervous system hyperactivity, including tachycardia, increased blood pressure, and anxiety.

Chronic pain is pain lasting more than 3 to 6 months. Objective clinical findings for chronic pain include depression, functional disruptions such as withdrawal from social activities, and personality and lifestyle changes. Therefore, in relation to pain, "chronic" describes not only duration, but also a syndrome with specific therapeutic implications.[49] Chronic pain is classified as organic versus psychologic pain.

Organic pain may be nociceptive or neuropathic.

Nociceptive pain is found in cutaneous or deep tissue (somatic) and organs (visceral). Nociceptive pain

results from direct stimulation of intact peripheral afferent nerve endings that are sensitive to noxious mechanical, thermal, or chemical stimuli.

Somatic pain is characterized as constant and easily identifiable. An example of chronic somatic pain is metastatic bone pain. This pain may be persistent, diffuse, and unrelated to position or movement, or it may be intermittent, localized, and related to position and weight-bearing or physical activity

Visceral nociceptive pain tends to be less well localized and is described as dull, dragging, and deep in nature.

Neuropathic pain is caused by injury or disease of the nervous system. It may be divided into central and peripheral nervous system disturbances and is designated as deafferentation pain. Nervous system injury may result from direct trauma, ischemia (e.g., thalamic syndrome), infection (e.g., postherpetic neuralgia), metabolic derangement (e.g., diabetic neuropathy), or tumor invasion.

Neuropathic pain may be constant and steady or intermittent and lancinating and is described as burning, shooting, or tingling. Pain may be experienced as abnormal or altered sensation (dysesthesia), paresthesia (electrical shock sensation), hyperalgesia (extreme sensitivity to painful stimuli), or allodynia (pain with touch).

Peripheral naturopathic pain is secondary to peripheral hyperexcitability, which is due to a series of molecular changes at the level of the peripheral nociceptor, in dorsal root ganglia, in the dorsal horn of the spinal cord, and in the brain. These changes include abnormal expression of sodium channel activity at glutamate receptor sites, change in γ-aminobutyric acid (GABA-ergic) inhibition, and an alteration of calcium influx into cells.[67]

Psychogenic pain is defined as a somatoform pain disorder, which is a diagnosis of exclusion.[49]

Chronic pain of the shoulder is usually due to somatic or neuropathic pain.

CHRONIC SOMATIC SHOULDER PAIN

Periarticular Soft Tissue

Impingement syndromes
Rotator cuff tear (partial and complete)
Bicipital tendon tear/tendinitis
Adhesive capsulitis/frozen shoulder
Glenohumeral instability

Joints

Osteoarthritis (glenohumeral or acromioclavicular)
Septic arthritis
Rheumatoid arthritis
Gout and pseudogout
Osteonecrosis
Subluxation (acromioclavicular or sternoclavicular)

Bone

Fracture
Osteomyelitis
Tumor

Muscles

Myofascial pain syndrome
Fibromyalgia

Regional Myofascial Pain Syndrome

The hallmark characteristics of regional myofascial pain are (a) localization within a circumscribed muscle or group of muscles in a particular area of the body and (b) the presence of trigger points.[140] Trigger points are hyperirritable nodules that feel like cords of rope within the body of a skeletal muscle or surrounding fascia. Trigger points can develop after different types of trauma, many of which are seemingly insignificant. Quite often, trigger points are the result of repetitive injury (e.g., trigger points with the body of trapezius in a typist).

On physical examination, the trigger point can produce a localized involuntary twitch response to palpation. This involuntary twitch response represents a brief contraction of muscle fibers within an involved muscle. At the same time, a patient also may have a voluntary response to palpation of a trigger point. This voluntary response is most commonly manifested as patient movement or vocalization and has been called the jump sign. Trigger points within specific muscles have well-described, characteristic referral patterns to other areas of the body.[158] These patterns of radiation may not seemingly follow normal anatomic radiation patterns (i.e., radiation along the course of peripheral nerves or dermatomes). However, the somewhat atypical and unusual patterns of trigger-point radiation are well described and reproducible.

The pathogenesis of trigger points is unclear. Injured muscles that do not heal and progress to chronic pain appear to develop problems within the sarcomere early in the natural history of the process. Uncontrolled contraction of the sarcomere may lead to development of a trigger point.[142] On a molecular basis, inability to physiologically utilize calcium may cause actin and myosin to function improperly. Thus, a muscle fibril becomes, in essence, paralyzed and unable to relax.[139]

Treatment of regional myofascial pain involves medical, invasive, and physical therapeutic modalities. Nonsteroidal antiinflammatory medications (NSAIDs) and tricyclic antidepressants (TCAs) have been shown to provide effective analgesia.[25,134]

Muscle relaxants have been widely used in the treatment of myofascial pain, despite the fact that they have not been

rigorously studied to the same extent as NSAIDs and antidepressants. However, some patients will anecdotally report great benefit from the use of muscle relaxants. Membrane-stabilizing drugs have not been shown to produce significant analgesia in regional myofascial pain.

Injection of active trigger points has been advocated by some investigators, although scientific studies have been inconclusive regarding their long-term effects.[134] Trigger-point injections have utilized saline, local anesthetics, and corticosteroids or have been given without medications (dry needling).

Physical medicine modalities are the most important treatment for regional myofascial pain.

Fibromyalgia

Fibromyalgia is a musculoskeletal disorder with marked similarity to regional myofascial pain.[53,98] The prevalence of fibromyalgia in the population is approximately 0.7% to 3.2%.[66] The high prevalence of disability among patients with fibromyalgia represents a significant cost to society in health care dollars.[90]

Although regional myofascial pain and fibromyalgia share many characteristics, they are distinct clinical entities. Myofascial pain is topographically discrete and localized, whereas fibromyalgia is diffuse and widespread. The American College of Rheumatology has developed criteria for the diagnosis of fibromyalgia.[172] When 11 of 18 tender points elicit pain by palpation (in nine paired anatomically defined sites) with concomitant widespread muscular aching, a sensitivity of 88.4% and a specificity of 8l.1% are achieved for the diagnosis of fibromyalgia. Trigger points are deemed to be specific for regional myofascial pain syndrome. Trigger points elicit pain upon palpation of the muscle belly, whereas tender points occur at the muscle–tendon junction. Moreover, trigger points have reproducible patterns of radiation, whereas tender points do not radiate.

Regional myofascial pain syndrome is the result of trauma, microtrauma, or repetitive injury to muscle. On the other hand, fibromyalgia presents without a history of trauma or even an inciting event.

Fatigue and sleep disturbance are found in fibromyalgia but not in regional myofascial pain.[173] The sleep disturbance is characterized by a nonrestorative pattern. The patient awakens each morning feeling unrested.[99]

Fibromyalgia has been reported in conjunction with connective tissue.[167] The prevalence of regional myofascial pain is not increased in rheumatologic disorders in comparison with the general population.

Two of the anatomically defined sites for the diagnosis of fibromyalgia by the American College of Rheumatology lie within the musculature of the shoulder.[172] Thus, pain in the shoulder musculature as a presenting feature of fibromyalgia is not unusual.

The treatment of fibromyalgia should involve interdisciplinary expertise. The cornerstone of therapy is pharmacologic management with NSAIDs and TCAs.[25,134] Use of trigger-point injections has been uniformly disappointing in patients with fibromyalgia because of the diffuse nature of the disease. Physical therapy is paramount in the treatment of fibromyalgia, but many patients reject it because of the painful nature of the disease process. However, all physical therapeutic efforts should be formulated within the framework of a strengthening and conditioning program. Many patients with fibromyalgia lack insight into their disease, as well as coping strategies, and may appear depressed and anxious.[71] Many patients may benefit from psychological intervention, as well as psychotropic medication when appropriate.

CHRONIC NEUROPATHIC SHOULDER PAIN

- Spinal cord lesions
- Cervical pathology
  - Spondylosis
  - Degenerative disc disease
  - Herniated nucleus pulposus
  - Radiculopathy
- Spinal nerve lesions
- Brachial plexopathy
  - Stretch, compression, trauma
  - Avulsion
  - Stingers and burners
  - Intraoperative injuries
  - Brachial plexus neuritis
  - Tumors
  - Radiation neuritis
- Thoracic outlet syndrome (TOS)
  - Neurologic TOS
  - Vascular TOS
  - TOS of pain and sensory symptoms
- Entrapment syndromes
  - Suprascapular nerve
  - Long thoracic nerve
  - Axillary nerve
- Complex regional pain syndrome (CRPS)
  - CRPS I (reflex sympathetic dystrophy)
  - CRPS II (causalgia)

Cervical Pathology

Cervical spondylosis involves osteoarthritic degenerative changes of the joints of the cervical spine: the disc itself, the facet, and the uncovertebral joints. Osteophytes may form at all of the aforementioned joints and encroach upon the nerve root foramina. Changes in the cervical discs themselves consist of chronic degeneration and desiccation.

The discs can flatten and bulge outward with promotion of osteophyte formation.

Disc herniation consists of protrusion of the nucleus pulposus through a tear in the annulus fibrosus. The nerve root may be compressed within its foramen when the herniation occurs in a lateral or dorsolateral direction, thereby potentially generating a cervical radiculopathy. Disc herniation may occur in conjunction with chronic spondylotic changes in the cervical spine.

Cervical radiculopathy and myelopathy may be generated by acute, subacute, or insidious (with the possibility of spinal cord compression) disc herniation. Acute cervical disc herniation is much less common than in the lumbar area. Pain is the predominant symptom of acute cervical radiculopathy. The pain is often widely distributed over the shoulder, scapula, upper chest, and arm, and may be appreciated deep within certain muscles. Radicular pain (paresthesia) is present in a dermatomal pattern. Muscle weakness is usually mild but can be severe. The dermatomal distribution of the radicular pain, as well as weakness within certain muscles (Table 42-1), may be diagnostic aids in determination of the involved root.

Subacute cervical radiculopathy is much more common than acute radiculopathy and results from disc herniation occurring in conjunction with chronic spondylotic changes. Pain and paresthesias are present, as in the acute form. Unlike acute radiculopathy, mild sensory loss or hyperesthesia, along with moderate weakness, is common. The most commonly involved discs are C5–6 and C6–7.[171]

Radicular myelopathy may result from compression of the spinal cord. Cervical degenerative discs, spondylosis of the facet and/or uncovertebral joints, and posterior vertebral osteophytes may be involved in the compressive mechanism. Furthermore, a hypertrophied and inelastic ligamentum flavum may compress the cord with neck extension.[153] The result of these chronic spondylotic changes with spinal cord compression is a myelopathic constellation of symptoms affecting both the arm and legs. In contradistinction to acute and subacute radiculopathy, pain and sensory changes are usually minimal with respect to upper limb symptoms. On the other hand, muscle weakness and wasting may be severe. Abnormalities in the legs include spastic weakness and variable sensory loss (with vibratory sense being most affected). Deep tendon reflexes in the legs are hyperreflexic, whereas those in the arms are hyporeflexic.

Computed tomography (CT), myelography, CT myelography, and magnetic resonance imaging (MRI) are essential

TABLE 42-1
PATTERNS OF MUSCLE WEAKNESS RESULTING FROM BRACHIAL PLEXUS LESIONS

Brachial Plexus	Nerve Branch	Muscles
Trunks		
Upper	Suprascapular (C5–6)	Supraspinatus, infraspinatus
	Lateral pectoral (C5–7)	Clavicular portion of the pectoralis major
	Musculocutaneous (C5–7)	Biceps
	Lateral portion median (C5–7)	Pronator teres, flexor carpi radialis
	Radial (C5–6)	Brachioradialis
	Axillary (C5–6)	Deltoid
Middle	Thoracodorsal (C6–8)	Latissimus dorsi
	Subscapular (C5–6)	Teres major
	Radial (C7)	All radial innervated muscles (except brachioradialis)
	Lateral portion median (C5–7)	Pronator teres, flexor carpi radialis
Lower	Medial pectoral (C8–T1)	Sternal portion of pectoralis major
	Ulnar (C8–T1)	All ulnar innervated muscles
	Medial portion median (C8–T1)	Flexors of hand, median innervated intrinsic muscles of hand
Cords		
Lateral	Musculocutaneous (C5–7)	Biceps
	Lateral portion median (C5–7)	Pronator teres, flexor carpi radialis
Posterior	Thoracodorsal (C6–8)	Latissimus dorsi
	Subscapular (C5–6)	Teres major
	Axillary (C5–6)	Deltoid
	Radial (C5–T1)	All radial innervated muscles
Medial	Ulnar (C8–T1)	All ulnar innervated muscles
	Medial portion median (C8–T1)	Flexors of hand, median innervated intrinsic muscles of hand

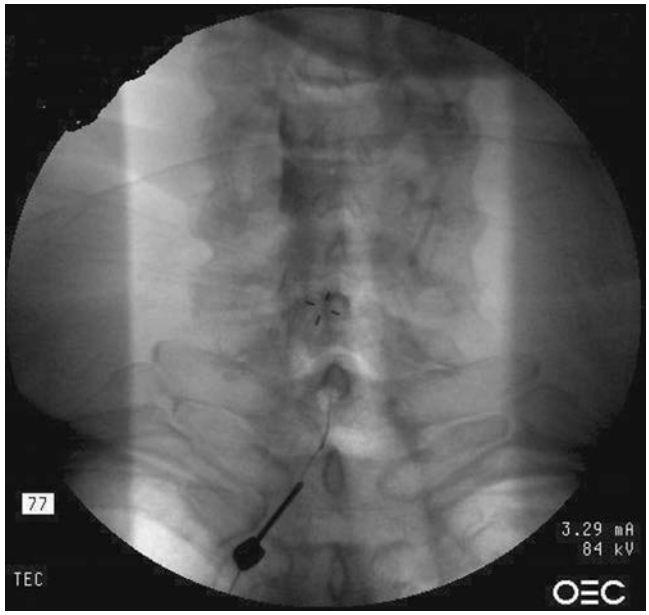

Figure 42-1 Anteroposterior view showing cervical interlaminar cervical epidural injection.

for the evaluation and diagnosis of mechanical and radicular cervical spine pain.

Treatment of the pain of spondylosis itself may be amenable to NSAIDs. Acute and subacute radiculopathy may be ameliorated by NSAIDs, cervical interlaminar epidural steroid injections (CESIs)[27–44] (Fig. 42-1), and cervical transforaminal steroid injections,[142,162] with rehabilitation facilitated by physical therapy. Judicious surgical intervention may guard against the devastating effects of radiculomyelopathy.

Cervical Epidural Steroid Injection: Interlaminar Versus Transforaminal Approach

The advantage of the transforaminal approach over the interlaminar one is that the transforaminal route is more target specific as the injectant is delivered to the target nerve and the anterior epidural space (pathology site) in maximum concentration.

The major disadvantage of the cervical transforaminal steroid injection is the higher risk of serious complications: injection in the vertebral or radicular artery, spinal cord or brainstem infarction, and nerve root injury. That is why it should be done only by experienced clinicians under real-time fluoroscopy.[117]

Brachial Plexopathy

Traumatic Injury

Because of its superficial location, the mobility of the shoulder and the neck, and its close proximity to bony structures, the brachial plexus is particularly susceptible to traumatic injury. Stretch (traction) injuries are particularly common after falls and motorcycle accidents, when the head and neck are moved in one direction and the shoulder is moved in the opposite direction. The brachial plexus also may be compressed or crushed between the clavicle and underlying ribs or damaged by bony fragments, displaced muscles, hematomas, or missiles.[150]

Fractures and dislocations of the shoulder frequently damage the brachial plexus.[83] Dislocations of the shoulder without a concomitant fracture more frequently injure the axillary nerve than the brachial plexus itself, however. Bony fragments or hematoma associated with fractures of the clavicle can damage the plexus and cause brachial plexopathy.[175] When damage to the brachial plexus does occur, it is usually diffuse, with varying degrees of injury associated with different parts of the plexus. However, injury may be restricted to just one trunk or cord.

Nerve Root Avulsion

Avulsion of spinal nerve roots involves tearing of the dorsal and ventral roots of one or more spinal nerves from the spinal cord. Of course, avulsion is a particularly serious result of stretch injuries to the brachial plexus. The lower cervical roots (including T1) are most often involved. Neuropathic pain is almost uniformly produced after traumatic root avulsion.[109]

The topographic distribution of pain conforms to the dermatome of the avulsed root(s). (Hand and forearm pain occurs after C6, C7, C8, or T1 root avulsions. Shoulder pain occurs after C5 root avulsion.)

Burning, electric shock, or pressure sensations form a continuous background of severe pain. Superimposed upon this background are paroxysms of pain of unbearable intensity that radiate from the hand to the shoulder and are described as sharp, shooting, or lightning-like. More typical paresthesias following normal dermatomal distributions also occur.

Careful physical examination (Table 42-1) is essential in localization of the injury to a specific area of the plexus. In conjunction with physical examination, radiographic studies and electrophysiologic studies are essential in the diagnosis of root avulsion.

The most optimal assessment of nerve root avulsion and plexus injury combines radiographic visualization with electrophysiologic testing. Electromyography (EMG) can assist in the assessment of nerve root avulsion, because it permits evaluation of the paraspinal muscles (which cannot be evaluated clinically) and because it may indicate the severity of damage. In nerve root avulsion, EMG of paraspinal muscles is abnormal, because damage to the roots is proximal to the dorsal rami.[22] Sensory action potentials recorded from the arm are normal in nerve root avulsion because distal sensory fibers are still viable, because they are not separated from their cell bodies. Sensory action potentials may be normal, even in the face of dense anesthesia in the distribution of the

avulsed root(s).[168] In contrast, sensory action potentials are reduced or absent in brachial plexus lesions, because damage to nerve fibers is distal to the dorsal root ganglion.

During the first 3 to 4 months after avulsion, spontaneous recovery from neurapraxia can occur. During this time period, neuropathic pain should be treated with TCAs and membrane-stabilizing agents. Aggressive physiotherapy is important to prevent contractures and joint ankylosis. If no nerve regeneration is present after 4 months and if electrophysiologic studies show evidence of severe axonal degeneration, the brachial plexus should be surgically explored. Surgical repair of the brachial plexus can restore function and reduce pain.[102,106]

Ablative procedures should not be entertained until the possibility of de novo nerve regeneration or functional recovery has waned. Amputation should not be considered, because it will not relieve the pain of root avulsion. Ablation of the dorsal root entry zone (DREZ) lesion can greatly reduce the pain of nerve root avulsion.[156]

Intraoperative Brachial Plexus Injuries

Damage to the brachial plexus may occur during general anesthesia. Such injuries are due to hyperabduction of the arm and stretch of the plexus because of poor positioning of the patient.[95] Intraoperative brachial plexus injury is particularly common after procedures involving median sternotomy and has been estimated to occur in 5% of all such patients.[81] The lower trunk or medial cord is most commonly involved, although any part of the plexus can be damaged.

Brachial Plexus Neuritis

Acute brachial plexus neuritis can develop at any age for reasons that are obscure. The pain is of great intensity and does not follow a radicular or peripheral nerve distribution. Sensory abnormalities are usually of less intensity than the pain and muscle weakness. The serratus anterior, deltoid, infraspinatus, and supraspinatus muscles are most commonly involved, although involvement of individual muscles or muscle groups is highly variable. Weakness of the musculature may indicate involvement of a single nerve, two nerves, or a discrete part of the plexus, usually the upper trunk. Acute brachial plexus neuritis is asymmetric in only about one-third of cases and is most commonly found as a bilateral condition.[161]

Different causes have been suggested as the key factors in the pathogenesis of brachial plexus neuritis including infections, trauma, vasculitis, and surgical events.[152,175] No cause-and-effect relationship has been definitively demonstrated.[14,151]

Malignant Invasion

Tumors invading the brachial plexus include breast cancer (most common) and Pancoast's tumor. In breast cancer, brachial plexus invasion occurs via spread from lymph nodes or bone metastases. The predominant symptom is shoulder pain that radiates down the arm and forearm into the fingers (depending on the trunk involved). Paresthesias and weakness develop later. Malignant invasion of the brachial plexus is rarely an early manifestation of breast cancer.

In contradistinction, brachial plexopathy presents as an early manifestation of Pancoast's tumor.[82] Pancoast's tumor (superior pulmonary sulcus tumor) is almost always caused by invasion of the lower trunk of the brachial plexus by carcinoma of the lung at the apex. The presenting symptom is usually pain radiating along the inner aspect of the arm. Weakness and sensory changes are usually localized to the lower trunk of the brachial plexus. Almost two-thirds of patients will develop Homer's syndrome, due to infiltration of the inferior cervical sympathetic ganglion.[60]

Radiation Neuritis

Radiation-induced brachial plexopathy with fibrosis of the brachial plexus may occur after radiation to the upper chest wall and supraclavicular area for the treatment of malignant tumors. Radiation damage to the brachial plexus is dose dependent. Onset of plexopathy may be years after irradiation. Radiation-induced brachial plexopathy can occur without any sign of radiation damage to the overlying skin. The presence of nonneurologic signs (e.g., lymphedema and induration) may be found in both radiation neuritis and malignant invasion of the brachial plexus.[157]

Thoracic Outlet Syndromes

The thoracic outlet syndromes comprise three different clinical pathophysiologic entities.[151]

Neurologic Thoracic Outlet Syndrome

Neurologic thoracic outlet syndrome is found mainly in women. It begins to manifest itself as pain along the medial aspect of the arm and forearm. Paresthesias are found extending into the ulnar border of the hand. Muscle weakness and atrophy occur later. Muscle atrophy is selective for the abductor pollicis brevis and the opponens muscles of the thenar eminence. Wasting of all the intrinsic muscles of the hand is possible with progression of the condition. There are no vascular symptoms. Sensory abnormalities are referable to the lower trunk of the brachial plexus.[51]

A well-developed cervical or rudimentary cervical rib with a fibrous band extending to the upper surface of the first rib may impinge on the lower trunk of the brachial plexus (Fig. 42-2). Similarly, anatomic abnormalities of the first rib may stretch and angulate the lower trunk of the brachial plexus. The scalenus anticus syndrome involves hypertrophy of the anterior scalene muscle and compression

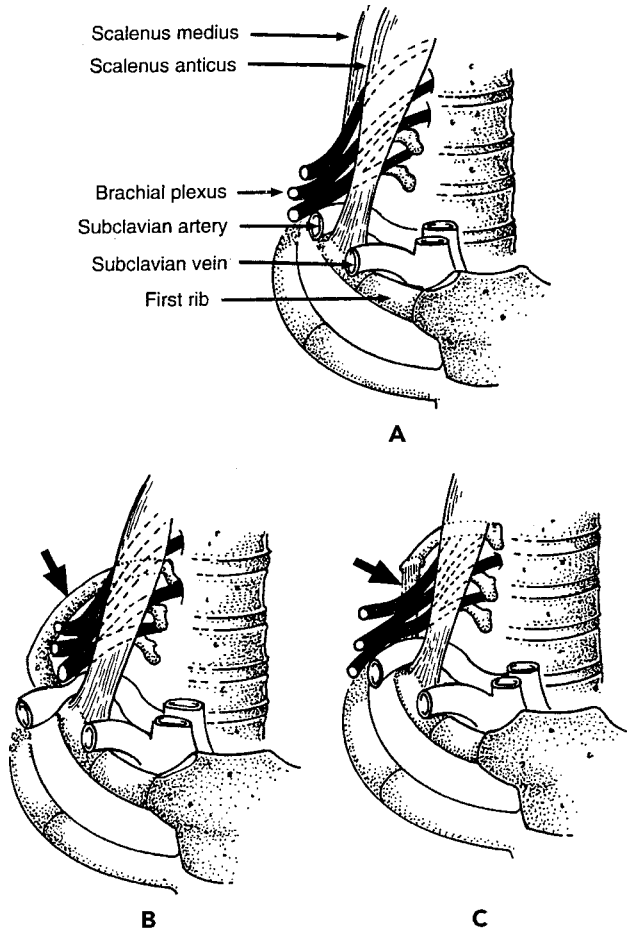

Figure 42-2 **(A)** Normal anatomic relationships of the subclavian artery and the brachial plexus at the cervicothoracic junction. **(B)** Vascular thoracic outlet syndrome. The subclavian artery is angulated over a well-developed cervical rib. The subclavian artery is also sandwiched between the cervical rib and the anterior scalene muscle. Both these anatomic relationships cause stenosis of the subclavian artery and poststenotic dilation of the artery. **(C)** Neurologic thoracic outlet syndrome. The normal anatomic relationships of the first rib, lower trunk, medial cord of the brachial plexus, subclavian artery, and anterior scalene muscle are shown on the right. On the left, a small cervical rib and a fibrous band are depicted. The lower trunk of the brachial plexus is angulated over this band, and this anatomic relationship can generate neurologic thoracic outlet syndrome. (Reprinted with permission from Wyburn-Mason R. Brachial neuritis occurring in epidemic form. *Lancet* 1941;2:662–663.)

of the brachial plexus as it passes through the interscalene groove between the anterior and middle scalene muscles (Fig. 42-3). Hyperabduction syndrome or subcoracoid-pectoralis minor syndrome involves compression of the brachial plexus with lateral abduction of the arm to an elevated position. In this position, the brachial plexus and vasculature are compressed by tension of the pectoralis minor muscle and, to a lesser extent, the coracoid process.[52,88]

Physical examination, roentgenographs, and electrophysiologic studies should be employed in determination of the diagnosis of neurologic thoracic outlet syndrome. Roentgenographs will almost uniformly show the characteristic bony abnormality responsible for the neurologic symptomatology. If a bony abnormality is not seen, then consideration must be given to the presence of fibrous bands, scalenus anticus syndrome, or hyperabduction syndrome. Electrophysiologic studies can differentiate neurologic thoracic outlet syndrome from carpal tunnel syndrome and ulnar neuropathy.[52] Short-latency somatosensory evoked potentials (SSEPs), following stimulation of the ulnar nerve at the wrist, may actually be more useful.[23]

Supraclavicular exploration with release of the compressive anatomy is the most satisfactory treatment for neurologic thoracic outlet syndrome. Patients experience immediate relief of pain. Unfortunately, muscle weakness and atrophy do not usually improve.[51]

Vascular Thoracic Outlet Syndrome

In patients with a well-developed first rib, the subclavian artery may be angulated over the cervical rib and wedged between the anterior scalene muscle and the cervical rib. The subclavian artery will become narrowed and will develop an area of poststenotic dilation. Thrombus may accumulate in the poststenotic dilation, fragment, and embolize to the hand. This produces vascular symptomatology with intermittent blanching of the hands and fingers. Pulses may be diminished or absent. A bruit can sometimes be auscultated in the supraclavicular and/or axillary areas. The vascular thoracic outlet syndrome consists only of vascular symptomatology without neurologic abnormalities. Angiography will confirm the diagnosis. Surgical excision of the cervical rib effectively treats vascular thoracic outlet syndrome.[40]

Thoracic Outlet Syndrome of Pain and Sensory Symptoms

The largest group of patients with thoracic outlet syndrome will present with less clearly defined symptomatology than the aforementioned two rare, but well-defined, thoracic outlet syndromes. Such patients will usually present with a diffuse nagging ache and numbness in the arm. The pain in the arm will be made worse by carrying heavy objects or holding the arm in particular positions. Paresthesias may occur concomitantly with the pain or may be temporally distinct. Paresthesias are usually appreciated in the medial aspect of the arm and forearm and sometimes in the hand.

Various proposed mechanisms include (a) compression of the subclavian artery and/or brachial plexus between the lower end of the anterior scalene muscle and the first rib[51]; (b) vascular factors, venous rather than arterial compression[124]; and (c) costoclavicular syndrome.[42,43] The costoclavicular syndrome suggests that the subclavian artery and/or the brachial plexus are intermittently compressed between the clavicle and a normal first rib. Patients

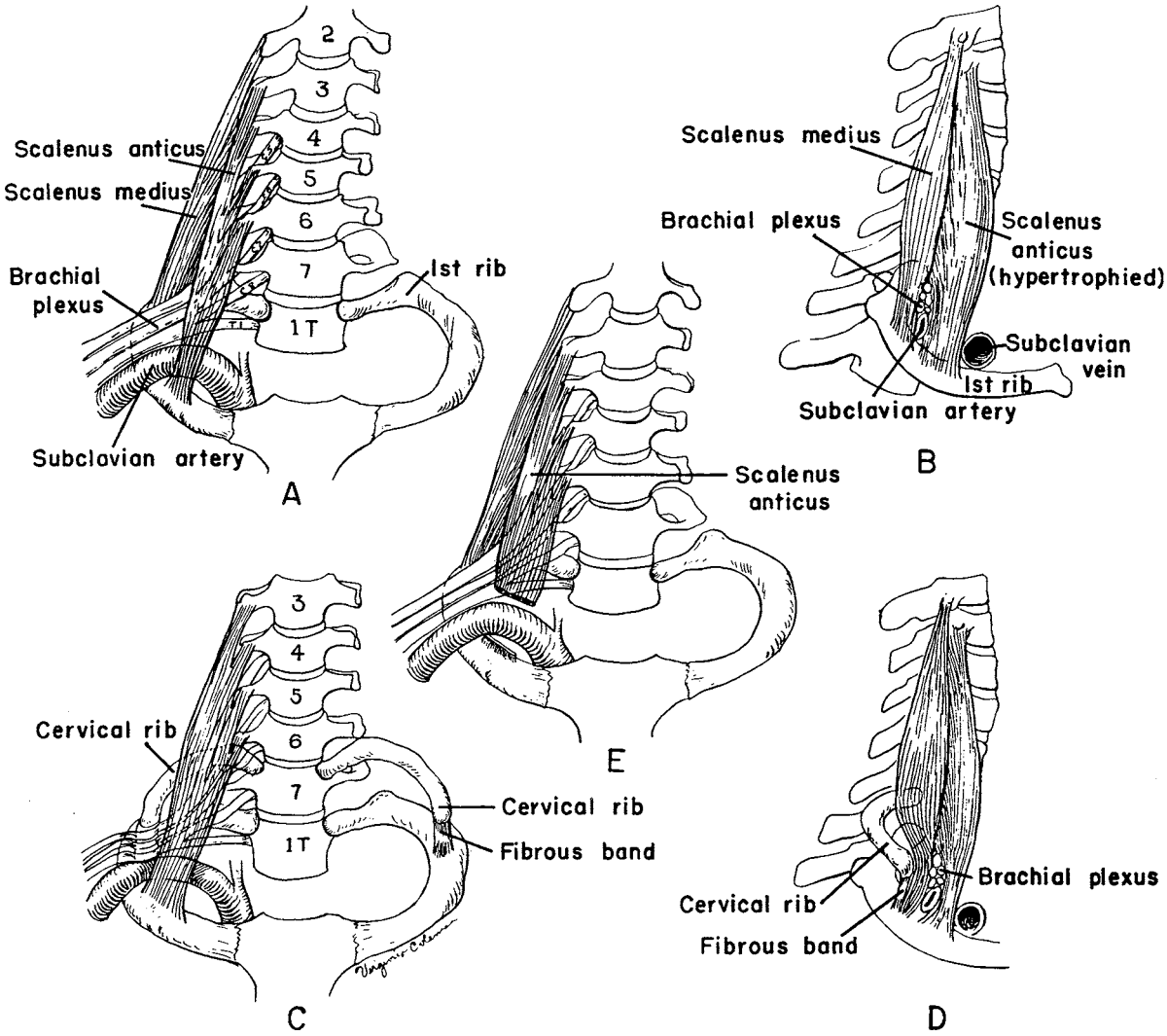

Figure 42-3 Anatomic relationships generating scalenus anticus syndrome in the presence and absence of a cervical rib. **(A)** Normal. **(B)** Hypertrophied anterior scalene muscle compresses the brachial plexus and the subclavian artery. **(C,D)** Compression of the brachial plexus by a cervical rib. **(E)** Relief of compression by scalenotomy. (Reprinted with permission from Yiannikas C, Walsh JC. Somatosensory evoked responses in the diagnosis of thoracic outlet syndrome. *J Neurol Neurosurg Psychiatry* 1983;46:234–240.)

believed to have costoclavicular syndrome are women with long necks and drooping shoulders. Symptomatology has been hypothesized to result from chronic stretching of the brachial plexus.

Investigations to confirm this more common but obscure form of thoracic outlet syndrome are usually unrewarding. Roentgenographs, EMG, nerve conduction studies, SSEPs, and arteriography are usually normal and of limited value.

Exercises to counteract and strengthen the drooping shoulders have been proposed as a possible treatment. First rib resection is often performed in patients with this more obscure but common form of thoracic outlet syndrome. Complete relief of symptoms has been reported in 55% to 85% of patients with partial relief in 7% to 35% of patients.[127]

Entrapment Syndromes

Suprascapular Nerve

The suprascapular nerve contains fibers from C5 and C6 and arises from the upper trunk of the brachial plexus. Coursing underneath the trapezius muscle, it passes onto the scapula through the suprascapular notch to the supraspinatus fossa of the scapula to supply the supraspinatus muscle (Fig. 42-4). A ligament overlies the suprascapular notch and transforms the notch into an actual foramen.[119] The nerve then passes around the spinoglenoid notch and ends in the infraspinous fossa to supply the infraspinatus muscle. Once again, a small ligament may be found at the spinoglenoid notch, which transforms the notch into a foramen.[1]

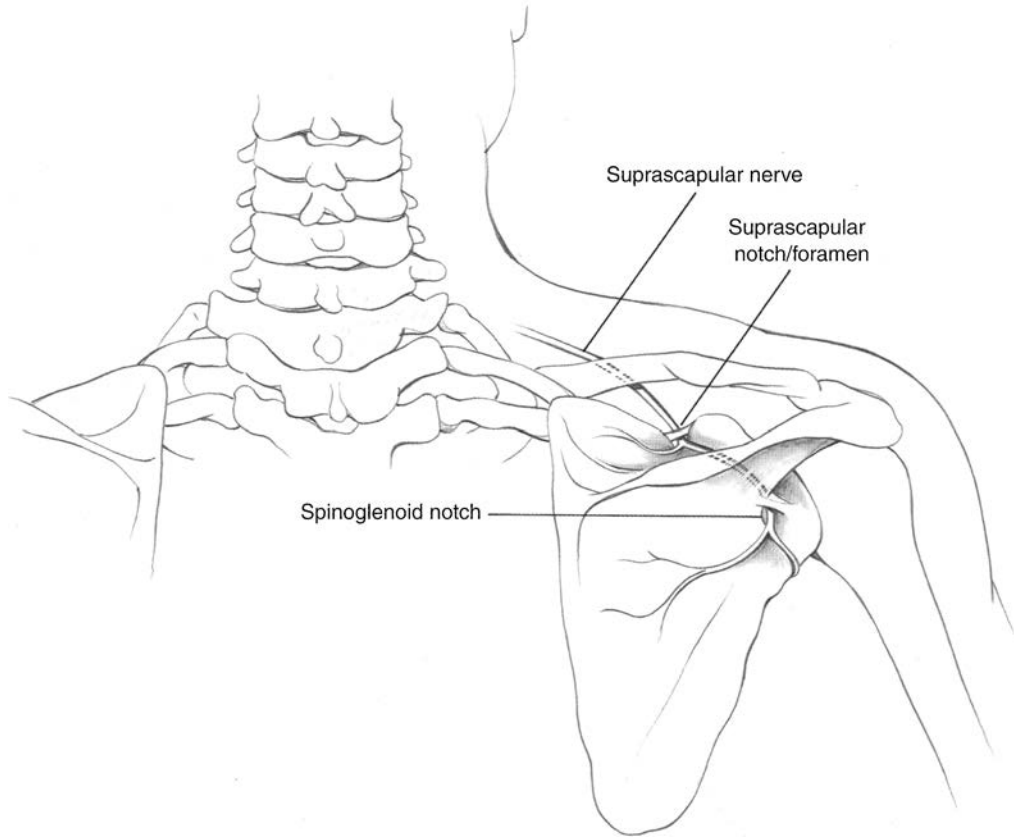

Figure 42-4 Suprascapular nerve entrapment. Posterior view of the left shoulder showing the course of the suprascapular nerve and potential areas of entrapment in the suprascapular notch/foramen and the spinoglenoid notch/foramen. (Reprinted with permission from Petrera JE, Trojaborg W. Conduction studies of the long thoracic nerve in serratus anterior palsy of different etiology. *Neurology* 1984;34:1033–1037.)

Selective wasting and weakness of the supraspinatus and infraspinatus muscles, rather than pain, are the usual presenting signs of suprascapular neuropathy.

Entrapment of the suprascapular nerve within the suprascapular notch or spinoglenoid notch occurs gradually over time. Entrapment of the nerve in either notch may occur from compression by a tight ligament **(61)**, although entrapment in the spinoglenoid notch by this mechanism is a rare event. **(62)** In the suprascapular notch, the presence of a bone spur also may cause entrapment. Fractures and shoulder injuries also may result in chronic compression of the suprascapular nerve in its notch. **(63)**

Treatment should be based on severity of the neuropathy. In the presence of a mild focal neuropathy, injections of local anesthetic and corticosteroid may be helpful **(64)**, otherwise surgical interventions may be indicated.

Long Thoracic Nerve

The long thoracic nerve arises from the ventral rami of C5–6 and sometimes C7. After leaving the brachial plexus, it travels down the internal aspect of the chest wall supplying the serratus anterior muscle.

Patients with a neuropathy of the long thoracic nerve complain of a dull ache around the shoulder girdle at rest or upon abduction of the arm. However, motor dysfunction of the serratus anterior and its sequelae are more prominent features. As the serratus anterior stabilizes the scapula against the chest wall, weakness and difficulty in using the shoulder are the primary symptoms of long thoracic neuropathy.

Because of the formation of the long thoracic nerve from the roots of C5–7 prior to the development of the trunks, long thoracic neuropathy will seldom occur in brachial plexus injuries. Long thoracic neuropathies most often occur in the face of blunt trauma to the shoulder or the anterolateral thoracic wall.[69] The long thoracic nerve also may be injured after a number of common surgeries, including thoracotomy and radical mastectomy.[59] Repetitive or extreme exertional shoulder movement also has been implicated in long thoracic neuropathy. Activity such as chopping wood, playing golf or tennis, and jumping rope have been described as potential factors in the genesis of the neuropathy.[111]

If the injury to the long thoracic nerve is due to excessive or repetitive use of the shoulder, cessation of activity usually results in full recovery. Physical therapy is the primary modality for management of long thoracic neuropathy

after acute blunt trauma or surgical injury. Unfortunately, prognosis is poor. Shoulder braces also may be of value.[160]

Axillary Nerve

The axillary nerve arises from the posterior cord of the brachial plexus. It lies lateral to the radial nerve and passes below the shoulder joint into the quadrilateral space. The boundaries of the quadrilateral space are the neck of the humerus, the long head of the triceps, the teres major muscles, and the teres minor muscles. The axillary nerve courses along the posterior and lateral surfaces of the humerus and divides into terminal branches supplying the deltoid muscle.

Wasting and weakness of the deltoid muscle, with marked impairment in shoulder abduction, are the chief clinical presentations of axillary neuropathy. As in other entrapment neuropathies, sensory abnormalities can be minimal in axillary nerve entrapment. Sensory loss may be present in a small area overlying the deltoid muscle near its insertion.[15]

Shoulder dislocation or fracture of the surgical neck of the humerus are the most common causes of axillary neuropathy.[17] The axillary nerve is usually not injured in isolation, and there may be concomitant trauma to the radial, suprascapular, or musculocutaneous nerves or the brachial plexus itself.[87] Other causes of injury to the axillary nerve include blunt trauma, intramuscular injections and bullet wounds.[135]

If traumatic damage to the axillary nerve is incomplete, no specific therapy is required. If the lesion is complete, some degree of recovery should be expected in several months. If recovery does not occur, surgical exploration and nerve grafting may be considered.

COMPLEX REGIONAL PAIN SYNDROME

To simplify CRPS and develop a rational diagnostic and therapeutic approach, the pain patterns in CRPS can be divided into two states: sympathetically maintained pain (SMP) and sympathetically independent pain (SIP).

Sympathetically Maintained Pain

This describes the pain that is dependent on and maintained by sympathetic input. Two particular features distinguish SMP: The pain is accompanied by signs of autonomic dysfunction and sympathetic blockade generally results in pain relief. Features usually include burning pain, hypersensitivity, allodynia, edema, and sometimes muscle spasms and dystonias.[8] The response to sympathetic blockade is generally good and was the reason behind the original disease nomenclature of reflex sympathetic dystrophy.[41]

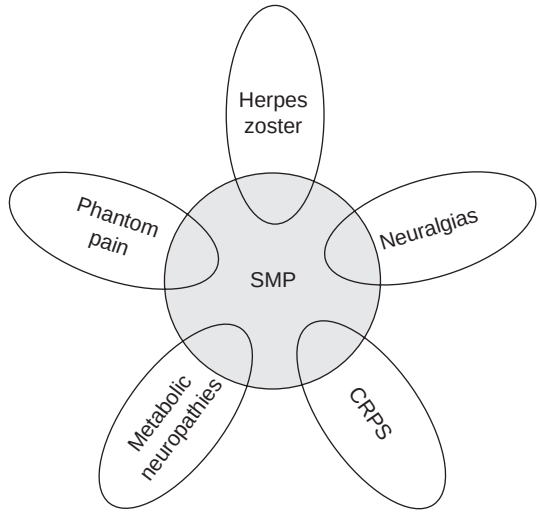

Figure 42-5 Relationship between sympathetically maintained pain and some selected painful conditions. This illustration is intended as a conceptual framework without indication of the quantitative relationship between the intersections. Varied pain disorders may have a component of sympathetically maintained.

It is not exactly known how the sympathetic blocks work, and unfortunately there are no well-controlled studies on the efficacy of the sympathetic blocks. A large number of uncontrolled studies, however, reveal excellent initial pain relief and long-term pain relief in greater than 50% of patients.[86–113] The goal of treatment in this condition is to break the cycle of the pain state while the patients are involved in progressive rehabilitation programs.[146] Occasionally, a single blockade can stop the process, especially if given early in the course of the disease. Many patients will have a progressively prolonged duration of symptomatic improvement following subsequent blockades. However, in some cases the effectiveness of the sympathetic blockade lasts for a short duration, and sometimes as the disease progresses, the sympathetic blocks become less effective. This may be due to decreased contribution of the sympathetic system to the painful state or may be secondary to development of fibrosis around the sympathetic ganglion shielding it from the injected local anesthetic. One must recognize that the term "sympathetically maintained pain" describes a mechanism by which pain occurs, and is not limited to CRPS. SMP occurs in a variety of other neuropathic pain conditions like diabetic neuropathy, ischemic peripheral vascular disease, postherpetic neuralgia, neuroma pain, and phantom limb pain[93] (Fig. 42-5).

Sympathetically Independent Pain

This describes the pain state that occurs most often in later stages of CRPS, where sympathetic blockade or sympathectomy yields no clinical improvement of the pain. The pain characteristics of this clinical subgroup suggest the involvement of various mechanisms.

Diagnosis of SMP

While diagnosis of CRPS is based on the clinical picture, determination of the sympathetic component of the pain can be made primarily by studying the effects of sympathetic blockade on the pain.

Complex Regional Pain Syndrome

CRPS is the new terminology proposed by the IASP to replace the old names (reflex sympathetic dystrophy [RSD], causalgia, etc.). The word "complex" describes varying clinical features of the condition. The *pain*, which is the sine qua none of the clinical entity, is usually *regional* in distribution. CRPS is further divided into CRPS types I and II, type I comprising the old RSD designation and type II encompassing the old causalgia as well as posttraumatic neuralgias. Unlike CRPS-I, CRPS-II occurs consequent to an injury to a specific nerve.[146] CRPS is not limited to adults; cases have been described in children. In adolescents it seems that girls are more affected than boys and the lower limbs more frequently affected than the upper limbs. These patients are usually high achievers and are active in sports.[9]

CRPS Type I

This is characterized by:

1. History of an initiating noxious event, however trivial
2. Ongoing pain or allodynia/hyperalgesia that is ***not*** limited to distribution of a single peripheral nerve, and is disproportionate to the inciting event
3. Evidence of edema, blood flow abnormalities, or abnormal sudomotor activity in the region of the pain
4. This diagnosis is excluded by the existence of conditions that would otherwise account for the degree of pain and dysfunction.

CRPS Type II

This is characterized by:

1. Developing after a nerve injury
2. Ongoing pain or allodynia/hyperalgesia occurring and not necessarily limited to the distribution of the injured nerve
3. Evidence of edema, skin blood flow abnormalities, or abnormal sudomotor activity in the region of pain
4. This diagnosis is excluded by the existence of conditions that would otherwise account for the degree of pain and dysfunction.

Clinical Presentation

Incidence

The epidemiologic data on the incidence of CRPS are sparse. Approximately 10% of patients referred to multidisciplinary pain clinics are diagnosed to have CRPS. SMP involves the upper limb twice as frequently as the lower limb. There is no clear sex prevalence for SMP. Several studies have reported that the incidence of causalgia following peripheral nerve injury varies between 2% and 14%.[119]

Predisposing Factors

Most patients with SMP present with a history of trauma to the soft tissue, bone, or elements of the nervous system that may be the result of accidental injury in the form of sprain, fracture, dislocation, crush, or blunt injury. Surgical or other iatrogenic injuries, or even vaccinations, have been reported to result in SMP. Additionally, SMP has been associated with neurologic diseases such as diabetic neuropathy, stroke, postherpetic neuralgia, and herniated disc lesions. The unanswered question remains: Why, following apparently identical types of injury, does only a small proportion of patients develop the pain and associated trophic changes while the majority of individuals do not? Recent studies suggest a possible genetic predisposition in individuals afflicted with CRPS.[91,163]

Clinical Features

The clinical signs and symptoms of CRPS-I are variable. The characteristic triad of signs and symptoms includes sensory abnormalities, autonomic dysfunction, and motor dysfunction. These signs and symptoms may be present in varying combinations and intensities depending on the severity and duration of the disease.

Sensory Abnormalities. Pain following a trauma that persists beyond the expected normal healing process is an early warning sign of CRPS- I. Spontaneous burning pain and/or pain to light mechanical stimuli are prominent signs of CRPS-I. Pain is typically not limited to the distribution of a specific peripheral nerve. These sensory abnormalities are most pronounced distally in the affected limb; however, symptoms of CRPS often extend beyond the involved extremity. Indeed, recent studies have shown hemisensory impairment, manifested as decreased temperature and pinprick sensation in the part of the body corresponding to the affected limb, in one-third of CRPS patients.[122] These patients, moreover, displayed increased frequency of allodynia and hyperalgesia, suggesting that in addition to peripheral up-regulation in alpha-adrenoceptors, central mechanisms enter into play in the pathophysiology of CRPS.

Autonomic Dysfunction. Altered skin temperature on the hyperalgesic region is often demonstrated in patients with CRPS-I.[16,169] Autonomic dysfunction can be also demonstrated by abnormal responsiveness to cold pressor test. Such abnormalities in the skin may be easily demonstrated by skin temperature measurement, or more accurately and quantitatively by thermographic imaging. Trophic changes such as local edema, abnormal hair and nail growth, and patchy osteoporosis may occur as a consequence of altered

microcirculation.[16,107] Because of their responsiveness to sympathetic blocks, these changes may be due to the hypersensitivity of affected organs to the sympathetic outflow.[12]

Motor Dysfunction. Not uncommonly, dystonia affecting movement in the distal extremity is noticed in patients with early CRPS-I. Muscle strength is reduced and may be lost. However, this is mostly due to disuse atrophy or limited by the pain state.[132] Of note, motor signs and symptoms were not included in the diagnostic criteria of the IASP due to their sporadic occurrence. The dystonia may be focal, multifocal, or diffuse and is notoriously resistant to treatment. A recent study evaluated the efficacy of intrathecal baclofen in CRPS-I patients with multifocal or generalized dystonias. Intrathecal baclofen was found to have a substantial therapeutic value in patients with CRPS-I, especially when the dystonia involves the upper extremities.[164]

The Shoulder in Complex Regional Pain Syndrome

CRPS commonly affects the shoulder and upper extremity. Many patients with sympathetically maintained pain localized in the hand or the wrist also have complaints of pain in the shoulder. Such coexistence of pain symptomatology is often called the shoulder–hand syndrome[148,149] or the shoulder–hand–finger syndrome.[103] Patients complain of pain, stiffness, and limited range of motion in the shoulder, with swelling and vasomotor disturbances in the hand. This limited range of shoulder motion may be passive or active because of pain. Frozen shoulder may occur in severe cases.[125,165]

Laboratory Tests/Diagnostic Procedures

Thermography. Infrared thermographic imaging using quantitative temperature difference has been used as a confirmatory test for CRPS-I (RSD). However, the quantitative temperature difference has inherent problems because skin temperature asymmetry may be present in neuropathic abnormalities, focal inflammation, or vascular disease. Recently, Gulevich et al.[55] used computer-generated side-to-side quantitative and qualitative temperature differences as well as functional autonomic response to cold water stress testing. The authors showed that stress infrared thermography is helpful in confirming the diagnosis of CRPS-I with 93% sensitivity and 89% specificity.

Three-Phase Bone Scan

CRPS-I (RSD) is characterized by pain, diminished function, joint stiffness, skin and soft tissue trophic changes, and vasomotor instability. Bone scanning has some role within the first year of onset of symptoms, and even then the sensitivity is only around 50%.[133,170] However, sometimes it could be of clinical use to help eliminate other differential diagnoses. Three-phase bone scanning usually shows hypervascularity in the affected extremity on early images followed by diffusely increased uptake in distal joints on delayed images.[77]

Quantitative Sensory Testing

Patients demonstrate that the mean threshold for pain to mechanical[137] and thermal stimuli[154] is dramatically decreased on the affected side. Even stimuli that are completely innocuous on normal skin, such as stroking with a camel's hair brush, applying a vibrating tuning fork to a bony prominence outside the hyperalgesic area, or moving a single hair follicle, can cause profound pain.

Response to Sympathetic Blockade

This is still a very important diagnostic test for CRPS, especially if pain is mediated through the sympathetic nervous system. Pain relief that outlasts the duration of the injected local anesthetic is an important diagnostic feature.[145]

Sympathetic blockade can be done either with a stellate ganglion block or using an intravenous regional block (IVRB). Other tests have been used to measure abnormalities of the sympathetic function including Doppler flowmetry,[13,131] infrared thermography,[20,55] sweat tests,[28] skin galvanic resistance tests,[31,153] and scintigraphic vascular[108] and bone scans.[77] Although these tests are indicative of sympathetic dysfunction, their specificity in the diagnosis of SMP has not been well documented.

Stellate Ganglion Block

This provides a more specific and definitive diagnosis of SMP depending on the result of a blockade of the sympathetic plexus that supplies the affected limb (e.g., the stellate ganglion when the upper limb is affected). Nonetheless, errors in the diagnosis of SMP can take place following sympathetic blockade with local anesthetics, and can be either false negative or false positive.

False-negative errors may result from technical failure in adequately blocking the sympathetic efferent fibers. Anatomic variability in sympathetic chain location and shielding of ganglia by adjacent fascial septae contribute to technical difficulties. Therefore, objective demonstration of increased skin temperature to 35°C in the distal extremity corresponding to the sympathetic ganglion blocked, in addition to other clinical signs of sympathetic blockade like Horner's sign or flushing of the skin of the limb, will verify that a proper sympathetic blockade has been achieved.[145]

T2 Sympathetic Blockade

False-negative results are particularly problematic with stellate ganglion blocks as sympathetic efferents from ganglia as caudal as T2 contribute to the innervation of the upper extremity.[12] False positives may occur when pain relief is due to somatic blockade. This can happen because sensory nerves could lie in close proximity to targeted sympathetic

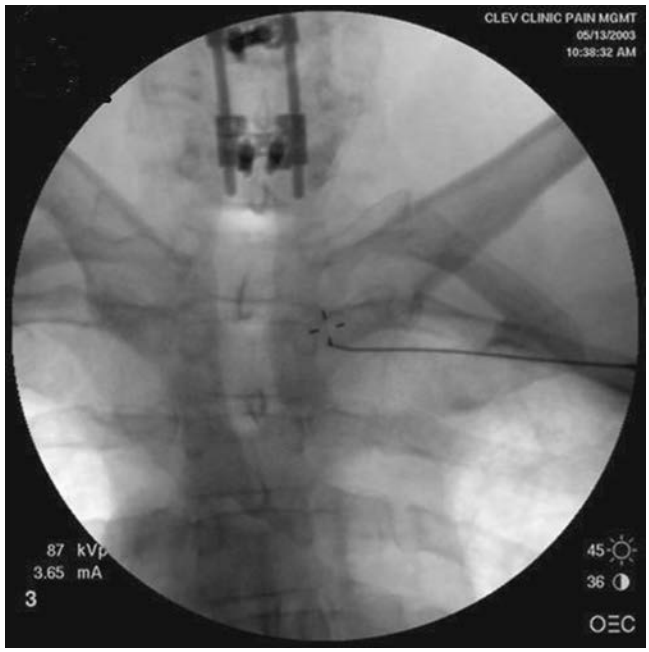

Figure 42-6 Anteroposterior view showing the final needle position at the costovertebral junction in T2 sympathetic block.

fibers at the level of the stellate ganglion. That is why T2 sympathetic blockade may be more selective (Fig. 42-6).

Attention should be paid to the diffusion pattern of the contrast agent to avoid tracking toward neighboring nerve roots. Also, sensory testing to verify that the affected area is not rendered hypoesthetic or anesthetic should be performed regularly.

Intravenous Regional Blocks

There are multiple reports on the analgesic effects of bretylium, guanethidine, reserpine, and others via the Bier block technique, and it has even been suggested as a diagnostic tool. However, there are no well-controlled studies supporting their effectiveness, and a recent meta-analysis looking at 21 such randomized clinical trials found insufficient numbers in most studies and important methodologic differences among the various trials, rendering conclusions difficult.[110]

A general pitfall to this technique is the possible confounding effect of an ischemic block of large-diameter afferent fibers. This can be differentiated by the dissimilar time course. Ischemic nerve block usually recovers within a few minutes, while sympathetic blocks due to sympathetic blockers last longer.

Moreover, false negatives with IVRB can occur in the presence of a sympathetic–afferent nociceptive interaction proximal to the tourniquet or by way of a sympathetic sudomotor (cholinergic) interaction with afferent nociceptors.[12]

Intravenous Phentolamine Test

It has been shown that the peripheral tissues express or upregulate alpha-adrenoceptors on the nociceptive primary afferent neurons at the site of injury. Activation of these nociceptors by the release of norepinephrine on previously inactive alpha-adrenoceptors results in pain.[37–32] Nociceptor activity leads to sensitization of the pain-signaling neurons (wide dynamic range neurons [WDR]). When sensitized, a minor input from the low threshold mechanoreceptors will induce pain. The systemic administration of the α-adrenergic blocking agent phentolamine has been suggested as an alternative test for the diagnosis of SMP. Intravenous phentolamine infusion has an advantage over the paravertebral sympathetic blockade in that it allows the injection of saline to test for placebo responders.[5,116] On the other hand, the drawbacks of IV phentolamine are the associated hypotension and tachycardia, which limit the benefit of this test in patients with coronary artery disease. These side effects can be avoided by pretreating the patients with IV fluids and beta-blockers if necessary. A major drawback of this test is that only a partial alpha-adrenoceptor blockade is achieved.[12]

Differential Diagnosis of CRPS

Although CRPS tends to present in an extremity in most cases, it may occur elsewhere in the body. Differential diagnoses include posttraumatic vasoconstriction from thrombophlebitis, arthritis, infection, soft tissue damage, tenosynovitis, fasciitis, fracture, and radiculopathy.

Management of CRPS

Physical Therapy and Rehabilitation

This is the fundamental step in the treatment of CRPS. Aggressive physical therapy and rehabilitation programs should be individually designed with the ultimate goal of regaining function of the affected extremity. Exercise includes various modalities and, more specifically, stress loading and increased endurance of the affected extremity.[145]

Sympathetic Blockade

The idea of the sympathetic blockade is to attempt to arrest the cycle of sympathetic hypersensitivity and to provide pain relief, which facilitates the physical therapy rehabilitation process. Various methods of sympathetic blockade can be used—stellate ganglion blocks, IV regional blocks, or repeated IV phentolamine infusion—if more than one extremity is involved or in patients where sympathetic blocks are contraindicated (e.g., anticoagulation).

Psychological Factors

Severe pain engenders emotional suffering and promotes behavioral changes that are subject to misinterpretation. The behavioral response to CRPS ranges from fully preserved function to complete invalidism. Such behavioral responses may be especially important in CRPS because of disuse, overprotection, and immobilization of the affected limb. These may lead to exacerbation of edema, vasomotor

changes, and demineralization associated with CRPS. Furthermore, major psychiatric illnesses could both exacerbate and reduce the ability to cope with CRPS-associated pain. For example, depression occurs in CRPS as in other chronic pain syndromes, and it also exacerbates the overall suffering. For these reasons, it is very important to address the psychological issues/psychiatric illnesses/personality disorders in an individualized basis and as a part of multidisciplinary program to treat CRPS.[50]

Adjuvant Medications

Pharmacologic treatment of neuropathic pain, including CRPS, is notoriously difficult, and there are very few well-designed trials addressing this subject. Numerous medications including tricyclic antidepressants, alpha-blockers, calcium channel blockers, membrane stabilizers, and alpha-2 agonists have been used to treat CRPS.[145] The efficacy of such agents still needs to be determined in case-controlled studies.

Continuous Infusion of Epidural Opioids and Local Anesthetics through a Tunneled Cervical Epidural Catheter

This modality has shown good outcomes in patients with CRPS, with acceptable rates of complications/side effects over periods of weeks to months. Favorable results were mainly noticeable when this treatment modality was initiated within 1 year of the onset of symptoms.[21,104,145] When contemplating surgery on an affected shoulder, it is imperative to implant a cervical epidural catheter to provide surgical anesthesia and maintain postoperative epidural analgesia for a variable period of time depending on the extent of the procedure and the severity of the illness, lest marked exacerbation of CRPS will occur (Fig. 42-7).

Peripheral Nerve Stimulation

Recently, peripheral nerve stimulation (PNS) has been used in the treatment of severe, intractable CRPS-II or causalgia. Criteria for patient selection for PNS include patients with severe intractable symptoms that are entirely or mainly in the distribution of one major peripheral nerve. Significant decreases in spontaneous pain as well as allodynia were noted up to 4 years following PNS placement, with 20% of patients returning to part-time or full-time employment.[57]

Spinal Cord Stimulation

Recent studies have shown that spinal cord stimulation (SCS) has a proven value in the management of refractory CRPS[72–121] (Fig. 42-8). It is worth noting that these patients had failed all other modalities of management. Improvements were noted in visual analog pain scales and perception of pain, in daily living and quality of life, and in a substantial decrease in analgesic consumption. The SCS-induced pain relief in CRPS is independent of sympathetic or vasodilatory effects.[73] No clinically relevant progress occurred in trophic

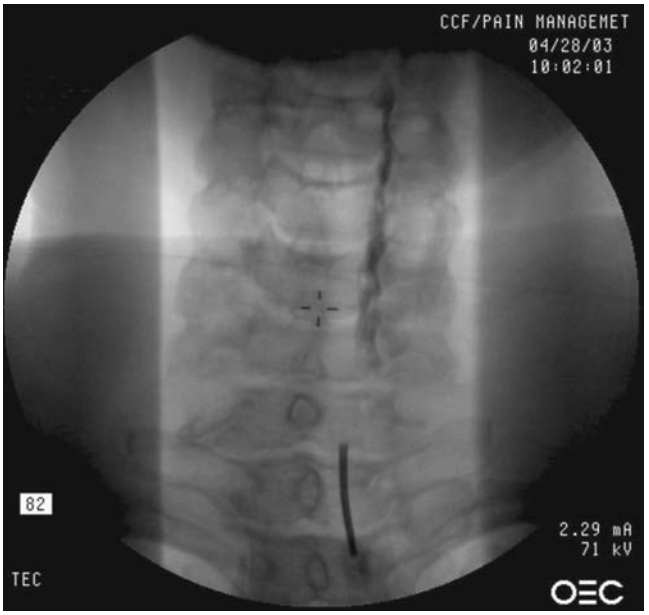

Figure 42-7 Anteroposterior view showing tunneled cervical epidural catheter with right-sided spread of the contrast agent.

alterations such as musculoskeletal changes or in the functional status of patients, but there was a clear propensity to return to work and productivity in implanted patients.[74–121]

PHARMACOLOGIC MANAGEMENT OF CHRONIC SHOULDER PAIN

Chronic pain is more of a treatment challenge; the pathogenesis may be unclear, with less opportunity to predict the course of recovery. The goal of pain management should include reconditioning, reducing pain, and improving function, sleep, and mood.

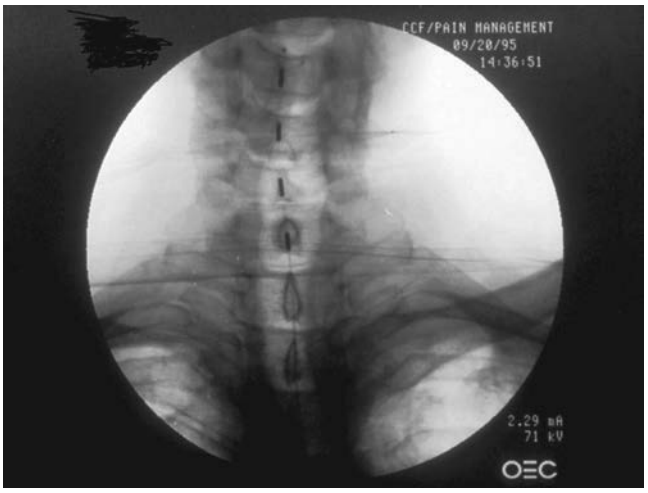

Figure 42-8 Anteroposterior view showing cervical spinal cord stimulation lead.

A multidisciplinary, comprehensive treatment plan is optimal, including:

- Individual psychosocial counseling in conjunction with patient/family education
- Noninvasive or minimally invasive procedures, such as massage therapy, physical therapy, transdermal or transcutaneous electrical nerve stimulation (TENS), or acupuncture
- Up-to-date pharmacologic and/or anesthetic therapies
- If necessary, surgical intervention and physical medicine and rehabilitation focused to enhance the patient's functional status[10,94]

Pharmacotherapy plays a key role in the management of chronic pain, as drug therapy may help to turn off noxious stimuli or dampen the underlying neuropathic disturbance.[4]

Nonopioid analgesics, which include acetaminophen, NSAIDs, and salicylate, provide first-line therapy for managing chronic pain and are classified as step 1 of the World Health Organization's (WHO) cancer-pain management ladder, which also has been applied to other, nonmalignant, chronic painful conditions.[49]

Acetaminophen

Acetaminophen inhibits prostaglandin synthetase in the central nervous system (CNS). Its action may be mediated through a central cyclooxygenase (COX)-2 mechanism, and also COX-3 has been proposed as a potential site of action. It has no or minimal effect on inhibition of peripheral prostaglandin synthesis, which accounts for the lack of antiinflammatory.[10,49]

Metabolism occurs in the liver, primarily by cytochrome P-450 (CYP-450), 1A2, 3A4, and 2E1.[10]

Acetaminophen has an excellent safety profile with regard to the gastroduodenal mucosa, platelet function, and nephrotoxicity but may cause hepatotoxicity at chronic doses higher than 6 g/day. Acute intoxication at a dose higher than 15 g/day can cause fatal hepatic necrosis.[4]

Prolonged use of acetaminophen in patients with severe liver disease and chronic alcoholism is not recommended. No more than 3 to 4 g of acetaminophen daily is currently recommended.[10]

Acetaminophen and NSAIDs have a ceiling dose effect for analgesia; beyond the recommended doses, no further pain relief occurs.[4]

Nonsteroidal Antiinflammatory Drugs

NSAIDs are among the most commonly used medications in the world. They constitute 4% of all filled prescriptions at a yearly cost in excess of $2 billion.[18,128] They are commonly used in the treatment of mild to moderate pain.[85]

Pharmacologic effects of NSAIDs include analgesia, antiinflammation, antipyresis, sodium retention and hyponatremia, the development of renal failure, vascular tone changes, gastric irritation, platelet inhibition, hepatic dysfunction, and CNS effect such as dizziness, sedation, and confusion.[4]

The ability of NSAIDs to exert analgesic and antiinflammatory effects is mediated by two mechanisms. The first is through suppression of proinflammatory and pain-enhancing prostaglandin synthesis at the site of inflammation. The second mechanism is through the modulation of neutrophil intracellular signaling function, which decreases the numbers of neutrophils migrating to inflammatory sites, resulting in a down-regulation of the release of free radicals and destructive enzymes at these sites.[128]

Two isoforms of COX enzyme exists: COX-1 and COX-2. COX-1 is found in most times throughout the body. It is expressed constitutively in both the gastroduodenal mucosa and platelet, and inhibition of COX-1 at these sites may predispose the patient to gastroduodenal ulceration and bleeding, respectively.

COX-2 is usually undetectable in most tissues but is expressed in response to inflammatory stimuli.

COX-1 and COX-2 are also expressed constitutively in the kidney, and inhibition of one or both of these isoforms may predispose the patient to renal adverse events, including renal insufficiency and hypertension. Traditional NSAIDs are nonselective inhibitors of both COX isoforms.[62,85]

Pharmacologic Classifications of NSAIDs

Carboxylic acids
Salicylic acids
Acetylsalicylic acid (aspirin)
Nonacetylated salicylates
  Choline magnesium trisalicylate (Trilisate)
  Salicyl salicylate (Disalcid)
  Diflunisal (Dolobid)
Acetic acids
Indole acetic acids
  Indomethacin (Indocin)
  Sulindac (Clinoril)
  Etodolac (Lodine)
Pyrrole acetic acids
  Tolmetin (Tolectin)
  Ketorolac (Toradol)
Phenyl acetic acids
  Diclofenac (Voltaren)
Naphthyl acetic acid
  Nabumetone (Relafen)
Propionic acids
Phenyl propionic acids
  Ibuprofen (Motrin)
  Fenoprofen (Nalfon)

Flurbiprofen (Ansaid)
Ketoprofen (Orudis)
Naphthyl propionic acids
Naproxen (Naprosyn, Anaprox)
Anthranilic acids
Fenamates
Meclofenamic acid (Meclomen)
Mefenamic acid (Ponstel)
Oxicams
Piroxicam (Feldene)
Pyrazoles
Phenylbutazone (Butazolidin)

The salicylate group of NSAIDs includes aspirin, choline magnesium trisalicylate, and diflunisal. Aspirin is commonly used not only as an antiinflammatory agent, but also for platelet-inhibiting effects in prevention of cerebrovascular accident and myocardial infarction.[4]

Aspirin produces irreversible inhibition of platelets. This inhibition is near complete and is sustained for at least 48 hours after a single dose.[76] A recent study suggests that concurrent treatment with ibuprofen may limit cardioprotective effects of aspirin by inhibiting aspirin's prolonged effect on platelet aggregation.[85]

Common side effects of aspirin include nausea and emesis, gastrointestinal (GI) hemorrhage, peptic ulcer, gastritis, and liver function abnormalities. Aspirin is the most nephrotoxic of the NSAIDs and has been associated with Rye's syndrome in children and adolescents. Prolonged use of high doses (more than 100 mg/kg/day) may result in chronic salicylate toxicity.[4]

Indole Acetic Acid Derivatives

Indomethacin, sulindac, and etodolac are indoleacetic acid derivatives.

GI side effects are quite common with indomethacin. Other adverse effects include psychosis, headache production, depression, hypertension, and fluid retention. Sulindac produces fewer side effects than indomethacin; however, a causal relationship to liver disease has been suggested. Etodolac is a mixed COX-1 and COX-2 inhibiting activity and therefore seems to have less toxicity.[4]

Pyrrol Acetic Acid Derivatives

Ketorolac is the only NSAID that is available as an injection in the United States. It is effective against pain of somatic origin. Concurrent use with opioids allows reduced dosing of opioids because it promotes release of endogenous opioids. Analgesic action of ketorolac is through the CNS. It has GI and renal toxicity. To avoid such toxicity, short-term use (less than 5 days orally and less than 48 hours parenterally) is recommended.[4,36]

Phenyl Acetic Acid Derivatives

Diclofenac (Voltaren) is used in the management of gout, low back and neck pain associated with degenerative joint disease, osteoarthritis, and rheumatoid arthritis. It has GI side effects, and there is a small risk of hepatic inflammation, so liver function tests must be done within 8 weeks of administration.[4]

Propionic Acid Derivatives

Ibuprofen, ketoprofen, and naproxen all have GI side effect. Naproxen is recognized as having more GI side effects than ibuprofen.[4]

COX-2 Selective Inhibitors

Celecoxib (Celebrex), valdecoxib (Bextra), and rofecoxib (Vioxx) were the first of a long line of COX-2 inhibitors to be available in the United States. These drugs have 200- to 300-fold selectivity for inhibition of COX-2 over COX-1.[18,85]

Celecoxib is the only COX-2 inhibitor available now in the United States, as rofecoxib (Vioxx) and valdecoxib (Bextra) were withdrawn from the market.

They are comparable to the traditional NSAIDs in their analgesic effects but have marked reduction in the gastrointestinal side effects.[62,85]

Two large outcome studies—the Vioxx Gastrointestinal Outcomes Research (VIGOR) trial[18] and the Celecoxib Long-term Arthritis Safety Study (CLASS)[138]—suggest that COX-2 selective inhibitors should be used whenever NSAIDs are indicated in patients with osteoarthritis or rheumatoid arthritis. The COX-2 selective inhibitors have comparable efficacy to nonselective NSAIDs in these patients.

In patient not taking low-dose aspirin, the risk of confirmed upper FI events including symptomatic ulcers and the risk of confirmed complicated upper GI events is significantly reduced in patients taking COX-2 selective inhibitors compared with those taking nonselective NSAIDs, particularly ibuprofen and naproxen.[138]

Caution needs to be exercised, however, in the use of COX-2 selective inhibitors in patients with uncontrolled hypertension, mild to moderate renal insufficiency, or congestive heart failure, as the renal adverse effects of COX-2 selective inhibitors are similar to those that occur with nonselective NSAIDs.

COX-2 Inhibitors and the Cardiovascular System. COX-2 selective inhibitors may increase the risk of serious cardiovascular thrombotic events, especially myocardial infarction, as these agents do not inhibit platelet aggregation.

In the VIGOR trial, the incidence of cardiovascular events was higher in patients receiving rofecoxib than in those receiving naproxen. However, in the CLASS trial, there was no increase in the incidence of cardiovascular events associated with celecoxib compared to ibuprofen and diclofenac.[105]

Recently, a large trial (not published yet) showed a twofold increase in acute myocardial infarction with rofecoxib compared to placebo. This prompted the voluntary withdrawal of rofecoxib (Vioxx) from the U.S. and worldwide markets in September 2004.

In April 2004, the U.S. Food and Drug Administration (FDA) requested Pfizer, Inc. to voluntarily withdraw Bextra (valdecoxib) from the market. This request was based on the lack of adequate data on the cardiovascular safety of long-term use of Bextra, increased risk of adverse cardiovascular events in short-term coronary artery bypass surgery (CABG) trials, and reports of serious and potentially life-threatening skin reactions (go to http://www.fda.gov/medwatch/SAFETY/2005/safety05.htm#Bextra for more information).

The FDA has also asked manufacturers of all prescription NSAIDs, including Celebrex (celecoxib), a COX-2 selective NSAID, to revise the labeling to include a boxed warning to highlight the potential cardiovascular and GI risks (go to http://www.fda.gov/medwatch/SAFETY/2004/safety04.htm#vioxx for more information).

NSAIDs and Gastrointestinal Complication

In patients taking NSAIDs, the clinical ulcer rate is in the range of 1% to 4% and a complicated ulcer rate is around 1%.[58] Increased risk of GI complication is dose related, and there is evidence of an interaction with concomitant glucocorticoid and acetaminophen therapy.[62] Dyspepsia is the most common side effect of NSAID therapy. It occurs in around 30% of chronic users and leads to cessation of therapy, switching therapy, and time-consuming and expensive investigations such as endoscopy.[33]

Risk factors for serious upper GI complications in patients treated with nonselective NSAIDs include older age, male sex, history of peptic ulcer disease or prior upper GI bleeding, concomitant use of oral glucocorticoids or anticoagulants, and, possibly, smoking and alcohol consumption. Ibuprofen has the lowest risk of gastrotoxicity among nonselective NSAIDs.[61,62]

Celecoxib at dosages greater than those indicated clinically was associated with a lower incidence of symptomatic ulcers and ulcer complications, compared with NSAIDs at standard dosages.

Upper GI toxicity was strongest among patients not taking aspirin concomitantly. It is also better tolerated than NSAIDs.[138]

NSAIDs and Renal Complication

Intake of nonselective NSAIDs is associated with about a twofold increase in risk of developing renal failure, the increase being dose related. Risk factors for acute renal failure in patients treated with nonselective NSAIDs include age greater than 65 years; presence of intrinsic renal disease, usually defined as a serum creatinine greater than 2.0 mg/dL; hypertension and/or congestive heart failure; and concomitant use of diuretics and angiotensin-converting enzyme inhibitors.[62]

Edema and salt and water retention are the most common adverse renal effects associated with NSAIDs, occurring in 2% to 5% of patients. Other rare renal effects include hyperkalemia, allergic interstitial nephritis, chronic interstitial nephritis, and nephrotic syndrome.[3]

Celecoxib appears to be associated with less renal toxicity compared with NSAIDs, and it does not increase the risk of cardiovascular thromboembolic events.[139]

NSAIDs and Hypertension

NSAIDs, with the possible exception of sulindac and aspirin, may, at least in the short term, increase blood pressure. Changes in mean arterial pressure are small, in the order of 3 to 5 mm Hg. The precise mechanism by which NSAIDs raise blood pressure remains obscure, but there is evidence for both an effect on the vascular tone, through decreased angiotensin-dependent prostaglandin release, and volume control (sodium retention).[34]

Hypertension and cardiovascular risk with the use of NSAIDs are more pronounced in elderly people and patients with preexisting hypertension. Among the NSAIDs, indomethacin, fenoprofen, and phenylbutazone are most strongly associated, while sulindac, aspirin, and ibuprofen are the least likely to be associated with this effect.[34]

Antiepileptics

Antiepileptic drugs have been used for many years, since the 1960s, in the treatment of neuropathic pain.[56,85] The older agents include phenytoin, carbamazepine, and valproic acid. The newer agents include gabapentin, lamotrigine, topiramate, zonisamide, and oxcarbazepine. The newer agents are more often used in the management of chronic pain than the older agents, because of lack of organ toxicity and lesser need to monitor therapy with blood tests.[49,56,85]

Possible mechanisms of actions are enhanced gamma-aminobutyric acid inhibition (valproate, clonazepam) or a stabilizing effect on neuronal cell membranes. A third possibility is action via *N*-methyl-D-aspartate (NMDA) receptor sites. By preventing bursts of action potentials, these drugs can eliminate the severe lancinating pain of trigeminal neuralgia and other neuropathic syndromes. The most common adverse effects are impaired mental and motor function, which may limit clinical use, particularly in the elderly.[56]

- **Drugs that block voltage-dependent sodium channels**
  Phenytoin
  Carbamazepine
  Oxcarbazepine
  Lamotrigine
  Zonisamide
- **Drugs that affect GABA metabolism**
  Tiagabine
  Vigabatrin
- **Drugs that affect calcium currents**
  Ethosuximide

- **Drugs with multiple mechanisms of actions**
  Topiramate
  Valproate
  Felbamate
  Pregabalin
- **Drugs with unknown mechanism of action**
  Gabapentin
  Levetiracetam

Phenytoin (Dilantin)

Phenytoin has been used to treat a number of pain syndromes, including postherpetic neuralgia, diabetic neuropathy, and complex regional pain syndrome, and it was the first drug to be used for trigeminal neuralgia. Phenytoin blocks Na^+ channels and reduces neuronal excitability of pain fibers mainly by this mechanism.[68]

Long-term treatment requires blood tests, and side effects include gingival hyperplasia, facial hair growth, rare cases of Steven-Johnson syndrome, and even peripheral neuropathy.[85]

Because of side effects, drugs such as carbamazepine, oxcarbazepine, and lamotrigine have replaced phenytoin, but phenytoin's availability for intravenous infusion makes it suitable for breaking an acute attack of neuropathic pain.[68]

Carbamazepine (Tegretol)

Carbamazepine is considered a drug of choice for the treatment of trigeminal neuralgia. It is also effective in migraine prophylaxis, postherpetic neuralgia, and painful diabetic neuropathy,[85] although the newer antiepileptic drugs show considerable promise in the management of cluster headache and trigeminal neuralgia.[159]

The effect of carbamazepine on pain suppression is probably mediated via central and peripheral mechanisms. It decreases sodium and potassium conductance and suppresses the spontaneous activity of A-delta and C fibers responsible for pain without affecting normal nerve conduction. Dosage of carbamazepine ranges from 300 to 2,400 mg/day in divided doses.[159]

Side effects of carbamazepine include somnolence, dizziness, and gait disturbances. It is also associated with chronic diarrhea, syndrome of inappropriate secretion of antidiabetic hormone (SIADH) and hyponatremia, rash, and, rarely, aplastic anemia, thrombocytopenia, cardiac arrhythmia, and hepatocellular jaundice. Complete blood count (CBC) and liver function tests should be monitored.[85,159]

Oxcarbazepine (Trileptal)

Oxcarbazepine is chemically similar to carbamazepine, has Na^+ channel-blocking actions, and is used in the treatment of partial seizures. It may be better tolerated than carbamazepine; however, the incidence of hyponatremia may be higher.

It has demonstrated efficacy in trigeminal neuralgia, and studies are under way to examine its role in other neuropathic pain syndromes.[68,85]

Lamotrigine (Lamictal)

Lamotrigine is a phenyltriazine derivative and was initially approved for treatment of partial complex seizures. It blocks voltage-dependent Na^+ channels with the inhibition of glutamate release.

Lamotrigine, 50 to 400 mg/day, has demonstrated efficacy in relieving pain in patients with trigeminal neuralgia refractory to other treatments, painful diabetic neuropathy, and pain related to multiple sclerosis.[159]

Adverse effects include dizziness, constipation, nausea, somnolence, diplopia, and skin rash.[159]

Oral lamotrigine, 20 mg/daily, is a well-tolerated and moderately effective treatment for central poststroke pain (CPSP).[166]

Tiagabine

Tiagabine is a nipecotic acid derivative that enhances GABA-mediated inhibitory neurotransmission. This antiepileptic drug inhibits presynaptic neuronal and glial GABA uptake by binding reversibly and saturably with GABA uptake carriers GAT-1 and GAT-3.[159]

Its antinociception effect appears to be mediated by GABAb receptors since it can be completely antagonized by pretreatment with selective GABAb receptor antagonists.[159] Tiagabine also has Na^+ channel-blocking action.[68]

Tiagabine, indicated as adjunctive therapy for treatment of complex partial seizures, is also being tried in a variety of neuropathic pain syndromes. Maintenance doses range from 32 to 56 mg/day in divided doses.[85]

Topiramate (Topamax)

Topiramate is a sulfamate-substituted derivative of D-fructose. It has several mechanisms of action: It blocks Na^+ channels-enhanced GABA activity, diminishes NMDA-mediated excitation, and blocks voltage gated Ca^{70+} channels.[10,68] Absorption is rapid with bioavailability of about 80%. Metabolism is minimal, with 70% of a dose recovered unchanged in the urine. Elimination half-life is 18 to 23 hours.[10]

In a randomized, double-blind, placebo-controlled trial using oral topiramate, 200 mg twice a day for weeks, a statistically significant reduction in average pain scores was reported.[159]

Side effects of topiramate include paresthesia, anorexia, and renal calculi. CNS-related side effects are dose related and generally not observed at doses less than 250 mg/day.

Cognitive motor slowing and speech or work difficulty are seen only in a minimal percentage of patients when high doses are initiated.[10,159]

Valproic Acid

Valproic acid will prolong repolarization of voltage-activated Na^+ channels and inhibition of sustained neuronal firing. It also increases the amount of GABA in the brain.[159]

Valproic acid is used in the prophylactic treatment of migraine headache. It is associated with weight gain, tremor, and alopecia.[56]

Pregabalin

Like gabapentin, pregabalin is a GABA analog without proven agonistic effect on GABA receptors. Pregabalin does not appear to interact directly with Na^+ channels, Ca^{70+}channels, or neurotransmitter responses (GABA, glutamate). A randomized controlled trial comparing three different doses of pregabalin (75, 300, and 600 mg/day orally) with placebo in 337 patients with diabetic neuropathy reported significant improvement in pain, sleep, and clinical and global impression of change scores for those who took 300 mg or more daily.[159] Dizziness, somnolence, and peripheral edema were the most frequent adverse effects reported in this trial.[159]

Felbamate

Felbamate is a dicarbamate that has Na^+ channel-blocking action, inhibits NMDA-evoked potential, and enhances GABA-evoked responses in hippocampal neurons. Clinical experience with felbamate in neuropathic pain is limited to a few case reports.[159]

Given its potential for fatal toxicity of the bone marrow and the liver, the use of felbamate is restricted to some patients with refractory epilepsy. Its use in treatment of pain conditions is not warranted given the existence of many other alternatives.[159]

Gabapentin (Neurontin)

Of the new generation of antiepileptic drugs used for treatment of neuropathic pain, gabapentin is perhaps the best agent studied so far. Gabapentin has an effect on alpha-2-delta types of calcium channels and acts as an antagonism of NMDA receptors. It has no direct GABAergic action and it does not affect GABA uptake or metabolism.[124,159]

Two large randomized clinical trials have established the efficacy of gabapentin for relief of neuropathic pain in patients with postherpetic neuralgia and painful diabetic neuropathy.[7,126]

Gabapentin has been used in different pain syndromes, including trigeminal neuralgia and painful tonic spasms associated with multiple sclerosis, reflex sympathetic dystrophy, painful HIV-related peripheral neuropathy, and neuropathic cancer pain, postpoliomyelitis pain, central dysesthetic pain following spinal cord injury and erythromelalgia, and headache syndromes.[124,159]

Gabapentin is not metabolized in humans and is eliminated unchanged in the urine. Renal impairment will consequently decrease gabapentin elimination in a linear fashion with a good correlation with creatinine clearance. Gabapentin is removed by hemodialysis, so patients in renal failure should receive their maintenance dose of gabapentin after each treatment. The recommended starting dose in the treatment of neuropathic pain is 300 mg three times a day with titration if necessary to a maximum of 3,600 to 4,200 mg as tolerated.[124]

Gabapentin is as efficacious at treating neuropathic pain with no significant difference in minor adverse effects as other anticonvulsants and antidepressants. A prospective open label trial reported that gabapentin was safe at doses of 26 to 78 mg/kg in 52 children and adolescents. An 8-week trial at doses of 1,800 to 2,400 mg daily should be used before treatment can be deemed a failure.[56]

The most common side effects are somnolence (20%), dizziness (18%), ataxia (13%), and fatigue. The most serious adverse effect is convulsion (0.9%) and Steven-Johnson syndrome.[124]

Opioids

The opioid analgesics interact with three opioid receptor types (μ, δ, and κ). These opioid receptors belong to the "G" protein–coupled receptor family and they signal via a second messenger (cyclic adenosine monophosphate) or an ion channel (K^+).[65]

The μ (mu) receptor mediates the analgesic, which is closely associated with the μ_1 subset of μ-receptors and adverse effects of morphine including supraspinal analgesia, respiratory depression, euphoria, and increased sedation, which are associated with the μ_2 receptors.[49,65] μ-Opioid receptors are found in the periphery (following inflammation), at pre- and postsynaptic sites in the spinal cord dorsal horn (laminae I–II), and in the brainstem, thalamus, and cortex, in what constitutes the ascending pain transmission system. In addition, μ-opioid receptors are found in the midbrain periaqueductal gray, the nucleus raphe magnus, and the rostral ventral medulla where they comprise a descending inhibitory system that modulates spinal cord pain transmission.[65]

δ-Opioid receptors have been found in cerebral and cerebellar cortex, hippocampus, thalamus and hypothalamus, brainstems, and medullary and spinal cord dorsal horns (particularly in laminae I–II).[92]

κ-Opioid receptor agonism produces effective spinal analgesia but is associated with miosis and significantly more sedation than is μ-receptor agonism.[49]

Drugs with high σ (sigma) receptor affinity cause dysphoria, psychomotor stimulation, and hallucination.

There exists a general overlap between distribution pattern of NMDA and opioid receptors within the CNS. Among all three types of opioid receptors (μ, δ, and κ), κ-opioids may have a unique role in antagonizing NMDA receptor–mediated electrophysiologic events by directly interacting with the NMDA receptor per se.[92]

At a cellular level, opioids decrease Ca^{70+} ion entry, resulting in a decrease in presynaptic neurotransmitter release (e.g., substance P release from primary afferents in the spinal cord dorsal horn), enhance potassium ion efflux resulting in the hyperpolarization of postsynaptic neurons and a decrease in synaptic transmission, and inhibit GABAergic transmission in a local circuit.[65]

Endogenous classic opioid peptides include the enkephalins, endorphins, and dystrophins and appear to function as neurotransmitters, neuromodulators, and, in some cases, neurohormones.[65]

Indications

There is strong consensus that opioids should be used aggressively when needed to relieve severe acute pain and pain associated with terminal cancer.[112] Severe pain with a clear diagnostic basis, supportive objective findings, and responsiveness to opioids deserve a trial of opioid therapy if other therapies are ineffective.[92]

Route of Administration

Opioids may be administered by oral, transmucosal, rectal, intravenous, subcutaneous, transdermal, and intraspinal routes. For the management of chronic pain, the oral and transdermal routes are generally preferred because they are readily available and less invasive than other routes and most often can provide satisfactory analgesia even when high doses are required. When these routes are not reasonable, parenteral routes or intraspinal may be indicated.[130]

Opioid Drug Choices

Opioids are classified according to their pharmacologic actions with the receptors as agonists, agonists–antagonists, or antagonists.

Factors to consider in selecting opioid analgesics are relative affinities to the different opioid receptors, pharmacokinetic characteristics that influence onset and duration of action, and whether the opioids are weak or strong with respect to analgesic properties. Morphine is the opioid analgesic of choice because it is relatively inexpensive, is available in a wide range of dosage forms, and is the most extensively studied and used opioid.[49]

Morphine

Morphine is generally considered the prototype of all opioids. Morphine is available for oral, parenteral, rectal, and intraspinal use. Morphine's oral bioavailability varies from 35% to 75%.[65,85]

Morphine's plasma half-life (2 to 3.5 hours) is somewhat shorter than its duration of analgesia (4 to 6 hours), which limits accumulations during repetitive administration. Its pharmacokinetics remain linear and there does not appear to be autoinduction of biotransformation even following large chronic doses.[65] Morphine accumulates in the kidneys, liver, and skeletal muscle.[85] The drug is metabolized via the hepatic system by phase II process to glucuronide metabolites (55%), which are then renally excreted.[75,135] Morphine-6-glucuronide (M-6-G) is an active metabolite of morphine (about 5% to 15%) and is a more potent μ-receptor agonist than morphine. M-6-G is eliminated by the kidneys and will accumulate relative to morphine in patients with renal insufficiency.[65] Morphine-3-glucuronide (M-3-G), the predominant metabolite of morphine (about 50%) in humans, is devoid of opioid activity but may be responsible for the neuroexcitatory effects sometimes seen with large chronic morphine dosing.[65]

Patients should be initially titrated on immediate-release morphine and once stabilized converted to the delayed release. To manage acute "breakthrough" pain, "rescue" medication (immediate-release morphine) should be made available to the patient receiving delayed-release preparation.

Morphine is the preferred drug for the management of moderate to severe chronic cancer pain. The best route of administration is by mouth, because it is simple, safe, convenient, inexpensive, and effective. Nonoral modes of administration should only be considered if (a) the oral route becomes unavailable or (b) there is documentation of failure of maximal doses of oral morphine and coanalgesic drugs.[2]

There are coincidental developments of morphine tolerance and tolerance-associated hyperalgesia. Repeated treatment with opioids could set up a condition mimicking ongoing nociceptive input through interaction between opioid and NMDA receptors. This concept is the basis for recommending a combined use of opioids and clinically available NMDA receptor antagonists.[97]

Hydromorphone

Hydromorphone is a semisynthetic opioid agonist and is a hydrogenated ketone of morphine. Hydromorphone is commonly used for moderate to severe pain. It is available in oral, rectal, parenteral, and intraspinal preparations. It is highly lipid-soluble and generally well tolerated, especially in the elderly when compared with morphine or meperidine.[85] The potency ratio for hydromorphone to morphine has been variously reported as between 3:1 and 7.5:1, depending on the route of administration.

Hydromorphone appears to be a potent analgesic as predicted for the μ-opioid receptor agonist. This effect is dose related. The adverse effect profile of hydromorphone is also predictable and similar to other strong opioid agonists. The evidence available does not convincingly demonstrate clinical superiority of hydromorphone over the other strong opioid analgesics.[114]

Codeine

Codeine is a narcotic agonist rarely used for management of mild to moderate pain because of its low analgesic potency. It is more frequently used as an antitussive. Codeine, unlike other commonly used opioids, has a "ceiling effect," above which additional doses fail to produce additional analgesia. Unfortunately, there does not appear to be a "ceiling" for side effects. Codeine is available in oral and parenteral preparation, but the oral product is only two-thirds as effective as the parenteral preparation for pain.[85] Codeine is metabolized to morphine, and this hepatic metabolism occurs through the CYP-450 2D6 pathway.[10]

Oxycodone

Oxycodone is available both as an immediate-release and a continuous-release (8 to 12 hours' duration) preparation (OxyContin), and these dosage forms can be used for moderate to severe pain. Oxycodone is commonly available as combination analgesia in conjunction with aspirin (Percodan) or acetaminophen (Percocet). The fixed-dose oxycodone combinations should not be used chronically in large doses for more severe pain because of the risk of dose-related toxicity from the nonopioid ingredient. For patients who require treatment for an extended period, it is beneficial to convert patients from immediate-release to controlled-release preparation. The controlled release tablets should be swallowed whole and never be broken. Taking broken tablets leads to rapid absorption and potentially fatal doses.[65,85]

Fentanyl

Fentanyl is a synthetic opioid commonly used as an analgesic and as an anesthetic because of its cardiac stabilizing effect. Fentanyl is approximately 80 to 100 times as potent as morphine and has a faster onset of action but a shorter duration of action. Fentanyl is highly lipid-soluble. This allows it to easily cross the blood-brain barrier and is responsible for its rapid onset of action.[65,85]

Fentanyl is extensively metabolized in the liver, so doses should be reduced in the setting of underlying liver disease. Doses should also be reduced in the elderly. Elderly patients are twice as sensitive to the effects of parenteral fentanyl as the younger population.[85] Parenteral fentanyl is used usually only in anesthesia and in the postoperative recovery period. Transmucosal and transdermal fentanyl are used in the management of chronic pain. Transmucosal fentanyl is available either as a lozenge (Oralet) or lozenge on a stick (Actiq). These transmucosal products are indicated only in the management of breakthrough pain in patients with cancer who are already receiving and are tolerant to opioid therapy.[85] Fentanyl lozenges are designed to be sucked, not chewed. Chewing and swallowing the medicine results in lower peak concentration and lower bioavailability of the drug.

Transdermal fentanyl is usually used to provide continuous analgesia, and the initial dose should not be greater than 25 mg/hr. It is important for the physician to recognize that it may take 1 to 2 days for transdermal fentanyl to provide proper analgesia. Patients should be advised to avoid exposing the fentanyl application site to direct heat (e.g., a heating pad, a nearby lamp, or a hot tub), because there is a temperature-dependent increase in fentanyl released from the transdermal system. Patients with fevers should also be monitored for side effects or signs suggesting a higher than appropriate dose of fentanyl.[85,130]

Methadone

Methadone oral bioavailability is 85%, and single-dose studies have estimated an oral–to–parenteral potency ratio of 1:2. Its plasma half-life averages 24 hours, whereas the duration of analgesia is often only 4 to 8 hours.[65] Repetitive analgesia doses of methadone lead to drug accumulation because of the discrepancy between its plasma half-life and the duration of analgesia. Sedation, confusion, and even death can occur when patients are not carefully monitored and the dosage is not adjusted as needed during the accumulation period, which can last from 5 to 10 days.[65] The normal starting dose for analgesia is 2.5 to 10 mg every 6 to 8 hours. Dosage should be adjusted in renal failure. No such dosage adjustments are necessary in liver disease unless it is severe.[85]

Methadone is inexpensive when compared with continuous-release opioid preparations and is often an excellent choice for many patients requiring long-term opioid therapy of pain on an around-the-clock basis. Caution should be used when increasing doses, especially in frail or elderly individuals, because of potential for accumulation of long-acting metabolites and multiple drug interactions.

The dosage form of methadone that is used is a racemic mixture of equal amounts of L-isomer, an opioid, and D-isomer, which has only weak opioid activity. Both the L- and D-isomers of methadone bind to the NMDA receptor, and the D-isomer has functional NMDA-receptor antagonist activity in animals, including antihyperalgesia and antiallodynia activity in animal models of painful peripheral neuropathy and the ability to prevent the development of morphine tolerance.[65]

Meperidine

Meperidine, an opioid with a short half-life, is used for the treatment of moderate to severe pain. Meperidine is only one-tenth as potent as morphine, but it has a quicker onset

of action.[10,85] The duration of action is 2 to 4 hours. Aside from analgesia, meperidine is also used for premedication, prevention, and treatment of postoperative shivering.[85,159]

The main drawback to meperidine is its side-effect profile. Ninety percent of meperidine is metabolized in the liver to normeperidine, a metabolite that has a half-life of its own up to 40 hours.[85] It has neuroexcitatory effects including mood effects, followed by tremors, multifocal myoclonus, and occasionally seizures.[65,85] These effects are more pronounced in the elderly or in cases of renal failure.

Propoxyphene

Propoxyphene, a weak μ-agonist, is a synthetic opiate analgesic with chemical similarity to methadone. It has low analgesic efficacy and is indicated for mild to moderate pain.[10,85] It is metabolized in the liver to norpropoxyphene, which is eliminated in the urine. Norpropoxyphene is not an opioid, has a long half-life, and is associated with neuroexcitation, proarrhythmic lidocaine-like effects, and pulmonary edema. Naloxone does not reverse the effects of norpropoxyphene.[10,85]

Long-term use of this agent is highly discouraged, and use in elderly patients is not recommended. It has little, if any, advantage over nonopioid analgesia.[130]

Opiate Agonist-Antagonists

The agonist-antagonist opioids, which include drugs such as pentazocine, nalbuphine, and butorphanol, have predominantly κ-agonist analgesia effect while antagonizing the μ-receptor.[65,130] They are relatively short acting and not available in continuous-release preparations. The agonist-antagonist drugs have reduced analgesic efficacy compared to pure μ-agonists and exhibit a ceiling effect.[65,130] In therapeutic doses, they may produce certain self-limiting psychotomimetic effects in some patients, and pentazocine is the most common drug associated with these effects.[65] These drugs play a very limited role in the management of chronic pain because the incidence and severity of the psychotomimetic effects increase with dose escalation and because they are not available in convenient oral dosage forms.[49]

Because of their antagonist activity, they may reverse analgesia and precipitate withdrawal in individuals who are physically dependent on μ-opioids; therefore, they cannot be used to manage acute pain or breakthrough pain in patients taking pure μ-agonists.[65,130]

Opiate Partial-Agonists

The partial agonist, buprenorphine, may precipitate withdrawal in patients who have received repeated doses of morphine-like agonists and developed physical dependence.[65]

Buprenorphine is a partial μ-agonist opioid that is used in addiction treatment for both withdrawal and maintenance therapies, as well as for pain relief.[130]

Adverse Effects of Opioids

Adverse effects appear to depend on a number of factors including age, extent of disease and organ dysfunction, concurrent administration of certain drugs, prior opioid exposure, and the route of drug administration.[65] The most common adverse effects are sedation, nausea, vomiting, constipation, and respiratory depression.

Central Nervous System

Opioids produce sedation and drowsiness, which can be expected to be a least additive with the sedative and respiratory depressant effects of other sedative hypnotics such as alcohol, barbiturates, and benzodiazepine.[65,85] Concurrent administration of dextroamphetamine in 2.5- to 5-mg oral doses twice daily has been reported to reduce the sedative effects of opioids. Tolerance usually develops to the sedative effects of opioid analgesics within the first several days of long-term administration.[65]

Opioids inhibit the hypothalamic release of both gonadotropin-releasing hormone and corticotropin-releasing hormone. In women, an opioid-induced decrease of follicle stimulating hormone (FSH) can cause anovulation and amenorrhea. In men, luteinizing hormone decreases during opioid administration, causing decreased testosterone release, decreased libido, and reduced sperm motility. Opioids can evoke the release of antidiuretic hormone, causing fluid retention.[96] Opioids can also cause paradoxical excitation, which is dose-dependent and is usually observed with high doses of potent opioids.

Respiratory System

Respiratory depression is the most dangerous complication and occurs in 0.1% of patients receiving oral or rectal opioids.[35] Opioids cause a dose-dependent respiratory depression. Activation of μ_2-receptors blunts the sensitivity of chemoreceptors in the brainstem to carbon dioxide.[65,96]

Individuals with impaired respiratory function or bronchial asthma are at greater risk of experiencing clinically significant respiratory depression in response to usual doses of these drugs.[85] Respiratory depression can be reversed by administration of the specific opioid antagonist naloxone.[65]

Cardiovascular System

Opioids cause bradycardia and decreased sympathetic tone. This may lead to hypotension, especially in the hypovolemic patient.[65,85,96]

Gastrointestinal Tract

Adverse GI effects include esophageal reflux, spasm of the sphincter of Oddi, and decreased pancreatic, biliary, and intestinal secretions.[96]

Opioids produce nausea and vomiting by an action on the medullary chemoreceptor trigger zone. The incidence of nausea is from 10% to 40%, but tolerance develops

rapidly. Nausea can be prevented by the use of antiemetics, such as prochlorperazine or metoclopramide.[65,85,96]

Constipation is probably the most common side effect associated with opioid therapy, occurring in 35% of patients taking opioids. These drugs act at multiple sites in the GI tract and spinal cord to produce a decrease in intestinal secretion and peristalsis, resulting in a dry stool and constipation. Tolerance develops very slowly. A diet rich with fiber and fluids combined with surfactant agents (e.g., docusate sodium) and lubricants (e.g., fish oil gel capsules) should routinely be prescribed with opioids. Stimulant laxatives (e.g., bisacodyl) and osmotic laxatives (e.g., lactulose) are also often necessary. An aggressive "bowel regimen" is particularly important in those most likely to experience constipation.[85]

Urinary System

Opioids can cause bladder spasm and an increase in sphincter tone leading to urinary retention, particularly in elderly patients.[65,85,96] These effects may be attributed to activation of μ- and δ-receptors supraspinally or spinally or to direct effects on the urinary system.[96] The severity of urinary retention may lessen with chronic use.[78]

Musculoskeletal Side Effects

At high doses, all of the opioid analgesia can produce multifocal myoclonus. This complication is most prominent with the use of repeated administration of large parenteral doses of meperidine.[96]

Muscle rigidity occasionally occurs with opioids, more commonly with higher doses of the potent, rapidly acting drugs. This is a dangerous side effect, potentially leading to difficulty in ventilation.[85]

Immune System

In vitro assays and animal studies indicate that opioids such as morphine can suppress a number of immunologic variables.[65] In the Palm et al. study, morphine did not affect cellular immune function. However, those patients with chronic pain produced smaller amounts of immunoglobulin than controls, and immunoglobulin production was reduced further by morphine.[65a]

Tolerance, Dependancy, and Addiction

Physical dependency reflects a state of neurophysiologic adaptation, which is present when withdrawal phenomena occur on abrupt cessation or precipitous decrease in doses of the medication or administration of an antagonist.[130]

Tolerance is present when increasing doses of an opioid are required to produce the initial effects of the drug.[56] The pain-facilitating systems including the NMDA receptors, nitric oxide, and COX systems may play important roles in opioid tolerance.[64]

Addiction in the context of opioid therapy of pain is reflected in a constellation of maladaptive behaviors, including loss of control over the use of the opioids, preoccupation with opioid use despite adequate pain relief, and continued use of opioids despite apparent adverse consequences because of their use.[130] Patients at risk of prescription opioid addiction are those with a history of substance or alcohol abuse, with a family history of addiction, using short-acting opioids, and with psychological problems.

Abuse in the context of opioid therapy of pain suggests that an individual is using the medication in a way that may cause harm to self or to others or is using it for an indication other than that intended by the prescribing clinician.

Pseudoaddiction refers to the perception by observers of apparent drug-seeking behavior in patients who have severe pain and have not received effective treatment of pain.[130]

The onset of *withdrawal* is characterized by the patient's report of feelings of anxiety, nervousness, and irritability and alternating chills and hot flushes. A prominent withdrawal sign is "wetness," including salivation, lacrimation rhinorrhea, and diaphoresis, as well as goose flesh. At the peak intensity of withdrawal, patients may experience nausea, vomiting, abdominal cramps, insomnia, and, rarely, multifocal myoclonus. Abstinence symptoms will appear within 6 to 12 hours and reach a peak at 24 to 72 hours following cessation of a short–half-life drug such as morphine, whereas onset may be delayed for 36 to 48 hours with methadone. The detoxification dose is equal to approximately one-fourth of the previous daily dose, which is divided to four doses and then decreased by half every 2 days until a total daily dose of 10 to 15 mg/day (in morphine equivalent) is reached. After 2 days on this dose, the opioid can be discontinued.[65]

The opioid contract or agreement maybe an appealing tool for managing many of the potential difficulties related to chronic opioid therapy for noncancer pain. It is also recommended that patients have a primary care physician who must also sign the opioid contract. The primary care physician signature indicates agreement with the decision to begin chronic opioid therapy and to become the prescriber once the patient's regimen has stabilized.[46]

Antidepressants

Most patients with chronic pain could benefit from antidepressants, especially if they have comorbid psychiatric conditions such as depression and sleep disorders; psychological factors affecting physician conditions; anxiety; somatization disorder; or somatoform pain disorder. There is a documented role for antidepressants in the treatment of chronic pain syndromes.[155] The rationale of prescribing antidepressants as analgesics is that they block reuptake of neurotransmitters (e.g., norepinephrine and serotonin) important to pain modulation.[6] There are consistent data that antidepressants do have an antinociceptive effect for various forms of chronic pain. Data for the efficacy of antidepressants are stronger for neuropathic pain than other types of pain.[45] Imipramine, amitriptyline, and desipramine have

demonstrated efficacy in painful diabetic neuropathy[167,170], with the latter two drugs being found to be analgesic in postherpetic neuralgia.[155]

The role of selective serotonin reuptake inhibitors (SSRIs) in neuropathic pain is more controversial than that of TCAs.[156] Fluoxetine has demonstrated analgesia properties in experimental animal pain models, but has failed to show analgesic effects in a clinical trial for neuropathy. Paroxetine is the first SSRI shown to be a highly effective analgesic in a controlled trial for the treatment of diabetic neuropathy.[155]

Antidepressants could be effective for pain associated with some specific pain syndromes such as osteoarthritis or rheumatoid arthritis, fibrositis, or fibromyalgia. Immediate or early analgesia effects occur within hours or days and probably are mediated through inhibition of synaptic reuptake of catecholamines. The later analgesic effects that peak over a 2- to 4-week period probably are due to the effect on receptors.[100,155] Antinociceptive effects of antidepressants seem to be independent of their effect on depression. Pain generally is felt to respond to a lower dosage than does depression.[155]

Antidepressant medications may be divided into four general categories: TCAs, monoamine oxidase inhibitors (MAOIs), SSRIs, and others. Further discussion of individual antidepressants is beyond the scope of this chapter.

Centrally Acting Agents

Tramadol Hydrochloride

Tramadol is a synthetic 4-phenylpiperidine analog of codeine. Tramadol is a weak μ-opioid receptor agonist and inhibits the uptake of norepinephrine and serotonin.[115–136]

The affinity of tramadol for μ-opioid receptors is 6,000-fold less than morphine, 10-fold less than codeine, 60-fold less than propoxyphene, and 1,000-fold less than methadone. Analgesia begins within 1 hour of oral administration and peaks in 2 to 3 hours. Tramadol is metabolized in the liver and exerted primarily in the urine.[84]

Tramadol has been shown to be effective in the treatment of various acute pains including postoperative pain after orthopedic and abdominal gynecologic surgery. The recommended oral dosage is 50 to 100 mg every 4 to 6 hours. The most common adverse effects of oral tramadol in patients with chronic nonmalignant pain occurred with the following frequencies: dizziness or vertigo, nausea, vomiting, constipation, headache, somnolence, pruritus, CNS stimulation, dry mouth, sweating, and diarrhea. Respiratory depression is significantly less than with morphine sulfate. Tramadol has caused seizures in humans receiving large oral or IV doses.[84] Predisposing risk factor for seizures include a history of epilepsy, head trauma, metabolic disturbances, alcohol or drug withdrawal, or a CNS infection. The risk of seizures was increased in patients taking agents that lower the seizure threshold, such as TCAs, SSRIs, anorexiants, MAOIs, and neuroleptics. In cases of tramadol overdose, naloxone may increase the risk of seizures.[84] Tramadol causes minimal dependency and tolerance; however, several cases of tramadol abuse have been reported, mostly in patients with a history of addiction or dependence on opiate agonists.[84]

There is potential for chronic tramadol use to induce withdrawal of the clinical opioid type upon abrupt cessation. Studies show that one in eight Ultram (tramadol hydrochloride) withdrawal cases presents as a mixture of classical opioid withdrawal with unusual features such as intense anxiety, depersonalization, delusions, confusion, hallucinations, and other symptoms. The rate of withdrawal is approximately 1 per 100,000 patients exposed.[136]

Local Anesthetics/Antiarrhythmics

Lidocaine and mexiletine, class 1b antiarrhythmic agents, have each been shown to relieve a variety of neuropathic pain disorders, such as peripheral nerve injury, chronic diabetic neuropathy, and postherpetic neuralgia. Clinically effective plasma concentrations of local anesthetic antiarrhythmic drugs suppress abnormal neuronal discharges from injured primary afferents through Na^+ channel blocking.[47]

Mexiletine attenuated hyperalgesia and mechanical allodynia in neuropathic rats with selective nerve root ligation.[29] Studies of experimentally induced neuromas in animals, as well as studies in cancer patients, suggest that systemic administration of these agents may be able to target spontaneously active nerves while sparing conduction in normal nerves. In particular, lidocaine appears to suppress spontaneous neuronal discharge in Aδ- and C-fibers by blocking sodium channels.[49]

Even temporary responsiveness to intravenous lidocaine does not predict success with other local anesthetic agents given orally, such as mexiletine hydrochloride. Several studies have documented the benefit of mexiletine in chronic pain. A dose of 450 mg/day appears to be optimal.[4] Systemic adverse effects of antiarrhythmic agents include both neurotoxicity and cardiotoxicity.[49]

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43 Anesthesia for Shoulder Surgery

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This chapter is written as a resource for practicing orthopedic surgeons. The objectives of this chapter are to provide preoperative evaluation information that is essential for the preparation of the orthopedic surgical patient; a discussion of the anesthetic options and why they are selected for a particular patient for a given shoulder surgery; a description of the regional anesthetic technique including the preoperative preparation of the patient and insertion of the interscalene block; and a discussion on postoperative pain management. The authors have focused on the postoperative pain management techniques as we believe this is a critical component to the management of the orthopedic surgical patient.

PREOPERATIVE EVALUATION

With the increasing complexity of the surgical procedures and the increasing severity of the coexisting illness present in surgical patients, the anesthesia team is required to evaluate and optimize the patient's health status. In this section we describe medical conditions that influence the health status of the patient and discuss how they are evaluated and optimized prior to surgery.

The patient's visit to the preoperative assessment clinic should provide the anesthesiologist with a number of important pieces of information that can be utilized to best care for the patient. These included the current list of medications, past anesthetic history, patient's concerns and desires regarding the planned anesthetic management, and an indication of major physical illness that will affect their anesthetic management. This visit also provides the opportunity for the anesthesiologist to discuss the anesthetic plan, to discuss any invasive monitors or special techniques that may be needed, and to obtain informed consent from the patient. In summary, the goal of the anesthesia team is to provide active, informed consent after complete evaluation of the patient's health status. This is performed after the surgery team has presented the traditional discussion of the risks and benefits of the proposed surgical procedure. The American Society of Anesthesiologists (ASA) physical status (Table 43-1) is a standard benchmark used for the evaluation of all surgical patients. Patients classified as ASA physical status 1 to 2 may be evaluated as late as the day of surgery. However, patients classified as ASA status 3 or higher should be evaluated sooner to avoid presurgical delays.

TABLE 43-1
THE AMERICAN SOCIETY OF ANESTHESIOLOGISTS' PHYSICAL STATUS CLASSIFICATION

ASA Class 1	A healthy patient (no organic, physiologic, biochemical, or psychiatric disturbance)
ASA Class 2	A patient with mild to moderate systemic disease Examples: Essential hypertension, diabetes mellitus, chronic bronchitis
ASA Class 3	A patient with severe systemic disease that limits activity but is not incapacitating Examples: Heart disease that limits activity, poorly controlled hypertension, diabetes mellitus with vascular complications, angina pectoris
ASA Class 4	A patient with incapacitating systemic disease that is a constant threat to life Examples: Congestive heart failure, advanced renal, pulmonary, or hepatic dysfunction
ASA Class 5	A moribund patient who is not expected to survive for 24 hours with or without an operation Examples: Pulmonary embolus, cerebral trauma, ruptured abdominal aneurysm. In the event of an emergency operation, the number is preceded with an E.

PREOPERATIVE LABORATORY TESTING

Preoperative laboratory testing is an area where rapid change is occurring. It is increasingly apparent that routine lab tests may actually hinder the preoperative process logistically and medically. The rate of detection of real medical problems by routine screening tests is extremely low by comparison to the cost of these tests and the number of patients subjected to unnecessary further workup. Routine screening of blood counts and electrolytes, electrocardiograms, or chest radiograms are probably not reasonable without indication, such as age, disease states, and medication. Specific noninvasive and even invasive testing may be indicated if suggested by the screening history and physical. Knowledge of the implications of the surgical procedure and common coexisting illnesses associated with that procedure could further focus the attention at preoperative screening. Occasionally, anesthetic techniques and procedures will be an indication for specific preoperative testing (i.e., regional anesthesia, invasive monitoring, and assessment of coagulation).

COEXISTING DISEASE—RHEUMATOID ARTHRITIS

Rheumatoid arthritis is a disease of synovial membrane destruction that causes many patients to present for orthopedic surgery. Virtually every joint in the body can be a target, although the pattern is variable by individual.[29] The consequences of this disease and its treatment present a number of preoperative issues.

Due to the existence of synovial joints at a variety of locations, the management of the airway in rheumatoid patients is the most common and often the most serious preoperative issue. The cervical spine, temporomandibular joint (TMJ), and arytenoid bodies are all subject to this disease process. Decreased or absent range of motion of the neck may predict difficult airway management. When this is combined with decreased mouth opening due to TMJ involvement, fiberoptic visualization may be the only possible approach to intubate the patient.

When synovial destruction progresses far enough to cause weakening of stabilizing ligaments, cervical spine instability can occur at the fixation of the axis of C2 to the ventral side of C1[28,42] or erosion of the axis.[53] In both cases, dorsal translocation of C2 on C1 places the spinal cord at risk.[18,43] This can occur even in a neck with limited range of motion at other sites.

When early instability is identified, it is assumed that progression will occur. Documentation with lateral neck x-rays is mandatory prior to anesthesia, unless fusion has been verified. Conventional airway management involves neck extension, and traumatic injury or even complete disruption of the cervical spinal cord has been reported. Simple

lateral films are not adequate, since instability may only be detectable with movement. The standard x-ray workup of the rheumatoid neck must include forced flexion and extension and open mouth odontoid views.

A preoperative dilemma occurs when the rheumatoid patient without known instability presents for elective surgery. While the patient may be asymptomatic, the incidence of silent C-spine instability is not rare. Some centers will require flexion-extension films for elective surgery. If intubation can not be performed without spine manipulation, either x-ray evaluation or awake intubation will be indicated. In cases of extreme instability, even awake intubation by itself may not be totally safe, and prior mechanical stabilization of the neck such as the halo device or cranial tongs with traction may be necessary. Emergency airway management requires manual in-line stabilization of the neck.

Other stigmata of rheumatoid arthritis can result in pulmonary compromise including greatly reduced range of motion of the thorax and destruction of pulmonary parenchyma leading to restrictive pulmonary disease.

The generalized loss of tissue stability presents positioning risks, especially in rheumatoid patients on chronic steroid therapy or cytotoxic drugs. These patients may have extremely thin skin with reduced tensile strength, which is subject to injury, with minor pressure or removal of adhesive tape. Bed surfaces need to be padded and surgical devices to stabilize the patient (bean bags, kidney rests) need to be applied with the decreased tensile strength of the skin in mind.

In some rheumatoid patients, chronic synovial effusion can lead to accumulation of a transudate in the pericardium. A large pericardial effusion can exist with minimal symptoms and create unexpected tamponade physiology during anesthesia care. Rheumatoid patients with limited activity should have echocardiography. Patients on chronic steroid therapy should be considered adrenal suppressed, and full stress-dose steroid given.

CARDIOVASCULAR DISEASE

Hypertension

There are no data available to evaluate the risk of hypertension in the orthopedic surgical patient. The information available applies to elective surgery in general and is conflicting, due to variation in study design and outcome definitions. Later work designed to address the need for perioperative control of blood pressure seemed to suggest that hypertensive patients have better perioperative care if their blood pressure was controlled prior to anesthesia. It is clear that poorly controlled hypertensive patients have more hemodynamic instability. It should also be noted that preexisting hypertension may limit the ability to safely decrease the blood pressure to the surgeon's desired limit for the surgical procedure.

Hypertension may influence the anesthesiologist to avoid the addition of epinephrine to the local anesthetic solution utilized for regional anesthesia blockade. These patients should be instructed to continue taking most hypertensive medications right up to the morning of surgery with the exception of diuretics, angiotensin-converting enzyme inhibitors, and angiotensin receptor blockers. If the diastolic blood pressure exceeds 110 mmHg, there is a reasonable indication for therapeutic adjustment of antihypertensive therapy. To facilitate monitoring and control of the blood pressure, the anesthesiologist may consider placement of an arterial line catheter for patients with uncontrolled blood pressure in the emergent setting.

CORONARY ARTERY DISEASE

The objective in preoperative screening is to identify those patients requiring further diagnostic evaluation and perhaps further medical or surgical procedures. It is also especially important to identify those patients who are scheduled for high-risk surgical procedures for whom the perioperative risk of myocardial ischemia is great so that the patient may make an informed decision. If there has been a prior event such as a myocardial infarct, angioplasty, or coronary artery bypass grafting, the anginal pattern since the event should be determined to detect those patients at risk for perioperative ischemia. Patients at risk for silent ischemia (such as diabetics) may present an indication for more aggressive diagnostic testing prior to elective major orthopedic surgery.

The simple electrocardiogram (ECG) is indicated as a screening test in patients with cardiac risk factors such as family history, hypertension, and diabetes mellitus. Any patient with known cardiac disease should have a current ECG, and it should be compared to a prior tracing to detect interval change. New arrhythmias, ST segment changes, Q waves, or signs of ischemia at rest must be further evaluated prior to elective surgery. Specifically, the new diagnosis of atrial fibrillation or left bundle branch block may oftentimes require a specific workup that may delay the scheduled surgery if the patient is being seen in the preoperative evaluation clinic on the day prior to surgery.

If the patient's history indicates that a stress test may be needed, then it should be performed in the preoperative evaluation phase. If the patient is unable to tolerate exercise testing due to arthritic joints, then pharmacologic stress testing may be chosen such as an echocardiographic dobutamine stress test or Persantine thallium or reperfusion imaging.

Knowledge of the function of the left ventricle (LV) can be important in planning the anesthesia for the orthopedic patient with previously known LV dysfunction. The least

invasive of the tests is echocardiography. The properly conducted transthoracic echo will reveal left ventricular ejection fraction almost as accurately as cardiac catheterization and will detect wall motion defects indicative of ischemia or prior infarct. It can also estimate the function of the major cardiac valves. Stress testing and echocardiography will provide information regarding ischemic risk, left ventricular function, and valvular disease. The primary component remaining to be evaluated if disease were present would be the degree of stenosis in the coronary vessels, obtainable through cardiac catheterization.

Cardiac catheterization is the most definitive and invasive preoperative evaluation of the myocardium. The major benefits to be gained are precise knowledge of the coronary anatomy and exact indicator of left ventricular filling and function. In some preoperative patients who have indications for cardiac catheterization, lesions will be found, which require revascularization prior to elective major orthopedic procedures. Undetected, these lesions could cause myocardial infarct, congestive heart failure, or sudden death in the perioperative period. It should be noted that a coronary artery that requires a stent prior to orthopedic surgery may require antiplatelet medications. This may result in the delay of an elective orthopedic surgical procedure. There may be a positive role for beta blockade in patients who are at risk for cardiac ischemia.[3] Given the low cost and low risk for this intervention, cardiac patients should be evaluated for perioperative beta blockade.

VALVULAR HEART DISEASE

The preoperative evaluation of the patient with valvular heart disease will be essentially the same as that of coronary artery disease, with the exception of quantifying the degree and clinical significance of the valvular lesion. This is particularly important in the stenotic lesions of the left ventricle and mitral and aortic stenosis. The coexistence of coronary artery disease must be suspected in patients with aortic stenosis since the combination of stenosis and significant coronary artery disease is common and poorly tolerated in the perioperative period. Function of the left ventricle is also important to evaluate, since most valvular lesions induce hypertrophy of the myocardium in response to increased work and the potential for cardiomyopathy or congestive heart failure.

PULMONARY DISEASE

The preoperative evaluation of the orthopedic patient with pulmonary disease is designed to identify correctable problems that could lead to pulmonary complications in the postoperative period. To be effective, intervention requires accurate timing since optimization requires time for treatment. The goals involve the elimination or suppression of all active infections, maximum treatment of bronchospasm, facilitation of sputum clearance, cessation of smoking, and treatment of any serious nonpulmonary sequelae of pulmonary disease.

If there is a question of adequate function, chest x-ray, room air saturation, arterial blood gas analysis, and formal pulmonary function testing (PFT) or bedside spirometry may be indicated. Short-term cigarette abstinence will favorably influence carboxyhemoglobin levels. If small airway disease is serious, diffusion studies may be necessary. With either serious obstructive or restrictive disease, flow-volume loops may help to quantify the degree of limitation. Diffusion capacity, carbon monoxide (DLCO) can help distinguish among obstructive lung disease between fixed lesions (decreased DLCO) and bronchospastic disease (normal DLCO).

Orthopedic patients with obstructive pulmonary disease should be optimally prepared for elective surgery. Evidence of acute infection must be sought, and if present, it requires delay of surgery in most instances. Any component of airway obstruction that is reversible with bronchodilator therapy during pulmonary function testing must be effectively treated. If theophylline therapy is part of the chronic prevention of bronchospasm, the blood levels should be therapeutic. If steroid therapy has been required, it is mandatory to continue steroid therapy through the perioperative period. If the forced expiratory volume (FEV_1)/functional residual capacity (FRC) ratio is less than 40% or if there is a 40% or greater reduction in diffusion capacity, the morbidity in the perioperative period greatly increases.

THE HIGH-RISK PATIENT

Patients with severe cardiovascular, pulmonary, or other serious illness can seek surgical intervention for orthopedic problems, such as total joint replacement. The severity of the coexisting disease can be so severe that purely elective surgery would not be reasonable due to the anesthetic and postoperative risk. However, some of these patients with a reasonable life expectancy have such extreme symptoms from their orthopedic pathology that they are extremely motivated to have elective surgery. The first step in dealing with a high-risk, elective orthopedic patient is to verify that all parties involved have the same understanding of the events. The medical specialist caring for the patient must understand the perioperative risk of the surgical procedure and the rehabilitation. It must be clear that all correctable conditions have been treated and that the serious coexisting disease has been optimally prepared. The preoperative planning must include informed consent, and the patient must be aware of what specific risks he or she is accepting. Pertinent family members involved in postoperative care

must also be aware of the potential outcome. Documentation must be explicit and detailed prior to anesthetic intervention.

ANESTHETIC OPTIONS

General and Regional Anesthesia Considerations

Shoulder surgery can be successfully performed under general anesthesia, regional anesthesia, or a combination of the two. In the authors' institution, the protocol is to place an interscalene brachial plexus block in the majority of patients undergoing shoulder surgery (Table 43-2). In general, patients who do not receive an interscalene block are those who have any contraindication to an interscalene block or are having surgical procedures that would not require an interscalene block for postoperative pain relief. The final anesthetic plan is based on the complexity of the planned surgical procedure and patient preference. A general anesthetic as the sole technique avoids the "disadvantages" associated with a regional anesthetic, including the time associated to place the regional anesthetic, the chance that the regional anesthetic might be inadequate, any patient discomfort associated with the placement of the regional anesthetic, and the need for special equipment such as nerve stimulators that might be necessary to place the regional anesthetic.

However, utilizing a general anesthetic as the sole technique does not offer the patient the valuable benefit of postoperative pain relief and preemptive analgesia. Preemptive analgesia, where the analgesic interventions are initiated prior to the surgical incision, may prevent peripheral hypersensitivity and thus result in a decrease in the overall pain experienced postoperatively by the patient. There exist concerns regarding the clinical relevance of preemptive analgesia and how to best study the claims that preoperatively administered analgesics provide long-lasting analgesia from 7 to 10 days postoperatively.[16] With regard to interscalene block for elective shoulder surgery, Wurm et al. found that patients who received an interscalene block prior to surgery versus those who were administered postoperatively had superior pain control for the first 12 hours. However, this benefit was not maintained during the week after discharge.[77] The decision regarding the anesthetic plan revolves around whether the procedure can be completed under the sole technique of an interscalene block versus a combined technique of general anesthesia plus an interscalene block.

The combined technique is selected when patient preference for unconsciousness is strong, such as unusual levels of anxiety or known claustrophobia, since the sterile field requires the patient's face to be nearly covered.[20] Certain surgical procedures may require a combined technique when anatomic areas are not anesthetized by an interscalene block. For example, incisions that extend onto the pectoral prominence are not covered by brachial plexus block. Anesthetic coverage of the pectoral prominence would require the somatic block of the first and second intercostal nerves (Fig. 43-1). This can be achieved with

TABLE 43-2
INTERSCALENE BLOCK IN ADDITION TO GENERAL ANESTHESIA—ADVANTAGES AND DISADVANTAGES

Advantages	Disadvantages
Enhanced pain control	Requires trained personnel to perform interscalene anesthesia
Minimal general anesthetic requirements	May require additional time to perform block if no induction room available
Earlier discharge for outpatient surgery	Known side effects of block include phrenic nerve blockade, hoarse voice, etc.
Reduction in recovery room of analgesic use with subsequent decreased side effects	Requires availability of nerve block equipment and resuscitation equipment
Increased patient satisfaction with postoperative pain control	Complications include small risk of pneumothorax, subarachnoid, and epidural block.

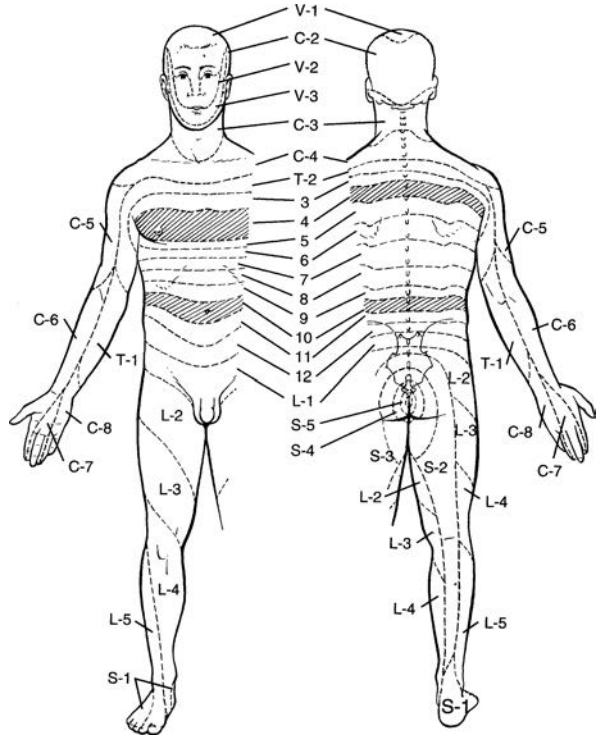

Figure 43-1 Posterior cutaneous innervation of the shoulder.

paravertebral blocks.[51] The combined technique may be required for procedures that require profound muscle relaxation, complex procedures that may require an extended amount of time in the operating room, or procedures that require positioning that could be uncomfortable for an awake patient.

General Considerations for Shoulder Surgery

There are general considerations regarding shoulder surgery that are independent of anesthetic technique. Standard monitoring is required for all shoulder surgery. The patient should have at least one functioning intravenous catheter. The patient should be checked for generic positioning concerns and all pressure points should be padded. Blood products may be made available for more complex procedures including some cancer surgeries and revision arthroplasties. Routine type and cross for blood products is not necessary for routine shoulder surgeries.

Accessibility to the Airway

During shoulder surgery, access to the airway is limited due to the surgical proximity to the airway. In patients with sleep apnea, where the ability to use sedation is limited, general anesthesia with a secured airway combined with the interscalene block for postoperative analgesia may be a reasonable choice. Interscalene block as a sole anesthetic may require urgent airway management.

Severe Airway Obstruction During Arthroscopic Shoulder Surgery

There have been case reports of patients who presented with severe airway obstruction caused by tracheal compression due to extraarticular arthroscopic fluid accumulation during arthroscopic shoulder surgery.[8] This resulted from massive swelling of the ipsilateral chest and anterior portion of the neck extending to the face. The initial management required tracheostomy, but with cessation of pressurized arthroscopic fluid injection, manual ventilation became possible and the patient was able to be orotracheally intubated.

Response to Methylmethacrylate

Methylmethacrylate is used to secure the prosthetic components used for total shoulder replacement. When the monomer form enters the circulation, venodilation and histamine release have hemodynamic consequences, which can be exaggerated if the patient is hypovolemic, if the patient has diminished cardiac function, or if the cement is injected under high pressure. Because the amount of cement is significantly less with shoulder replacement compared to hip or knee replacement, hemodynamic instability is less common and severe.

Beach-Chair Position

Beach-chair position (Figs. 43-2 and 43-3) presents risks accentuated by the uniquely physical aspects of orthopedic surgery. The need for traction, manipulation, and other physical interventions such manipulation of bone place even the most careful positioning of body parts at risk. Frequent reassessment after periods of aggressive movement is important. In the "beach chair," which is a semi-sitting modification of the supine position, the head is raised as much as 45 degrees and the body is shifted to the edge of the bed on the side to be operated on. Positioning devices that suspend the arms overhead in the supine position can cause stretch to the brachial plexus if either extreme abduction or anterior flexion is performed and especially if combined with contralateral neck rotation. Venous air embolism is a potential issue since the wound area is above the heart, although the clinical experience has not demonstrated this to be a major concern. Spontaneous hypotension and bradycardia during shoulder surgery and interscalene block have been attributed to the Bezold-Jarisch reflex.[17] The cause is related to venous blood pooling (induced by the sitting position) and a heightened cardiac contractile state (induced by the beta adrenergic effects of epinephrine in the local anesthetic solution for the interscalene block) that may be activating this reflex.[39]

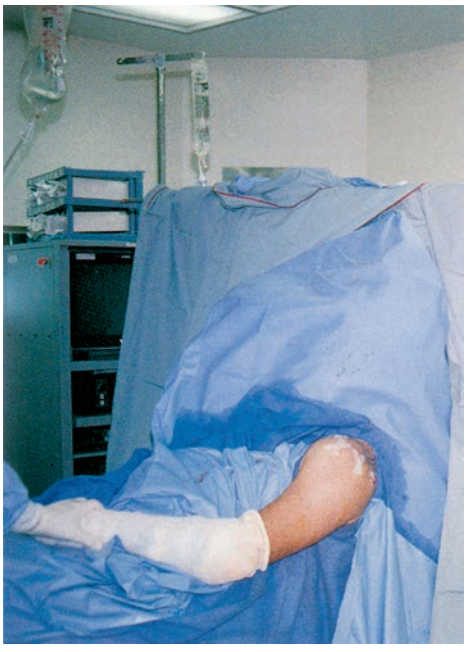

Figure 43-2 The surgical view of the draped patient in the beach-chair position.

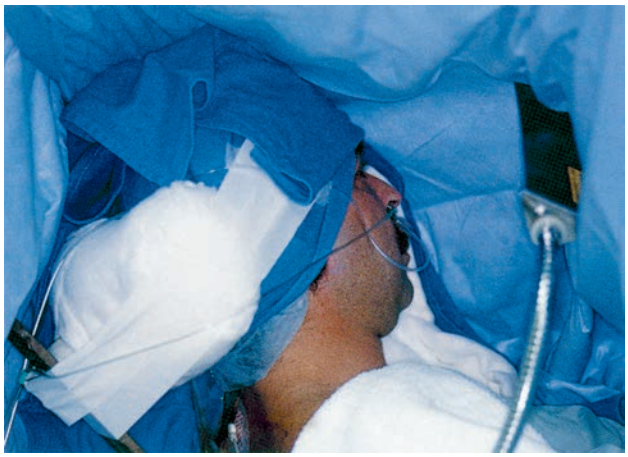

Figure 43-3 The anesthesia care provider's view of the draped patient in the beach-chair position. This demonstrates the reduced access to the face and airway of the shoulder surgery patient.

Lateral Position

Many of the positioning issues in the supine position also apply when the patient is in the lateral position. In addition, the pressure of the body on the dependent parts presents additional risk to nerves, muscle, and skin. The brachial plexus is at risk in the lateral position in both the dependent and the superior limb. The superior brachial plexus is at risk in those cases where extreme position causes traction, stretch, and subsequent ischemia to the brachial plexus. When the arm is moved superior, to above the level of the head, hyperabduction is possible, with the brachial plexus stretch occurring between the humeral head and the coracoid as lever points.

The dependent limb is at risk because the brachial plexus may be compressed between two bony structures, the rib cage and the head of the humerus. To avoid this, the weight of the thorax must be kept off of the humerus to protect the dependent brachial plexus. On the dependent limb, placing a foam or axillary roll of the proper size in the axilla and verifying that it is positioned properly prevents compression (Fig. 43-4). If the limb is positioned improperly, vascular embarrassment may be detected by venous engorgement or poor capillary refill. Pulse oximeter waveform and signal also should be checked on the dependent limb. It is important to remember that orthopedic surgery involves considerable physical movement of the body and that correct positioning can evolve into a position where the brachial plexus could be at risk, and should be checked at regular intervals.

General Anesthesia

General anesthesia with field avoidance usually requires that the airway be securely under control prior to surgical incision. While it may be possible to do these cases with laryngeal mask ventilation under general anesthesia, the airway must be very easy to manage and the provider skilled, because movement associated with airway adjustment would be disruptive to the surgical procedure. The need to suddenly intubate the trachea during the surgical procedure would be both difficult to accomplish and very disruptive, particularly if the sterile field had to be broken. The endotracheal tube must be protected from dislodgment, and it is important to remember the potential for accidental injury to the head from sharp or heavy objects placed on the drapes by the surgical team over the face and neck.

Regional Anesthesia

At the authors' institution it is the usual practice to place the interscalene block preoperatively in the awake patient in a special induction room area. Interscalene block is not placed in patients under general anesthesia, unless compelling reasons exist. The risk of interscalene block under general anesthesia is highlighted in a case where permanent neurologic deficit resulted after a nerve stimulator–guided interscalene brachial plexus block was performed during general anesthesia.[48] It was concluded that if the patient had been awake, there could have been feedback identifying intraneural placement of the needle and subsequently damage to a neural structure could have been avoided. This would have served as a protective mechanism allowing the anesthesiologist to discontinue the injection. In addition to this case, Benumof[6] has reported a series of four cases where interscalene blocks were placed under general anesthetics, resulting in cervical cord lesions visible on magnetic resonance imaging. Additionally, it should be noted that a successful regional anesthetic includes anxiolysis or, conversely, if the patient is extremely anxious the regional technique may fail as the sole anesthetic.

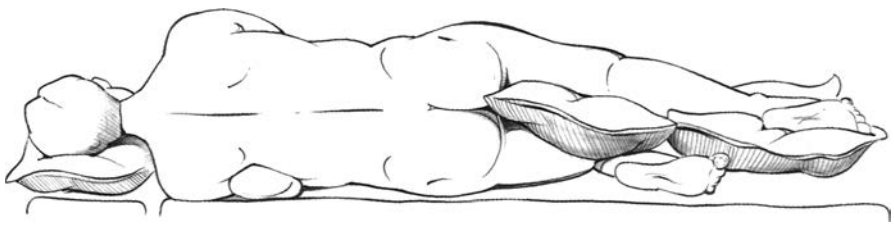

Figure 43-4 Patient in the right lateral decubitus position demonstrating a chest roll in place to protect the neurovascular bundle in the axilla.

Successful Block

Verifying a successful nerve block is important to ensure that the tissue within the surgical field is anesthetized. Although the interscalene block can provide complete anesthesia to the shoulder joint and surrounding muscles, this may not be adequate if the surgical incision extends into the upper axillary area or into the posterior aspect of the shoulder. The duration of the block may be a consideration if there is an unpredictable length of surgery. Blocks that are of a longer duration provide anesthesia throughout the duration of the surgery and then also into the postoperative period. Another surgical consideration is for adequate muscle relaxation, especially in very muscular patients and for more complicated procedures including complex and revision arthroplasties. While bupivacaine does exhibit some muscle relaxation at 0.5%, mepivacaine at 1.4% tends to provide complete muscle relaxation, especially when bicarbonate has been added.

Comfortable Patient

Many patients experience anxiety when considering being awake for a surgical procedure. They also have concerns about the pain experienced when the interscalene block will be placed. These patients can be reassured with constant communication and rapport established in the preoperative visit and throughout the perioperative processes. After consent is obtained and the patient's questions are answered, the administration of sedatives and/or analgesics helps to comfort the patient. Deep sedation should be avoided to prevent masking of paresthesias during the placement of the interscalene block. The nursing and surgical team will need to be cognizant of their behaviors as the awake patient may be listening to the sounds and conversations occurring during the operation.

Optimal Operating Conditions

These conditions include both the operating room management aspects of regional anesthesia and the surgical team's comfort with operating on an awake patient. The awake patient, at the conclusion of the procedure, can assist in the placement of braces and special dressings.

Uncomplicated Postoperative Course

The patient should understand the expected duration of the block and care should be taken to explain how to protect the anesthetized limb. This includes describing the risks of the excessive pressure or poor positioning on the limb. The patient may be instructed to take an oral analgesic at the initial sign of discomfort as this will establish analgesia before the block wears off completely. Specifically, in the outpatient setting, early treatment of pain may be associated with reduced analgesic requirements and higher patient satisfaction.

Preparation for Regional Anesthesia

Equipment and Supplies

If the block is to be performed in an induction room, then the room should be fully equipped with monitors and the ability to induce general anesthesia along with airway management devices providing the ability to ventilate and oxygenate. The anesthesia provider must be able to deal with outcomes resulting from intravascular injection of local anesthetic. These events could result in a spectrum of side effects ranging from a mild anesthetic toxicity to the abrupt onset of generalized seizures. High subarachnoid, epidural, or subdural injection is also possible and would result in hemodynamic collapse and unconsciousness. With the proper resuscitation equipment readily available, these complications can be easily managed.

The equipment required to place the interscalene block depends upon the technique selected by the anesthesia care provider. There are two main methods to perform an interscalene block: intentional elicitation of paresthesias and the nerve stimulator technique. Several different types of needles are available for the different techniques that may be utilized.[47] (Fig. 43-5). An additional technique includes the use of combination needles that have the capability of ultrasound guidance and stimulation. These needles initially stimulate through the cutaneous layer and then are advanced with stimulation.

Preparation of the Patient

To avoid undue anxiety, the patient should have a full understanding of the process of the placement of an interscalene block. The patient should also have an understanding of why the block is being placed and how that will affect his or her postoperative outcome, including how the patient will remain relatively pain-free for a number of hours in the postoperative period and have a motor block for several hours postoperatively, depending on which local anesthetic is injected. Additionally, common side effects such as the probability of decreased functioning of the ipsilateral phrenic nerve, stellate ganglion, and ipsilateral recurrent laryngeal nerve should be presented so that the physical experience will not surprise the patient. Altered chest excursion, dry eye, droopy eyelid, and hoarse voice should be described in advance. If there are any questions about the capability of the block to serve as the sole anesthetic for the surgical procedure, it may be wise to proceed with a general anesthetic with the utilization of the interscalene block for postoperative pain purposes. This avoids the situation where a patient is uncomfortable "under the drapes" and disrupts the surgery with sudden movements.

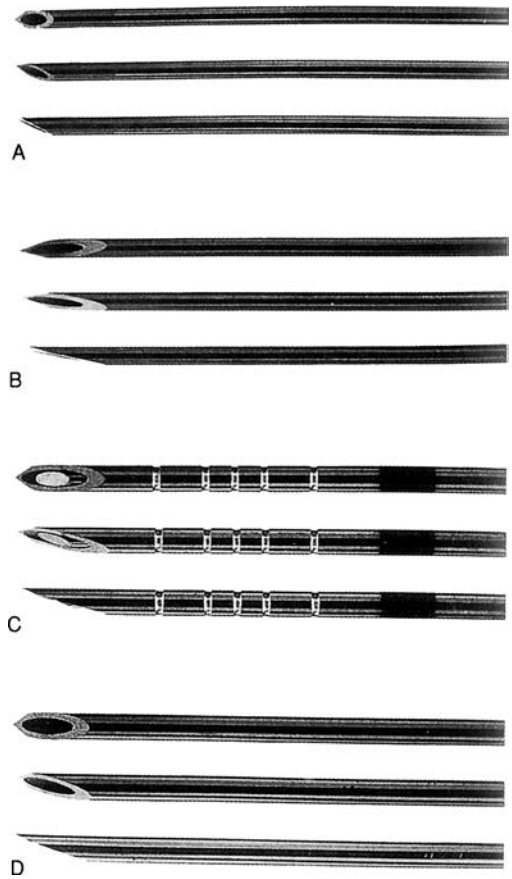

Figure 43-5 Frontal, oblique, and lateral views of regional block needles. **(A)** Blunt-beveled, 25-gauge axillary block needle. **(B)** Long-beveled, 25-gauge (hypodermic) block needle. **(C)** Ultrasound "imaging" needle. **(D)** Short-beveled, 22-gauge regional block needle. (Reprinted with permission from Neal J, McMahon D. Induction of regular anesthesia: equipment. In: Brown D, ed. *Regional anesthetic analgesia.* Philadelphia: WB Saunders, 1996: 159–172.)

INSERTION OF THE BLOCK AND INTRAOPERATIVE CARE

The interscalene approach to the brachial plexus is the most proximal and cephalad brachial plexus block.[76] It is performed at the level of the trunks of the plexus, and most often at the site of the origin of the superior trunk over the transverse process of C6. Both the effect of the block and the types of complications are related to this proximal site of approach.[74]

Applied Anatomy of the Brachial Plexus

The anatomic basis for the interscalene block is the reliable location of the brachial plexus in the groove formed by the overlap of the anterior and middle scalene muscles at the level of the cricoid cartilage. This occurs over the transverse process of the vertebral body of C6. The entire brachial plexus is theoretically accessible at this point since the sheath of the brachial plexus is formed, and any injection within this sheath will have access to the entire brachial plexus at the level of the trunks (Fig. 43-6). Since the sheath is thin, due to the need for hypermobility of the neck, confirmation of needle placement within the sheath is necessary. Confirmatory signs must be identified by dermatome to confirm placement. If the needle stimulates the brachial plexus at this site, the terminal nerves likely to respond are the axillary and musculocutaneous, sometimes in conjunction with the radial nerve. When there is a motor-evoked response or a paresthesia in the arm, it is important to be able to identify the nerve of origin, both to confirm needle placement within the sheath and to predict outcome. Knowledge of branches of the plexus that occur high in the neck will allow identification of cases where these nerves are stimulated, since injection of the drug at these sites is not within the sheath of the brachial plexus and will not result in a block. The suprascapular nerve, the long thoracic nerve, and the distal aspects of the axillary nerve after it leaves the sheath are examples. A paresthesia to the anterior chest or to the area of the scapula should not be mistaken as a confirmatory sign of brachial plexus entry.

The entry at the cephalad aspect of the plexus predicts the nerves most and least likely to be blocked by the classical interscalene block. The site closest to the injection will receive the highest exposure to local anesthetic at the injecting concentration. Those areas within the sheath that are furthest away, geographically, will receive the least.[38] This is why the interscalene block is best suited to the postaxial and proximal upper-extremity surgery. The dermatomes served by the most caudad aspects of the plexus (the ulnar nerve) will be incompletely blocked in a finite number of cases. This makes the interscalene approach less ideal for hand and wrist surgery. The work of Winnie[76] with contrast injected into the sheath of the brachial plexus suggests that at high volumes, the sheath should be equally well filled from any site. The clinical work of Anderson et al.[2] demonstrates otherwise, with a 30% sparing of the ulnar nerve with classical interscalene block, even with high volumes. The variability within the sheath of the brachial plexus demonstrated by Thompson and Rorie[69] in the axillary area may explain this effect in the interscalene sheath area.

The interscalene brachial plexus can be identified by elicitation of paresthesia or motor-evoked response. Active confirmation of proximity to nerves is important, as opposed to regional techniques based on feeling the sheath, since the interscalene brachial plexus sheath is so thin that elicitation of an active sign is the only way to achieve a high success rate.

Locating the Interscalene Groove

The patient is positioned supine with the head turned to the contralateral side. The external landmarks are identified: the

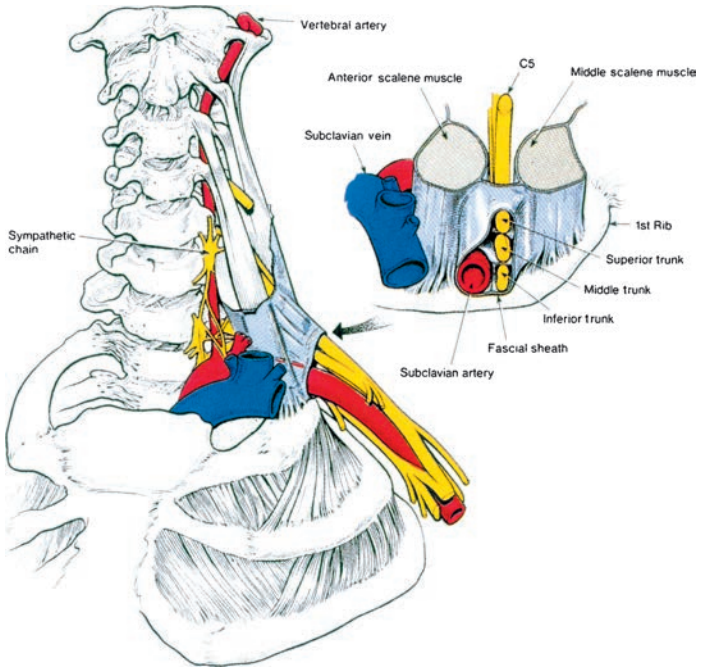

Figure 43-6 Brachial plexus sheath and scalene muscles. Note the brachial plexus sandwiched between the anterior and middle scalene muscles, and the prevertebral fascia splitting to enclose scalenes and then forming a fascial sheath around the brachial plexus. Note also the relationships to the vertebral artery, subclavian artery, and sympathetic chain. (Reprinted with permission from Bridenbaugh L. The upper extremity: somatic blockade. In: Cousins M, Bridenbaugh P, eds. *Neural blockade*, 2nd ed. Philadelphia: JB Lippincott, 1988:387–416.)

cricoid cartilage, the external jugular vein, and the prominence of the posterior boarder of the clavicular head of the sternocleidomastoid (SCM) muscle[15] (Fig. 43-7). The interscalene brachial plexus is most easily approached in the interval between the anterior and middle scalene muscle, just caudad to the level of the cricoid, over the transverse process of C6. To identify this position, the nondominant hand of the anesthetist is placed at or below the horizontal level of the cricoid cartilage on the posterior border of the SCM and swept from medial to lateral. As the prominence of the SCM is no longer felt, there is an open space, which the inexperienced regionalist may perceive as the

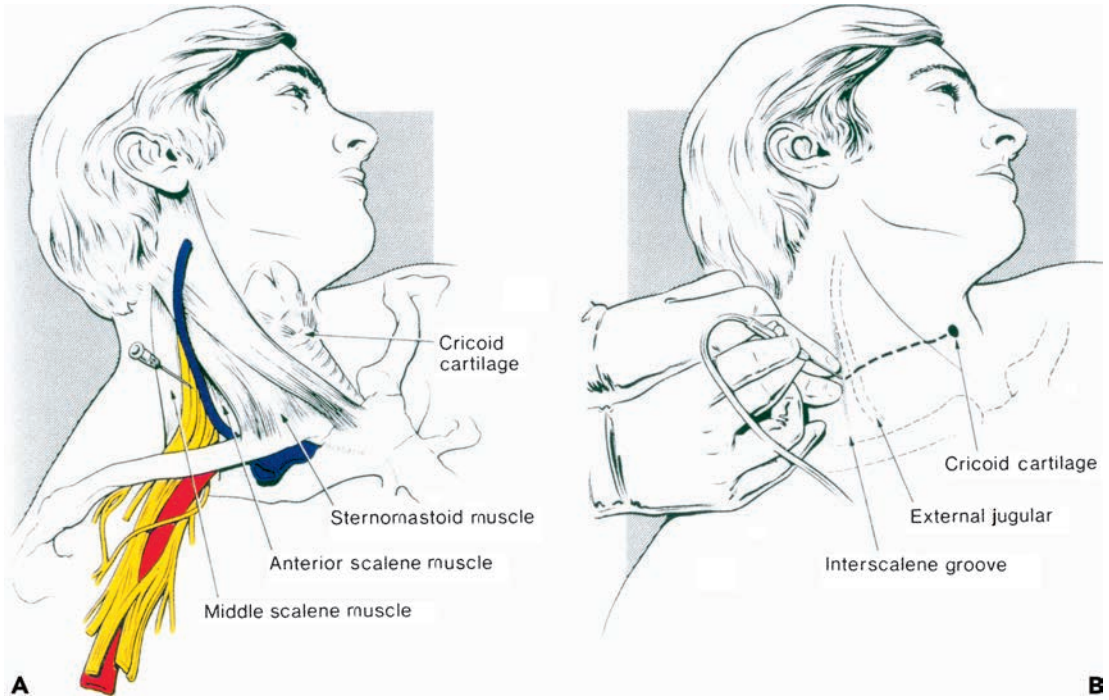

Figure 43-7 Interscalene block: anatomic landmarks. (Reprinted with permission from Bridenbaugh L. The upper extremity: somatic blockade. In: Cousins M, Bridenbaugh P, eds. *Neural blockade*, 2nd ed. Philadelphia: JB Lippincott, 1988:387–416.)

"interscalene groove." The only structure in this area is the lateral border of the internal jugular vein, and the brachial plexus will not be found in this interval. As the fingers move more lateral, an up-slope is encountered, which is the medial aspect of the anterior scalene muscle. As the flat belly of the muscle is encountered, an indentation is eventually felt. This occurs where the middle scalene and the anterior scalene muscle cross each other. It is this crossing that creates the "interscalene groove," and directly dorsal to this will be found the superior trunk of the brachial plexus.[60]

Different Techniques to Determine Location of Block

Paresthesia and nerve stimulator techniques are widely accepted in clinical practice and result in high rates of successful blockade. Studies have been done to evaluate the comparative placement of needles with respect to the two different types of techniques. Urmey and Stanton utilized a paresthesia technique to locate where the interscalene block injection should take place.[71] After fixing the needle in place, they applied a nerve stimulation with up to 1.0 mA with a pulse width of 0.2 million seconds and found that only 30% of the patients exhibited any motor response to electrical stimulation. Urmey and Stanton also noted that there was no relation between the site of paresthesia and associated motor nerve response. These results may be related to the location of the motor nerve fibers in relation to the sensory nerve fibers.

Paresthesia Technique

The paresthesia technique is performed with a short, blunt-beveled needle. It is assumed that the short-beveled needle tends to push the nerve aside instead of cutting, as a sharp-beveled needle would do. This author favors the use of paresthesias, performed in an awake responsive patient who is able to cooperate while the block is being performed. Success rate with the utilization of the paresthesias technique for interscalene block is over 90%.[68]

For paresthesia technique, a 25-gauge, blunt-beveled needle, less than 1 in., is selected. It is connected to an extension set resulting in the "immobile needle" of Winnie for smoother operation.[75] After appropriate sterile technique, skin wheal is placed, most often just lateral to the prominence of the external jugular. The needle is directed perpendicular in all planes, except slightly caudad and mesiad, to approach the plexus straight on as opposed to a tangent, which decreases the target area. Prior to advancing the needle, the patient is told what to expect; the paresthesia is described, and the patient is told that correctly identifying the paresthesia is required to perform the block. The patient should be told to first identify a feeling to stop the movement of the needle, and then be asked to locate the feeling. The lag between feeling and being able to localize the sensation is long enough that needle placement will not be as accurate. A clue of impending paresthesia is a change of facial expression consistent with the feeling of stimulating any nerve in the human body. When a sensation is identified originating in the neck, passing through the shoulder and into the arm, perhaps as far as the thumb (radial nerve), the patient needs to identify this and injection occur directly over the superior trunk of the brachial plexus. It may be helpful to describe the paresthesia in terms of the feeling that most people have had when they strike their ulnar nerve at the elbow, the "funny bone." Perceived feeling anywhere in the arm is a reliable indicator of the superior trunk; paresthesia appreciated in the area of the scapula, the acromion, or the pectoral area is an indicator of the suprascapular, axillary, or long thoracic nerves, which are not located reliably within the sheath of the brachial plexus and should not be accepted as confirmation of placement within the brachial plexus sheath.[55] With elicitation of the correct paresthesia, pressure should be applied cephalad to the needle and injection of 40 mL of a local anesthetic solution in incremental doses should be completed. Given the proximity of the vertebral artery and the epidural/subarachnoid space, the first injection should be a very small amount. The elicited paresthesia may briefly accentuate to a mild degree. Any dramatic increase should be taken as a sign of potential intraneural injection and the needle withdrawn very slightly. Then a very small first injection should be repeated. One excellent indicator of intravascular injection is the local anesthetic solution with 1:200,000 epinephrine added; 3 mL of this solution contains 15 μg of epinephrine, which if rapidly injected should cause a rapid, transient 30% or greater increase in the heart rate in most patients. Unless the vascular structure happens to be the carotid or vertebral artery, this should not be enough of any local anesthetic to cause a toxic reaction. The potential for vertebral artery injection must be detected by the profound patient response to the first, very small injection. Once test dosing has been completed, incremental injection follows, with frequent, gentle aspiration to rule out migration into vein, artery, or the subarachnoid space. The brief augmentation of the paresthesia should be rapidly followed by complete resolution of the feeling. When rapid- or intermediate-onset local anesthetic solutions are selected, there may be early signs of the onset of the block such as warmth in the arm, tingling, or areas of noticeable numbness in proximal dermatomes prior to the completion of the injection.

Nerve Stimulator Technique

The nerve stimulator technique can be utilized in one-shot block technique or can be utilized for the placement of an interscalene catheter that may provide postoperative pain relief. The selection of the use of a nerve stimulator to place an interscalene block seems to be more a function of

the "comfort level" anesthesia care providers experience than any other reason. Both methods are well described and accepted in the literature, with no overwhelming differences in complications or side effects. Interestingly, the utilization of a nerve stimulator to perform an interscalene block has been associated with the rare complication of prolonged hemidiaphragmatic paralysis.[23]

When a nerve stimulator technique is chosen, a 22-gauge, blunt insulated regional needle is utilized with a variable voltage nerve stimulator. The landmarks and approach is the same and the endpoint is a motor-evoked response in the arm. After the motor-evoked response is elicited at 1 mA or greater, then stimulating current is decreased to determine the lowest amount of current to produce a stimulus. If the lowest current output is 0.6 mA or higher, then the success rate of the block will only be 36%.[7] However, when the lowest stimulating current is 0.5 mA to 0.3 mA, then localization is adequate and block success rate is 82% to 93%.

Placement of the Interscalene Catheter with a Nerve Stimulator

Generally, most practitioners who place an interscalene catheter utilize a nerve stimulator technique. This can be achieved by using a catheter-through-the-needle versus a catheter-over-the-needle technique. The interscalene brachial plexus is identified using a nerve stimulator connected to the proximal end of the metal inner needle, with the endpoint of contraction of the upper-extremity muscles being obtained with a current of less than 0.5 mA with an impulse duration of 0.1 millisecond. When utilizing the cannula-over-needle technique, the catheter is introduced distally and advanced between the anterior and middle scalene muscle up to 2 to 3 cm. The catheter will be subcutaneously tunneled 4 to 5 cm through an 18-gauge intravenous cannula and fixed to the skin with adhesive tape. Advances in needle design permit the use of insulated Touhy-type needles through which catheters can be placed, including stimulating and nonstimulating catheters for achieving continuous brachial plexus blockade.

Assessing the Block

It is essential when you perform a regional anesthesia to be able to assess if the block will be successful for the intended purpose. Depending upon the local anesthetic that was utilized, the block will generally start to "set up" within 3 to 5 minutes and should have sensory and motor components initiated by 20 minutes. This author utilizes the "numb thumb" sign, where the thumb on the side of the interscalene block becomes tingly and numb. If this occurs within 5 to 10 minutes of completion of the block, clinical experience has demonstrated that the block will be appropriate for a surgical anesthetic. Other authors have reported that adequate surgical anesthesia is present when, within 20 minutes after the administration of the local anesthetic, there existed a sensory block with the inability to recognize cold temperature on the first and third fingers and a motor block with the inability to extend the arm involving the radial and median nerve.[24]

SELECTION OF A LOCAL ANESTHETIC

The choice of agent for interscalene block depends on the anticipated duration of the surgical procedure, the degree of motor block required, and the plans for regional anesthesia for postoperative pain relief.[70] There are a number of concentrations and doses of medications used for interscalene block anesthesia (Table 43-3). One factor influencing the selection of a medication and its concentration is the desire for a motor blockade. The degree of motor block required varies from complete motor block necessary for open shoulder procedures to insignificant when soft tissue procedures of the proximal upper extremity are performed, unless the dissection is so tedious that minor movement would be either disruptive or dangerous to the structures being operated on. As the concentration of the agent is decreased from the maximum commercially available, the degree of motor block decreases. With mepivacaine and lidocaine, the concentration can be varied from complete motor block to variable motor block. This is especially true with bupivacaine, where excellent sensory analgesia can be provided with minimal motor block. While this is an ideal property for obstetrics and postoperative analgesia for extended use after surgery, it can be less than ideal for surgical anesthesia for proximal arm and shoulder surgical procedures. With the volume required to achieve complete blockade, the stronger concentrations of bupivacaine, particularly 0.75%, can be toxic and the lower concentrations may achieve only partial motor block. There are several medications that may be routinely added to local anesthetics to modify their block characteristics, including bicarbonate,[67] epinephrine, and clonidine[30] (Table 43-4)

When revision surgery or technical difficulty is expected, long-acting blockade may be important. The longest-acting agent for conduction blockade is bupivacaine, with 8 to 12 hours of surgical anesthesia likely and pain relief for much longer than this. Given the cardiac toxicity issues with bupivacaine, an alternative long-acting agent is tetracaine. Since the onset is slow and variable with tetracaine, it is most often compounded with a short- or intermediate-acting agent like mepivacaine ("supercaine") or lidocaine to take advantage of their more rapid onset. As long as the concentration exceeds 0.15%, the motor block achieved with tetracaine should be complete and last 6 to 8 hours, with pain relief much longer, although not quite as long as bupivacaine. 2-Chloroprocaine will provide 45 to 60 minutes of surgical anesthesia and may not be useful unless used with a catheter technique or

TABLE 43-3
REGIONAL ANESTHESIA MEDICATIONS—DOSING AND COMMENTS

	Medication Dosing	Comments
Primary surgical anesthetic	1.4% mepivacaine with 0.2% tetracaine, epinephrine 1:2000,000 and bicarbonate with 35 mL injected	Provides a quick onset of sensory and motor blockade, with excellent postoperative pain relief lasting 8 to 12 hours
Postoperative analgesia	0.2% ropivacaine with 2 μg/mL of clonidine with 20 mL injected	Provides excellent postoperative analgesia with minimal motor block lasting 8 to 12 hours
Blockade placed at time of the catheter placement	0.5% bupivacaine or 0.5% ropivacaine with 30 mL injected	Provides surgical anesthesia
Continuous catheter infusion	0.125% bupivacaine or 0.2% ropivacaine at 6 mL per hour continuous infusion	Provides a good postoperative analgesia

compounded with a longer-acting agent. Lidocaine and mepivacaine at 1.0% or greater will provide complete surgical anesthesia for 2 to 3 hours when used with epinephrine (slightly longer for mepivacaine).

Complications of Interscalene Nerve Block

The most common complications of the interscalene block occur when adjacent nonbrachial plexus nerves are blocked. As described earlier, a 30% to 100% block rate of the ipsilateral phrenic,[33,58,72] recurrent laryngeal nerves,[59] and stellate ganglion[1,31] is expected. As long as the patient is seated and the physical effects are presented in advance to avoid frightening the patient, these are not associated with morbidity in most instances. Morbidity can occur with phrenic nerve block in the patient with pulmonary disease who may not tolerate either the subjective dyspnea or the minor loss of functional residual capacity when respiratory function must run from one side of the diaphragm only.

The serious complications of interscalene block are intravascular injection, high central block,[4] and nerve injury. The anatomy previously described makes it obvious that the interscalene block may be associated with a risk of

TABLE 43-4
MEDICATIONS ADDED TO LOCAL ANESTHETICS TO MODIFY BLOCK CHARACTERISTICS

Medication	Desired Effect	Concerns/Side Effects
Bicarbonate	Quicker onset and more solid motor block. Dosing is routinely one equivalent of bicarbonate for every 10 mL of local anesthetic solution	May cause mepivacaine to precipitate in catheter. Needs to be "freshly" added to mixture
Epinephrine	Prolonged duration of block and utilized for determination of intravascular injection	Tachycardia may occur with absorption
Clonidine	Prolonged duration of block	Systemic hypotension, bradycardia, and sedation are seen with 150 μg dosing

intraarterial injection.[49] Since two of the major arteries at risk are direct feeders of cerebral circulation (carotid, vertebral), central nervous system (CNS) toxicity would obviously be rapid and severe. The vascularity of the anterior neck is dense, so the potential for rapid vascular uptake of agent injected correctly is high at this site, compared with other approaches to the brachial plexus. Signs of intravenous injection would be slower than the abrupt onset of seizure activity expected with intraarterial injection at this level. Rapid vascular uptake would be slower yet, with easily defined progression from prodrome, through excitation prior to seizure activity. Obviously, the slower the toxic CNS level is achieved, the more potential there is for therapeutic intervention to raise the seizure threshold and prevent seizures. The relatively short distance to the heart from this venous circulation will accelerate the progression by comparison with the arm or especially the leg.

High central block at either the epidural, subdural, or subarachnoid spaces is possible due to the close proximity of the dural sleeve, directly behind the transverse processes at the intervertebral foramina.[4] If the needle selected is long enough and the direction medial enough, any of these spaces can be reached. Aspiration will not detect location in the epidural or subdural space, and the dural sleeve placement may allow entry into the subarachnoid space without easy aspiration of cerebrospinal fluid. The onset will be most rapid and abrupt in the subarachnoid space with apnea, unconsciousness, and the profound hemodynamic compromise associated with total sympathectomy. The onset of epidural block will be slower, and since the epidural space ends at the foramen magnum, unconsciousness is variable. Subdural injection is very slow and patchy, involves cranial nerves, and is a diagnosis of exclusion. Episodes of hypoxia following interscalene blockade have been reported,[56] including an episode of asymptomatic profound oxyhemoglobin desaturation following interscalene block in a geriatric patient.[64]

Nerve Injury

Nerve injury from interscalene block occurs because of either directed intraneural injection or needle trauma to the nerves during attempted block, especially during repeated attempts to relocate the brachial plexus after local anesthetic has already been injected. Avoidance of paresthesia or care to avoid searing paresthesia associated with intraneural injection should decrease this risk. The complications of indwelling interscalene catheters will be discussed as a part of the postoperative pain management discussion.

Postoperative Pain Management

Postoperative pain is a significant issue in the management of shoulder surgery patients. The ability to be pain-free at the end of the procedure may play an important role in the instance where early mobilization is encouraged. Methods of postoperative pain relief include regional anesthesia with a long-acting local anesthetic medication, a cervical epidural catheter, or a continuous interscalene catheter. Other methods of postsurgical pain control, which may be used as the primary method or as an adjunct to the local anesthetics, include oral narcotics, nonsteroidal antiinflammatory drugs (NSAIDs), and intravenous patient-controlled analgesia with opioids.

Local Anesthetics

Due to the nature of orthopedic surgery, there can be numerous applications of regional anesthesia for surgical anesthesia. Knowing that the acute postoperative pain is severe after reconstructive orthopedic procedures, the long-acting local anesthetics are often selected to provide acute postoperative pain control. These can be applied at any level from skin infiltration, conduction block, or central block with and without the addition of opioids.

Preoperative Single-Shot Interscalene Block for Postoperative Pain Relief

Single-shot interscalene placement of meperidine was reported by Davidas et al.[21] with a long duration of analgesia (up to 24 hours) after open shoulder procedures. Brandl and Taeger advocated interscalene block with bupivacaine as a part of a balanced anesthetic technique for both intraoperative anesthesia and extended postoperative analgesia for up to 24 hours after open shoulder procedures.[14]

Interscalene Catheter

If the postoperative pain plan includes the need for a prolonged local anesthetic blockade, a catheter can be placed into the sheath of the brachial plexus at the level of the interscalene groove, with continuous infusion of low concentrations of local anesthetic. If an interscalene catheter is inserted, it could be maintained into the postoperative period with low-dose local anesthetic and used during physical therapy sessions. With continuous interscalene analgesia, pulmonary function improves,[41,45,50] opioid needs decrease,[26] and the conditions for rehabilitation improve.[73]

When compared to patient-controlled analgesia (PCA) with morphine, an interscalene catheter with patient-controlled dosing provided superior analgesia with reduced side effects attributable to morphine, such as nausea or pruritus.[13] The same benefit can be achieved with ambulatory patients when an interscalene catheter and a disposable local anesthetic delivery device are compared to parenteral opiates in the postanesthesia care unit and oral narcotics for ambulatory analgesia.[35] The techniques for patient-controlled interscalene analgesia have been studied, and it

was reported that a continuous infusion combined with patient-controlled doses improved the quality of analgesia compared with the patient-controlled doses without the basal infusion.[62] The safety of interscalene catheters and continuous infusion of 0.25% bupivacaine has been established by showing acceptable plasma levels up to 52 hours after the start of the infusion.[34]

Tuominen et al.[70] described the technique, noting the technical difficulty of placing and maintaining a catheter in the neck. Fixation of the catheters to the skin was felt to decrease catheter dislodgment during physical therapy. Haasio et al.[26] reported minimal need for supplemental opiates after shoulder surgery with an interscalene catheter, although 10% of the patients experienced at least mild signs of local anesthetic toxicity within the 24 average hours that the catheter was used.

There have been more reports of technical difficulties with the placement and maintenance of interscalene catheters than with any other peripheral catheter technique. Tuominen et al. reported six catheter failures and one pump failure among 24 patients.[70] The use of continuous interscalene block resulted in severe bupivacaine toxicity in a case reported by Pere[49] involving the migration of the catheter into an artery at a prolonged interval after the placement. Use of an epidural needle and catheter may improve this failure rate.[9] Continuous interscalene block will maintain the decreased function of the diaphragm based on ipsilateral phrenic nerve block[12] and could be associated with a decrease in ventilatory function.[50] Even reduction of the concentration of the bupivacaine to 0.125% or further reduction with the addition of fentanyl did not decrease this potential adverse effect.[50] Hassio et al. reported ventilatory compromise in one patient, CNS toxicity in four patients, and catheter failure in five patients in a series of 40 patients.[26] Despite the technical issues and potential adverse effects, the use of interscalene catheters is significantly increasing.[40]

There are a number of complications associated with indwelling interscalene catheters. One of the obvious concerns is the risk of infection. Other complications include lower lobe collapse,[57] pleural effusion and chest pains,[66] and intrapleural migration of the interscalene catheter.[65]

Signs of infection at the site of catheter insertion are always a concern, and in a study of 700 patients by Borgeat et al., six patients (0.8%) demonstrated signs and symptoms of infection.[11] Local pain, redness, and induration were noted in one patient after 3 days and in four patients after 4 days. All five of these patients were successfully treated with antibiotics. One additional patient required surgical drainage and after 10 days of antibiotics had a complete recovery. Ekatodramis et al.[24]reported a study of 27 patients in which one patient (3.7%) had the interscalene catheter removed at 18 hours because of the appearance of painful swelling at the site of catheter insertion and complete paralysis of the affected arm. It is not clear from the publication if this was an infection or resulted from some other cause.

Suprascapular Nerve Block

The suprascapular nerve has sensory fibers to the acromioclavicular joint, the subacromial space, and the posterior capsule of the glenohumeral joint. The suprascapular nerve supplies 70% of the sensory innervation of the shoulder joint. Richie et al.[54] found that utilizing 10 mL of 0.5% bupivacaine with 1:200,000 epinephrine resulted in no complications in 25 arthroscopic shoulder surgery patients. In the immediate postoperative period, there was a reduction in morphine utilization and decreased incidence of nausea along with a decreased duration of hospital stay, versus a control group of patients who received a sham block with saline. However, when comparing pain relief after arthroscopic shoulder surgery, Singelyn et al.[61] found that interscalene block is the most efficient analgesic technique with a significant reduction in morphine consumption and a better satisfaction score than suprascapular block or intraarticular injection. They did conclude that suprascapular nerve block would be a clinically appropriate alternative.

Additional Techniques of Pain Control

Hadzik et al.[27] compared local wound infiltration after general anesthesia with brachial plexus anesthesia for outpatient rotator cuff surgery and found that patients with brachial plexus blocks experienced a better recovery profile including bypassing the first phase of recovery more frequently, having less pain, ambulating sooner, and being ready for discharge sooner. Despite the simplicity of local wound infiltration, interscalene blocks provided a superior analgesia for outpatients. Intraarticular injection of opiates seems to have a benefit. There is evidence that local anesthetics injected into joint spaces may be more effective if there is opioid in the solution.[22,52] This is based on the discovery that there may be peripheral opioid receptors that could modulate the expression of nociceptive signals on the tissue level. Intraarticular placement of a catheter may seem to be technically appealing from the surgical aspect, but analysis reveals that the analgesia is superior with interscalene plexus blockade.

Cervical epidural anesthesia has been shown to provide effective relief of pain after shoulder reconstruction.[5,10] The advantage of a cervical epidural catheter is enhanced stability for prolonged postoperative use. The catheter should be placed under fluoroscopic guidance and the tip of the catheter should be preferentially located closer to the nerve roots of the affected side. Tunneled cervical catheters have been used to provide several weeks of prolonged postoperative analgesia.

Opioids

The use of narcotic analgesics as a part of surgical anesthesia has a rational basis during orthopedic surgery, since the manipulation of bone and soft tissue results in a high density of nociceptive stimuli from these structures. Many of the synthetic narcotic analgesics available for general anesthesia have the advantage of a rapid onset, with stable hemodynamic profile. They also have a relatively limited duration, which limits their use to control pain into the postoperative period unless administered by PCA devices. The termination of the analgesic properties of the short-acting, synthetic narcotics in the fentanyl family is rapid enough that they have limited postoperative analgesia even after short surgical procedures. Longer-acting narcotics, like morphine and meperidine, can be used for their general anesthetic effects and their prolonged duration of analgesia into the postoperative period. If parenteral narcotics will be the primary means of acute pain control after general anesthesia, it may be more rational to use these medications during the final minutes of the surgical procedure. The onset will occur prior to emergence and the patient will get the maximum possible analgesic duration. The only limitation is that the sedative and respiratory depressant properties of the narcotics can interfere with awakening from general anesthesia. If the medication is administered during a period of spontaneous, assisted ventilation during surgical wound closure, the respiratory rate and depth can be used as a guide to prevent excessive narcotic treatment. If administered in incremental doses during spontaneous ventilation and stopped at the first sign of respiratory depression, it is unlikely that excessive opioids will be administered.

Nonsteroidal Antiinflammatory Drugs

It is also possible to administer nonnarcotic analgesics, like NSAIDs, during emergence from anesthesia. There has been an increase in NSAID use since the release of ketorolac. Intramuscular, intravenous routes can be chosen with ketorolac,[19,37] and with other NSAIDs such as ibuprofen and indomethacin, the oral and rectal routes are possible. There is good evidence of efficacy in orthopedic patients from these compounds, but there is caution because of the effects of NSAIDs on the coagulation cascade.[36] It is still to be determined whether intraoperative administration of ketorolac increases the postoperative bleeding. Rectal indomethacin did not.[44] This is a critical issue with reconstructive orthopedics, both from the perspective of transfusion and the issue of hematoma formation, which increases the risk of wound infection, especially when there is instrumentation left in the operative site. Caution should be exercised in the utilization of NSAIDs in patients who are hypovolemic, as this will inhibit prostaglandin synthesis and thus lead to unopposed renal vasoconstriction, which may result in a precipitous decline in renal function. This effect could be even more pronounced in patients who have chronic renal failure preoperatively. It should be noted that a cyclooxygenase-2 inhibitor, rofecoxib, was withdrawn by the U.S. Food and Drug Administration because of adverse cardiovascular effects. Initially investigators may have concluded that the difference in cardiac events from the rofecoxib group and naproxen group was due to the cardioprotective effects of naproxen. By utilizing meta-analysis, it has been demonstrated that naproxen has only a small cardioprotective effect, thus resulting in the conclusion that rofecoxib has a significant cardiovascular risk.[32] Controversy still exists as to why the manufacture and drug licensing authorities did not continuously monitor this risk, which was suspected in the literature as early as the year 2001.[46]

Patient-Controlled Analgesia

PCA devices may be ideally suited to orthopedic patients. The procedures cause considerable pain, and patients know this prior to the surgery. The preoperative instruction required to select any PCA pain-control choice serves as a reassurance to the patient that there will be acute pain control available as soon as the surgery is completed. If general anesthesia is chosen, the patient can count on the PCA pump to be available as soon as the patient is able to operate the pump. The spirit of preemptive analgesia is served by continuing the interruption of the pain cycle from one modality to another without interruption.

CONCLUSIONS

Regional anesthesia techniques play an important role in the management of pain in the perioperative period surrounding shoulder surgery. The surgical procedure may be conducted under a general anesthetic, regional anesthetic, or a combination of both. Many patients and surgeons enjoy the benefits conferred by regional anesthesia, including its utilization for postoperative analgesia. Indwelling catheters may be utilized for prolonged postoperative analgesia. Despite advantages attributable to the utilization of regional anesthesia for shoulder surgery, there continues to still be a limited utilization of these blocks. Some authors have attributed this to the limited number of regional anesthesia instructors available in teaching institutions in addition to the focus on general anesthetics in residency programs. A nationwide survey of anesthesiology residents demonstrated that the majority of third-year residents expressed a low confidence in their ability to perform most peripheral nerve blocks.[63] Evans et al.[25] in a general review of peripheral nerve blocks and continuous catheter techniques concluded that to foster the continued use of peripheral nerve blocks for postoperative analgesia, it is

essential to improve practitioner-teaching modalities and that additional research may be needed in demonstrating improved patient outcomes to further their expanded use. Continuous interscalene catheters may have a role in providing extended analgesia, but the ultimate risk and benefit ratio still remains to be determined.

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